NBER WORKING PAPER SERIES

THE INTER-GENERATIONAL AND SOCIAL TRANSMISSION OF CULTURAL TRAITS: THEORY AND EVIDENCE FROM SMOKING BEHAVIOR

Rebekka Christopoulou
Ahmed Jaber
Dean R. Lillard

Working Paper 19304
http://www.nber.org/papers/w19304

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
August 2013

This research is funded by the National Institute on Aging (grant 1 R01 AG030379-01A2). The paper has benefited from discussions with participants of the Health Behaviors and Disparities Research Seminar at Cornell University; the Conference on the Economics of Interactions and Culture at the Einaudi Institute for Economics and Finance; the Applied Microeconomics Seminar at the Ohio State University; and the 2013 Conference of the European Society of Population Economics at Aarhus University. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

The Inter-generational and Social Transmission of Cultural Traits: Theory and Evidence from Smoking Behavior

Rebekka Christopoulou, Ahmed Jaber, and Dean R. Lillard

NBER Working Paper No. 19304
August 2013
JEL No. D1,I1,Z1

ABSTRACT

The extant literature on cultural transmission takes competing cultures in society as given and parental cultural preferences as fixed. We relax these assumptions by endogenizing both societal and parental preferences. We use smoking as a case-study of a cultural trait which did not always exist, and which over time has switched from being perceived as socially acceptable to being perceived as undesirable. In our model, parents' preferred cultural traits depend on the perceived health costs of smoking, and societal preferences depend on the behavior of a tobacco industry that aims to maximize smoking prevalence. We derive conditions for the emergence and persistence of the smoking habit, and find new implications for the relationship between parental and societal influences. We then test explicitly for the validity of our theoretical framework using novel US data. We find that our framework is able to capture features of smoking behavior which existing models are unable to explain.

Rebekka Christopoulou
The Ohio State University
1787 Neil Avenue
235C Campbell Hall
Columbus, OH 43210
Christopoulou.1@osu.edu

Ahmed Jaber
Cornell University
457 Uris Hall
Ithaca, NY 14853
jabera@nber.org

Dean R. Lillard
Department of Human Sciences
The Ohio State University
1787 Neil Avenue
Columbus, OH 43210
and NBER
lillard.13@osu.edu

1 Introduction

Cultural traits and norms, like risk-preferences, corruption, and altruism, are important in shaping individual economic behavior. The process by which such traits get transmitted from one generation to the next determines whether they survive and and how they are geographically distributed. In this study, we explain how cultures first emerge in society and how they may persist across generations even when parental preferences change over time. Our study adds to a growing economics literature that looks at the joint role of parental and social channels of cultural transmission.

Researchers from a wide range of scientific disciplines have long debated the nature/nurture question to evaluate the relative contribution of cultural (or environmental) and genetic effects on cognitive and psychological traits (Sacerdote 2011). Economists have also contributed to this debate, and have provided elaborate theory predicting that the genetic transmission of traits determines behavior (Robson and Samuelson 2011). However, this literature generally concurs with the standard Darwinian prediction of the survival of the fittest, and is at odds with evidence for the resilience of minoritarian ethnic and religious traits, e.g. among the US immigrant population. The 'melting pot' hypothesis, which suggests that characteristics and traits of the immigrant population in the US will converge over time until they resemble those of the general population, is not consistent with observed patterns. The observed behavior and characteristics of migrants conforms more closely to the 'salad bowl' hypothesis, which suggests that different ethnic and minoritarian communities can simultaneously co-exist and maintain their respective cultural identities. In fact, norms in the country of immigrant origin are found to significantly predict the behavior of second- and third-generation US immigrants (e.g Borjas 1992, Fernandez and Fogli 2009, Algan and Cahuc 2010). Starting with Bisin and Verdier (2001), the cultural transmission literature emerged to provide a rationale for this previously unexplained persistence of cultural traits (see Bisin and Verdier 2011 for a review).

Bisin and Verdier (2001) developed a model in which children acquire traits either via societal role models or via parental socialization efforts. They assume that children are more likely to acquire a given trait the more prevalent that trait is in society, and that all parents want to transmit their own cultural trait to their children. Given these assumptions, their model identifies a key feature of the social environment that ensures the co-existence of cultural traits in equilibrium. This property, which Bisin and Verdier term cultural 'substitutability', states that parents socialize their children less when their cultural trait is more prevalent in the population. Although ground-breaking, Bisin and Verdier's model fails to explain the persistence of traits that all parents want to avoid passing on to their children, even if they themselves possess them (e.g. insincerity, preferences for unprotected sex, substance use, low educational attainment). Saez-Marti and Sjogren (2008) address this failure by imposing that societal role models who belong to the minority group have a disproportionate influence on children. This condition ensures the resilience of traits that parents do not actively transmit.

Still, existing theory fails to account for two important aspects of cultural transmission. First, it says little about how traits emerge. If societal role models of a new cultural trait do not exist then it is unclear how that culture would arise in a society where all parents socialize their children to the same preferred culture. Second, the theory does not inform us about how and why parents with the same cultural trait may prefer different traits for their children. Most efforts have focused on developing a framework for contexts where parents' cultural attitudes are fixed. Yet, history is alive with examples of newly born cultural traits (e.g. the culture of social networking) and traits that switched from being perceived as virtuous or socially acceptable to being perceived as bad or undesirable, and vice versa (e.g. preferences about smoking, polluting the environment, pre-marital sex, polygyny, divorce, womens' rights etc.). Using the smoking habit as a case-study, we adapt the cultural transmission framework to account for this general class of traits.

Smoking of tobacco was first adopted before the 15th century by native Americans, who used it for recreation, medicinal purposes, or as a hallucinogenic in rituals. When Christopher Columbus and his crew discovered the American continent, they also discovered smoking of tobacco and introduced the practice to Europe. In 1854, Philip Morris made the first hand-rolled cigarettes in London's Bond Street. The second industrial revolution then saw the invention of the cigarette rolling machine, which made it possible to mass produce cigarettes cheaply. From that time and till the 1960s, the habit of smoking diffused rapidly, producing high profits for the tobacco industry. By the time of the world wars, smoking had become so socially acceptable that governments in most countries distributed cigarettes to troops as part of their regular daily rations. Some even continued to subsidize cigarette consumption during peacetime. The social perception of smoking started to change only after the publication of the Royal College of Physicians 1962 report on Smoking and Health (RCPL, 1962) and the US Surgeon General's Report on Smoking (USDHEW 1964). Those reports compiled and distilled for public consumption scientific evidence about the health consequences of tobacco use that had been accumulating for more than three decades. In the ensuing years, public campaigns against tobacco consumption followed. Over time, those campaigns and the ever accumulating evidence impacted the popularity of the smoking habit.

What this brief history of smoking teaches us is that the dynamics of the smoking culture have been shaped by the strategic behavior of a profit-maximizing industry, and the discovery and diffusion of scientific evidence. We take this lesson to theory and build the first model of cultural transmission that is able to predict both the emergence and the long-term persistence of culture in a world where outside forces may affect parents' preferred trait for their children. In our model, the flexibility in parental preferences is due to the availability and spread of information affecting perceptions about the health cost of smoking. This makes our setup more general than existing models, each of which assumes a particular distribution of parental preferences, e.g. that all parents promote their own trait (Bisin and Verdier 2001) or that all parents promote the same trait (Saez-Marti and Sjogren 2008). We also relax a standard assumption in the literature that children are more likely to acquire a given trait the more popular that trait is in society. Instead, we assume the existence of a tobacco industry which

can manipulate youth smoking behavior (e.g. via advertising). We respectively define as cultural conformity and cultural distinction the positive and the negative relationship between the probability of adopting a trait and the prevalence of that trait in society. Our theory formalizes the relationship of parental and social influences that existing models predict by showing that cultural substitution in the parental channel of transmission is always tied to cultural conformity in the social channel of transmission. It also makes the novel prediction that cultural distinction is always tied to cultural complementarity. To establish support for our predictions, we carry out an empirical investigation of smoking behavior using U.S. data.

Our empirical exercise adds to a small but growing group of 'structural socialization studies' that explicitly test the properties of the transmission mechanism (e.g. Jellal and Wolff 2002, Namoro and Roushdy 2008, Patacchini and Zenou 2011, Dohmen et al. 2012). Consistent with our theoretical set-up, we model smoking participation by children as a function of parental socialization efforts and societal influences. We then estimate this function using a novel dataset. Specifically, we use data on the smoking behavior of parents and children, and on parental efforts to socialize children against substance use, from the Panel Study of Income Dynamics. We combine these data with newly constructed smoking prevalence rates from the Current Population Survey, and newly collected data on individual exposure to anti-smoking information based on the content of magazine articles. Our identification of the causal effect of parental socialization relies on state- and time-variation in parental exposure to anti-smoking information. Our identication of the causal effect of the societal influences relies on the state-level measurement of the smoking prevalence, which rules out bias due to peer-choice and residential selection, and on the use of fixed effects, which account for unobserved factors that drive both individual and group behavior. We find evidence that supports our variant of the cultural transmission theory. Using our results, we project smoking participation rates of children to future generations and show that they converge to a steady-state in which smoking persists.

The paper is structured as follows. In section 2, we summarize the health economics literature on smoking behavior and explain how this falls short of describing smoking dynamics across generations. In Section 3 we formally present our model. In Sections 4-6, we describe our empirical strategy, the data we use, and our empirical results. Finally, Section 7 concludes the paper.

2 Health economics literature on the transmission of smoking

Empirically, children whose parents smoke are themselves more likely to smoke. But researchers have not established that this simple correlation reflects a causal relationship. Available evidence is often suspect because studies use poor data, small samples, or fail to account for the endogeneity of parental smoking decisions. Even the better studies have produced mixed evidence. Loureiro et al. (2010) find correlations consistent with sex-specific transmission using a UK sample. Göhlmann et al. (2010) find no sex differences using German data. Using the same data but a different identification strategy, Lillard (2011) finds that parental smoking behavior does not influence

whether children start smoking (for a review of earlier studies see Avenevoli and Ries Merikangas 2003). One explanation for these mixed findings is that parental behavior may have countervailing influences on their children smoking habits and these influences may cancel out each other's effect. For example, a child of a smoker may be discouraged from smoking by parental advice and anti-smoking rules in the home (Powell and Chaloupka, 2005). Conversely, a child of a non-smoker may be attracted to smoking as an act of revolution against parental control (Huver et al., 2007). Such diverse influences of parenting styles have been overlooked by most of the empirical studies.

Further, social transmission mechanisms of smoking may also be at play. Children may copy the smoking behavior of their peers or societal role-models irrespective of their parents' behavior. This channel of smoking transmission has been the subject of a large and growing literature in economics, which consents that 'peer-effects' are important drivers of smoking participation. This general conclusion is qualitatively robust across different definitions of peer groups, e.g. school-mates, classmates or friends (Gaviria and Raphael 2001, Powel, Tauras and Ross 2005, Lundborg 2006, Clark and Loheac 2007, Fletcher 2010); siblings (Harris and Lopez-Valcarcel 2008); partners (Clark and Etile 2006); and the population residing in the same state or prefecture (DeCicca et al 2008, Yamamura 2011). Christopoulou and Lillard (2013) argue that, in fact, societal influences on smoking participation stretch beyond current generations and across national borders. They show that the smoking prevalence of the children of British immigrants in Australia, South Africa, and the US, varies systematically with the smoking behavior of their parents' birth cohort in the UK when that cohort was at the same point in their life-cycle. The implication is that immigrant parents who grew up in a culture that tolerated (or even condoned) smoking will carry and transmit those values (consciously or subconsciously) to their children, thereby increasing the probability that they smoke.

The available theory on the economics of smoking does not reflect the intuitive processes that the empirical evidence describes. To analyze smoking behavior, researchers typically use either a myopic addiction model or a rational addiction model. These models aim to explain the life-course dynamics of the consumption of addictive goods assuming that individuals introspectively change their own preferences. The former model assumes that current consumption depends on one's own past consumption (Pollak 1970, 1976a); the latter model assumes that current consumption depends both on its past levels and on expectations of future consumption (Stigler and Becker 1977; Becker and Murphy 1988). Both models ignore the possibility that one's consumption may depend on the (past, present, or future) consumption of another person. As importantly, both models stipulate that the initial consumption of the addictive good (i.e. when previous consumption has been consistently zero) depends only on current factors and characteristics (e.g. prices). Thus, neither model is able to account for the fact that two individuals with similar characteristics who face exactly the same environment may take different smoking initiation decisions depending on their familial experiences during childhood. In effect, the models fail to achieve their purpose of describing consumption dynamics over the life-course, and they completely ignore consumption

dynamics across generations. The same holds for subsequent models that were built to reconcile the myopic and rational addiction theories (e.g. Orphanides and Zervos 1995, 1998).

The economic theory on social interactions, which was developing concurrently with the theory of addiction, offers a less restrictive framework of analysis. The benchmark study by Pollak (1976b) developed a model of habit formation and learning, allowing individual preferences to depend on others' behavior, which either provides information about the costs and benefits of behavior, or establishes a point of reference. However, rarely have the theoretical implications of this approach been drawn with respect to smoking behavior, and in the few occasions that they have, they have focused on peer effects and have ignored the parental channel of transmission (e.g. Nakajima 2007, Poutvaara and Siemers 2008). In this paper, we draw on the literature of intergenerational transmission of cultural traits to extend the theoretical analysis in this direction.

3 The model

3.1 Environment

Assume that there are an infinite number of periods ($t = 1, 2, 3, ...$), and each individual is alive for two consecutive periods. A person born in period t is a child in period t and an adult in period $t + 1$. Each individual bears one child in adulthood. Hence, in period $t + 1$ the population consists of two overlapping generations: adults (born in period t) and children (born in period $t + 1$). We use "he" to refer to a generic child and "she" to refer to a generic adult.

Individuals are either smokers or non-smokers; we call q_t the proportion of the youth who smoke in period t. We then denote Q_{t+1} the proportion of adult smokers in period $t + 1$. An individual's smoking behavior need not be constant throughout the course of a lifetime, since a smoker may decide to quit smoking. To simplify the exposition, we however impose $Q_{t+1} = q_t$. This is consistent with the literature on cultural transmission, which assumes a cultural trait is acquired once and for all during childhood.[1] Our results are qualitatively robust to extending the model to one where parents may quit smoking in adulthood.

3.2 Socialization process

Children are born without predefined traits and acquire their smoking behavior through a transmission process. A child is first exposed to his parent's influence, a process we refer to as **vertical transmission**. The parent's *preferred* trait need not coincide with her *exhibited* trait, and so our specification does not force parents to socialize their children to their own traits. In particular, a smoker parent may choose to socialize her child away

[1] By implicitly assuming that socialization is a function of a parent's youth (rather than adult) smoking behavior, we are imposing that attitudes towards smoking are acquired early on in life. This modeling assumption allows us to abstract away from the strategic considerations that would arise from a situation where it would be Pareto optimal for all parents to quit smoking for their childrens' sake, but who cannot credibly commit to doing so because they each have an incentive to freeride on others' efforts.

from smoking, while remaining a smoker herself. The driving force for this divergence between own behavior and desired behavior could be, for instance, that quitting has addiction costs that are only incurred by someone who already smokes. To our knowledge, Saez-Marti and Sjogren (2008) and Patacchini and Zenou (2011) are the only studies to have theoretically investigated in a cultural transmission framework the possibility that parents agree on the trait to be passed on to future generations (e.g. educational attainment). In contrast, the bulk of the literature on cultural transmission has been motivated by the study of traits like religion, where parents want to promote their own trait.

Bisin and Verdier call 'direct' transmission the process of having parents socialize children to their own traits. 'Vertical transmission' can thus be thought of as a generalization of 'direct transmission'.[2] We will then refer to a parent socializing her child to her own trait as *direct socialization*, and to a parent socializing her child to a trait other than her own as *active socialization*. From here on, we focus on the case where all parents want to discourage their children from becoming smokers. A smoker parent does not directly socialize her child to smoke, nor does a non-smoker parent actively socialize her child to smoke. This feature arises endogenously once we let parents choose the extent of vertical transmission, as it would be counterproductive to ever encourage children to acquire the unwanted cultural trait. In the terminology of the model, non-smoker parents directly socialize their children to their own trait with probability $d(q_t)$, while smoker-parents actively socialize their children to the other trait with probability $a(q_t)$. With remaining probability $1-d(q_t)$ for non-smoker parents, and $1-a(q_t)$ for smoker parents, the child acquires his trait through non parental influences, a channel which the literature typically labels as **horizontal transmission**. With probability $S(q_t)$ / $1-S(q_t)$, the child then becomes a smoker/non-smoker. We think of $S(q_t)$ as reflecting not only the influence of societal role models but also the advertising efforts of a profit-maximizing tobacco industry, as well as the child's own choices.

Throughout the analysis, we impose one of two possible assumptions on the vertical transmission process: *cultural substitution* or *cultural complementarity*. The concept of cultural substitution has been a cornerstone of the literature starting with Bisin and Verdier (2001), while cultural complementarity has received less attention (e.g. Bisin, Topa and Verdier 2004; Patacchini and Zenou 2011).

Assumption V1 (Cultural Substitution)**:** *$d(q_t)$ and $a(q_t)$ are increasing in q_t, and $d(0)=a(0)=0$.*

Assumption V2 (Cultural Complementarity)**:** *$d(q_t)$ and $a(q_t)$ are decreasing in q_t, and $d(1)=a(1)=0$.*

Assumption V1 says that a parent's vertical transmission effort is an increasing function of the unwanted trait in society, and that parents exert no effort when the unwanted trait is absent from society. When smoking is the unwanted trait, parents want to make sure their children do not fall prey to the influence of smoker role models, and so $d(q_t)$ and $a(q_t)$ are increasing functions of smoking prevalence q_t. Assumption V2 says that a

[2]Note however that our terminology deviates from the literature, which interchangeably uses "vertical" and "direct" transmission to refer to children mimicking their parents.

parent's vertical transmission effort is a decreasing function of smoking prevalence in society. This phenomenon can emanate in two possible ways. Parents may either give up on socializing their children when the outside threat becomes greater, or the threat of the unwanted trait might be decreasing with its prevalence in society.

We also impose one of two assumption on the horizontal transmission process: *cultural conformity* or *cultural distinction*. While cultural conformity has been an implicit feature of existing models on cultural transmission, cultural distinction has been largely overlooked by theoretical studies. In a recent paper, Bisin et al. (2013) call cultural conformity (distinction) the drop (increase) in psychological costs of interacting with a member of an outside cultural group when that outside group becomes more dominant. They use those notions to understand the implications of ethnic identify on marriage outcomes in a cultural transmission framework. We, however, define those concepts differently:

Assumption H1 (Cultural conformity): $S(q_t)$ is increasing in q_t.

Assumption H2 (Cultural distinction): $S(q_t)$ is decreasing in q_t.

A large literature on identity formation in psychology, sociology, and political science argues that cultural or ethnic minority groups may either pursue cultural assimilation into the majority population or they may strive to keep their distinct identities.[3] We follow this reasoning to assume that the child culturally distinguishes himself when he horizontally adopts the minority culture, and he culturally conforms when he horizontally adopts the majority culture. Thus, cultural distinction predicts that the greater smoking prevalence is in society, the less likely the child is to adopt this trait. Cultural conformity makes the opposite prediction, that the greater smoking prevalence is in society, the more likely the child is to adopt this trait.

As we noted above, our definition of cultural conformity has been implicitly assumed in the literature as part of the horizontal socialization. Existing work has thought of $S(q_t)$ as representing a *matching process*, i.e. the likelihood a child is matched to a smoker role model from the population of adults (random-matching (e.g. Bisin and Verdier 2001; Bisin et al.2013); non-random matching (e.g. Bisin et al. 2004; Saez-Marti and Sjogren 2008)).[4] This matching process must satisfy two basic properties: (i) A child can only be matched to a non-smoker/smoker when there are only non-smokers/smokers ($S(0) = 0$ and $S(1) = 1$), and (ii) the likelihood of being matched to a smoker increases with the proportion of smokers in society (i.e. our definition of cultural conformity). As we will show in the empirical section of this paper, there is strong support for the possibility of having cultural distinction, which cannot be accommodated by existing models. More importantly, the study of dynamics in Section 3.5 reveals that under cultural substitution, assuming $S(0) = 0$ leads to the disappearing of the smoking culture in steady state, which is at odds with real-world behavior. The mechanism for horizontal transmission that we propose addresses this gap in the theoretical literature.

[3] See Bisin and Verdier (2011) for a survey of this literature and a list of references.

[4] Saez-Marti and Sjogren (2008) refer to as conformism a non-random matching process that favors the dominant trait. Their definition of conformism thus differs from both our and Bisin et al. (2013)'s definition of cultural conformity.

3.3 Endogenous vertical transmission

We now explicitly define the likelihood that a child follows his parent's desire to not smoke as a function of parental socialization. We find the optimal level of parental investment, and derive comparative statics for the effect of smoking prevalence and health costs of smoking.

As before, the subscript $i \in \{0,1\}$ denotes the adult's smoking behavior. When the child is born, his parent decides how much to invest in his anti-smoking socialization, $\lambda_i \geq 0$. The cost of socialization is $c(\lambda_i)$, and causes the child to become a non-smoker with probability $v(\lambda_i)$. With remaining probability $1-v(\lambda_i)$, the child becomes a smoker with probability $S(q)$, and a non-smoker with probability $1-S(q)$.

We normalize a parent's utility from not seeing her child smoke to 0. We then denote by $u_i(H) < 0$ the utility of having a child who smokes, where H represents the *perceived* (but not necessarily actual, $\overline{H}$) detrimental health effects of smoking. All parents alike value their children's health status, and so $u_i(H)$ decreases with the perceived health costs of smoking. Formally, an adult's utility function can be written as follows:

$$U(\lambda_i; q, H) \equiv (1 - v(\lambda_i)) S(q) u_i(H) - c(\lambda_i),$$

where a parent's investment is associated with a probability $(1-v(\lambda_i))S(q)$ of seeing her child smoke. Proposition 1 gives the optimal interior investment in the anti-smoking acculturation of a child.

Proposition 1: *Let $v(.)$ be a differentiable, increasing and concave function, and $c(.)$ be a differentiable, increasing and convex function. When interior, a parent's optimal investment choice λ_i^* solves*

$$\frac{v'(\lambda_i^*)}{c'(\lambda_i^*)} = \frac{1}{-u_i(H) S(q)}. \tag{1}$$

The assumptions on $v(.)$ and $c(.)$ are standard to ensure the existence of a unique solution to the maximization problem. They state that (i) more effort leads to a greater likelihood (cost) of dissuading the child from acquiring the unwanted behavior, and (ii) the effectiveness (cost) of this investment decreases (increases) in the amount of effort that is invested. The optimal investment choice simply equates the marginal cost of investing to its expected marginal benefit. The assumption that $u_i(H) < 0$ for $i = 0, 1$ makes it not optimal for any parent to invest in pro-smoking culturalization, as this would promote the parent's undesired trait. Had we assumed instead that smoker parents perceive a non-negative net utility from smoking, $u_1(H) \geq 0$, we would be in the world of Bisin and Verdier (2001) where each type of parent promotes her own trait. Ceteris paribus, this possibility is more likely to arise when the health costs of smoking are small, since both parents perceive children's utility to be decreasing the more harmful the smoking habit is. From the result in Proposition 1, we can then derive comparative statics on the investment choice:

Proposition 2: *Performing comparing statics on the optimal investment derived in Proposition 1, we obtain the following predictions:*

- *Prediction 1: Parents who perceive* larger *health costs from smoking invest* more *in anti-smoking culturalization.*
- *Prediction 2: We have cultural substitution iff cultural conformity holds, and cultural complementarity iff cultural distinction holds.*

Prediction 1 is straightforward; it states that parents invest more in the anti-smoking culturalization of their children the more serious the health costs of this habit. Prediction 2 says that vertical socialization follows cultural substitution when horizontal socialization follows cultural conformity, which is a well-known theoretical result in the literature, starting with Bisin and Verdier (2001). It implies that when a higher smoking prevalence increases the likelihood that a child smokes, it is more worthwhile for the parent to exert socialization effort. However, Prediction 2 also says that vertical socialization follows cultural complementarity when horizontal socialization follows cultural distinction, which is a result that is not emphasized in the literature. It implies that when smoking prevalence decreases the likelihood that a child smokes, it also lowers the value of socialization to the parent. Patacchini and Zenou (2011) produce the opposite finding in a cultural transmission model augmented with peer effects at the vertical transmission stage. Their result requires assuming that both the cost and marginal cost of socialization are increasing in the proportion of smokers. Like the rest of the literature, we instead favor a framework where society only plays a role through horizontal transmission; i.e. in the event that the parent is unsuccessful at socializing her child to the desired trait.

3.4 Endogenous horizontal transmission

To complete the model, we explicitly define the likelihood that the child adopts smoking through non-parental channels. Before parents decide how much to socialize their children, a profit-maximizing monopolist chooses a level of investment θ into increasing the appeal of smoking to the youth. Such a feature could arise, for example, through celebrity endorsements of this habit. Advertising θ can be thought of as stimulating "demand", but to have diminishing marginal returns, so that $\frac{\partial S(q,\theta)}{\partial \theta} > 0$ and $\frac{\partial^2 S(q,\theta)}{\partial \theta^2} < 0$.[5] The function $\kappa(\theta)$ represents the convex cost of advertising ($\kappa' > 0$ and $\kappa'' > 0$). Keeping the firm's investment constant, we assume that $S(.)$ can still be thought of as a matching process, so that $\frac{\partial S(q,\theta)}{\partial q} > 0$. We finally impose a regulatory condition to ensure an interior level of investment for the firm, $\frac{\partial S(0,\theta)}{\partial \theta} > \kappa'(0)$. Proposition 3 describes the equilibrium of the game implied between the monopolist and parents.

[5] We put this word in quotation marks given that children do not make any active choice in the cultural transmission framework.

Proposition 3: *In a subgame perfect equilibrium, we have* $S(0,\theta^*(0)) > 0$, *which ensures a heterogeneous distribution of traits in steady state. Moreover, both cultural substitution and cultural conformity may obtain.*

$S(0,\theta^*(0)) > 0$ follows directly from assuming an interior solution to the problem. It ensures that the tobacco company would always want to invest a positive amount in making the smoking habit emerge. The second part of the result comes from a study of the sign of $\frac{dS}{dq}\Big|_{\theta^*} = \frac{\partial S}{\partial q} + \frac{\partial S}{\partial \theta}\Big|_{\theta^*} \frac{\partial \theta^*}{\partial q}$. We have cultural complementarity whenever this quantity is positive, and cultural substitution otherwise. While determining the sign of this quantity involves the interaction of numerous terms, it is possible to talk loosely about when each possibility obtains using the findings in Proposition 2. For example, cultural distinction obtains when: 1) an increase in smoking prevalence decreases the value of the investment sharply ($\frac{\partial^2 S(q,\theta)}{\partial \theta \partial q}$ negative enough), or 2) smoker parents are less tolerant of their children becoming smokers ($a > d$). The main take away message is that both cultural conformity and cultural distinction are realistic possibilities. We leave it to the data to tell us when each obtains.

3.5 Dynamics

We summarize the period $t+1$ transmission process into a transition matrix $P_t \equiv \begin{bmatrix} P_{t0} \\ P_{t1} \end{bmatrix}$, where $P_{t0} \equiv (1-d(q_t))\,S(q_t)$, $(P_{t1} \equiv (1-a(q_t))\,S(q_t))$ gives the proportion of children of non-smokers (smokers) who adopt smoking. This matrix is subscripted by the time period t since transmission is a function of the parents' smoking behavior, which is acquired in their youth. We can represent the evolution of smoking in society through the equation

$$\dot{q} = P_t^T \begin{bmatrix} 1-q_t \\ q_t \end{bmatrix} - q_t, \tag{2}$$

where the superscript T indicates the transpose of a matrix. The following result describes the steady state behavior of the system.

Proposition 4: *In a steady state, there always exists a fraction of non-smokers. Moreover, under cultural complementarity the smoking habit always persists, while under cultural substitution it persists as long as* $S(0) > 0$.

Under cultural complementarity, we must have cultural distinction (Proposition 2), and therefore children always have the proclivity to reject the status-quo so that neither traits disappear in steady state. Under cultural substitution, smoking never becomes the unique trait since parents have an incentive to prevent this from happening. In contrast, smoking may disappear if no outside factor forces it to persist. Given Proposition 3, we know that the existence of a tobacco industry can guarantee the coexistence of both traits under this scenario.

4 Empirical strategy

We use the following baseline specification:

$$\begin{aligned} Pr(ever\ smoke = 1)_c &= \alpha_0 \\ &+ \alpha_1 * Pr(socialization = 1)_p \\ &+ \alpha_2 * sm.prevalence\ of\ role\ model\ population_{cs} \\ &+ \sum_j \alpha_{3j} * X_{jcs} \\ &+ \nu_c \end{aligned} \tag{3}$$

$$\begin{aligned} Pr(socialization = 1)_p &= \beta_0 \\ &+ \beta_1 * exposure\ to\ health\ information_p \\ &+ \beta_2 * sm.prevalence\ of\ role\ model\ population_{cs} \\ &+ \sum_j \beta_{3j} * X_{jcs} \\ &+ \upsilon_c \end{aligned} \tag{4}$$

Equation (3) is the empirical counterpart of P_T, as described in the previous section. It is a structural form equation that treats $Pr(socialization = 1)$ as the endogenous regressor. Equation (4) is the empirical counterpart of (1). In this first-stage equation we identify parental socialization using different indicators of the parent's *exposure to health information* as instruments. X denotes exogenous control variables; α denotes a structural parameter; and β denotes a reduced-form parameter. Indexes c and p stand for child and parent, respectively; s stands for state; and j identifies each characteristic (individual, parental, or state) that we include as a control variable. Finally, ν and υ are the jointly distributed error terms.

We estimate (3) and (4) as a system by IV probit, even though this method is meant to be used when the endogenous regressor is continuous rather than binary. Because Heckman's (1978) maximum likelihood bivariate probit was built to accommodate binary endogenous regressors, it would have been more appropriate to use in our case. However, we choose not to use it because it is computationally cumbersome,[6] and it does not significantly outperform IV probit or even IV linear probability models in terms of accuracy (see Nichols 2011 and references therein). To confirm the latter point, we test the robustness of our baseline specification to a range of alternative estimation methods, including the bivariate probit.

To statistically identify exogenous variation in parental socialization we assume that, controlling for the child's own exposure to anti-smoking articles, we can exclude parental exposure to health information as a direct determinant of the child's decision to smoke. To test this exclusion restriction, we calculate the Amemiya-Lee-Newey

[6] Researchers find that to run a bivariate probit often takes 10 or 20 times as long as other similar models, and that standard statistical software like Stata and R frequently fail to find the maximum of the likelihood (e.g. Freedman and Sekhon 2010)

(ALN) minimum χ^2 statistic under the null that the instruments are valid (i.e. uncorrelated with the error term) and correctly excluded from the outcome equation.[7] To test whether our instruments have weak explanatory power, we calculate the χ^2 statistic under the null that the instruments are jointly statistically insignificant in the reduced form. We also calculate the Hausman χ^2 statistic to test whether there is a statistically significant difference between IV probit and probit (naive) estimates. The null of this test is that the probit model provides both consistent and efficient estimates while IV probit estimates are only consistent, and that the difference between the two is normally distributed with mean zero. Further, we calculate the Wald χ^2 statistic to test the null that the correlation coefficient between ν and υ is zero and, therefore, $Pr(socialization = 1)$ can be treated as exogenous. Finally, we check the robustness of our baseline estimates to the inclusion of a wide range of controls and instruments.

To statistically identify the social transmission of the smoking trait we rely on the fact that the smoking prevalence of the role-model population is measured at the state-level. Because we can plausibly assume that state-specific smoking prevalence is exogenous to the parental choice of the state of residence and it cannot be affected by endogenous peer-choice, we rule out selection and simultaneity bias from the estimated effects. Bias due to exogenous correlated effects, however, remains a possibility (Manski 1993, 2000). The smoking prevalence of the role-model population is the aggregation of individual behavior which (depending on how the role-model population is defined) may include the parent or the child. Thus, our estimate of α_2 may reflect the fact that individuals in a given state have similar smoking behavior because they have unobserved similar characteristics or because they are exposed to the same institutional or contextual factors ('Manski's reflection problem'). To account for such unobserved common factors, we follow the health economics literature and use a fixed effects specification (see, for example, Nakajima 2007 and references therein). Because, as we describe below, the smoking prevalence of the role model population varies by state and child age, we include a full set of state and age fixed-effects. We thus identify causality of the social effects by using variations in the proportion of smokers between age-groups within a state.

We use the results to assess whether socialization by parents and role models affect a child's smoking decision, to identify the relative contribution of the two types of socialization to the transmission of the smoking culture, and to test important properties of the transmission process; namely, cultural substitution versus complementarity, and cultural conformity versus cultural distinction. Finally, we use the structural parameter estimates to forward project how the rate of ever-smoking of 10-18 year olds will evolve under different policy scenaria.

[7] The Amemiya-Lee-Newey test is only possible after running the two-step Newey (1985) IV probit estimator. All other results we present in this paper are derived using the maximum likelihood IVprobit estimator.

5 Data

Our empirical analysis exploits new as well as existing data in novel ways. Below we discuss the source and construction of each type of data. Table 1 provides summary statistics of selected variables.

5.1 Individual level data on children and care-givers

We draw individual level data on children aged 10-18 from the 2002 and 2007 waves of the Panel Study of Income Dynamics (PSID) - Child Development Supplement/Transition to Adulthood (CDS-TA) surveys, and supplementary data on parents of each child from the main family files of the PSID. The CDS-TA sample was originally drawn from PSID families with children 0-12 years in 1997 and reinterviewed in 2002 and 2007. We use sample weights provided by the PSID to account for unequal probabilities of being selected into the CDS sample and for differential attrition rates of PSID and CDS participants. The 2002 and 2007 wave of CDS-TA directly surveys children age 10 and older about tobacco use and other types of behaviors. These data were collected by an audio computer assisted self-interview. In such interviews, youth listen to the questions through a headset and record their responses directly into a laptop computer. Neither the child's parents nor the interviewer knew how s/he answered the questions. Aquilino (1994) documents that this method generates more accurate data on socially sensitive topics such as psychological well-being, sexual behaviors, and experiences with tobacco, alcohol, and drug use. We use the data on whether a child ever smoked (defined on the survey as smoking at least 1 cigarette every day for 30 days) to represent $Pr(ever\ smoke = 1)_c$. Eighteen percent of our sample smoked at some time between age 10 and 18.

The CDS-TA surveys asks questions not only of the child but also of the person in the PSID household who identified herself/himself as the 'primary' care-giver (PCG) of that child. Table 1 documents that biological mothers comprise 93 percent of self-identified care-givers, six percent of care-givers are biological fathers, and the rest are adoptive mothers or step-mothers. Because of the disproportionate share of biological mothers in our sample, we cannot separately model how cultural transmission varies with the nature of the parent-child relationship.

While the CDS-TA surveys do not ask questions that are specifically about smoking socialization efforts of the PCG, the surveys do collect information that is likely to proxy for it. Ideally the surveys would ask each PCG to report how much effort she spends socializing her child about the health risks of smoking. Instead the CDS/TA surveys asked each care-giver to report how frequently during the past month she talked to each child about the dangers of substance use (e.g. drinking alcohol or taking drugs). The survey specified five response categories that ranged from "not in the past month" to "every day." While these data are not ideal, we expect answers to them to be correlated with the conceptual variable of interest. In addition, we identify variation in the pattern of responses in these data using variation in information that is specifically about health risks of smoking. As a

result, though imperfect, the CDS data are likely to proxy well for the conceptual variable of interest. Table 1 shows that 21 percent of care-givers reported they had not spoken with their child about the dangers of substance use in the past month. Most (42 percent) care-givers discussed this subject once or twice per month. However, 37 percent of care-givers discussed substance use at least once a week during the previous month.

In addition to the socialization data, we draw CDS-TA data on age, sex, race, and religion of the child; household income; family size; and measures of parenting styles, reading habits, and employment status of the PCG.

We also draw data on the smoking behavior and educational attainment of the PCGs from the main PSID files. We use data on smoking behavior from the 1986, 1999, 2001, 2003, 2005, 2007, and 2009 family files and data from a special 1990 questionnaire administered to all PSID household members over the age of 55 to construct our measure of whether a parent ever smoked. Finally, we draw data on years of completed schooling from all waves of the PSID. In our sample, 45 percent of PCGs smoked at some point in their lives and the average PCG has completed 13 years of schooling.

5.2 Smoking prevalence rates of the role model population

To construct measures of role-model smoking behavior, we use data from the Tobacco Use Supplements to the Current Population Survey (TUS-CPS). Sponsored by the National Cancer Institute and administered as part of the U.S. Census Bureau's continuing labor force survey, the TUS-CPS data have been collected intermittently since 1955. We use responses from 21 surveys conducted in August 1967, August 1968, September 1989, September 1992, January and May 1993, September 1995, January and May 1996, September 1998, January, and May 1999, June and November 2001, February 2002, February, June, and November 2003, May and August 2006, and January 2007. Each survey asks respondents: "Have you ever smoked regularly?"; "If yes, what is the age when you started?"; "Do you currently smoke?"; and "If not, what is the age when you last smoked regularly?".

After dropping multiple observations for each individual across monthly waves of the same calendar year, we pool all data from these waves and use the smoking questions to construct the smoking history of every TUS-CPS respondent. To do this we identify all respondents who ever smoked and who report a start age, a current smoking status, and a quit age (former smokers only). We then assume that a person smoked in every year between the age she started and either the age at the survey date (current smokers) or the age she quit (former smokers). Because in each calendar year our sample includes all respondents who were alive in that year and retrospectively answered the smoking questions in any later year, we start with an enormous sample of current, ever, and never smokers (approximately 81 million observations). We combine our computed smoking life-histories with data on the state of residence (at the time of the survey) to construct smoking prevalence rates by sex, cohort, state, and calendar year (weighted by the CPS sampling weights).

To match our empirical specification as closely as possible with our theory, we assume that a child's role model is drawn from his parent's generation. This assumption means that we create smoking prevalence rates of the role model population using males and females who are 20-29 years older than the child.[8] Table 1 shows that among the potential social role models of children in our sample, the average smoking prevalence rate is 26 percent. Later in the analysis we experiment with different definitions of the role-model population (by age and sex).

To illustrate the variation in the data we develop, Figure 1 plots the smoking rates by sex, state, and calendar year for the social role models of children who are age 14 at the time of the survey (i.e. at mean age). While these data have rich variation across all dimensions, the variation we can exploit is limited because we only observe a child's smoking behavior and his parent's socialization effort in 2002 and 2007. Despite that limitation, plenty of variation remains available to us: across states, gender, and by child age. Figure 2 showcases the data that we actually use in the analysis.[9] Clearly, the smoking rates vary significantly by gender and state, but note that, because the state-specific curves cross, the rates also vary by age of the child/parent generation.

5.3 Information about the health risks of smoking

To instrument parental socialization we use temporal and geographic variation in exposure to information about the health risks of smoking. The basic data consist of counts of articles published between 1924 and 2009 that warn readers about the health risks of smoking. We use counts of articles published in each of more than 21 popular consumer magazines. To generate additional geographical, but also temporal variation, we also exploit data on the number of issues of each magazine that were sold in each state in each year.

The data on articles were generated by first searching two electronic databases (ProQuest and the Historical Reader's Guide to Periodical Literature) using a keyword search on "smok* and cancer," "smok* and health," "cig* and cancer," "cig* and health" and similar text strings. Successive searches produced roughly 5,000 titles of articles published between 1890 and 2009. Two undergraduate research assistants then independently reviewed all 5,000 titles to identify articles that potentially warned about the health risks of smoking. This review eliminated roughly 2,500 articles that focused on the effect of the health risk information on financial returns of tobacco companies, tobacco growing agriculture, and international trade in tobacco. The remaining set included articles whose titles suggested that the articles discussed content about risks individuals faced. A team of research assistants collected copies of all articles and read them. Two of them independently rated the articles as a) "pro-smoking," b) "neutral," and c) "anti-smoking" when they judged that an article conveyed to readers the impression that smoking a) improved, b) did not affect, or c) degraded the health of smokers. Any disagreement was discussed

[8] i.e. we assume an average generation gap of 25 years, which is slightly smaller than the average generation gap that we observe in our data (28 years).

[9] Note that instead of 2007 data we use data that correspond to 2006 because the 2007 survey was conducted in January. Aside from the fact that the sample is smaller, smoking behavior in January does not represent average smoking behavior throughout the year because more people quit smoking in January to implement a New Year's resolution. Many of these people fail in their resolution. Consequently, smoking prevalence rates in January are lower and more widely distributed than smoking prevalence rates over the whole year.

and resolved. The resulting list of articles generated a list of magazines in which the articles appeared.

We then compiled data on sales of each of those magazines in every state in each year. We got the sales data from the Audit Bureau of Circulation. The Audit Bureau of Circulation is an organization that publishers voluntarily join. Its sole purpose is to audit and verify circulation figures the publishers provide to them. Their independent auditing provides a valuable service to publishers because they charge advertisers more for space in more widely circulated magazines. Advertisers therefore demand (and publishers willingly provide) an independently verified count of circulation. The magazine circulation data vary by month, year, and state.

We assume that, when a magazine is sold, it is seen by all members of the household in which the purchaser resides.[10] To capture this exposure, we divide estimates of each state's population from the Current Population Reports of the Census Bureau by 2.3 (the average household size) and divide the number of issues sold in each state in each year by that number. The resulting figure is an estimate of the fraction of each state's population that read each magazine (in each year). We then multiply the fraction of each state's population that read each magazine by the number of articles that appeared in that magazine. This step yields the exposure of a randomly drawn person from a given state to an article that appeared in a given magazine in a given year. Finally, we sum across all magazines in which an anti-smoking article appeared. The final data proxy for the total potential exposure to anti-smoking magazine articles in a given state in a given year. Currently we compute this sum using articles that appear in 21 magazines that accounted for 70 percent of all anti-smoking magazine articles produced by the above searches. Formally our measure is given by:

$$\text{Anti-smoking articles read}_{st} = \sum_{m=1}^{21} \text{Articles}_{mt} \frac{\text{Issues}_{mst}}{\text{Population}_{st}/2.3} \tag{5}$$

where s denotes state, t denotes calendar year, and m denotes each of 21 magazines.

Figure 3 plots the resulting measure for all states between 1929 and 2009. We use these data to compute several alternative measures of exposure to anti-smoking information of both PCGs and children. The two measures we select to use in the baseline specification are: (i) the accumulated sum of articles (potentially) seen by the PCG or the child since age 10, which should capture the degree of exposure; and (ii) the standard deviation in the articles read since age 10, which should capture the *in*frequency of the information flow. Similar measures have been shown to predict changes in consumption of fats and oils as information developed and spread about the health risks of consumption of saturated, monounsaturated, and poly unsaturated fats (Chern et al. 1995). We start counting exposure from age 10, assuming that it is the earliest age a child can comfortably read. Year 1929 is the earliest year in which a PCG in our sample was age 10, and year 1994 is the earliest year in which a child was age 10. Thus, our measures of exposure for PCGs (i.e. our instruments) encompasses all temporal and state variation illustrated in Figure 3. In contrast, our equivalent measure for children (which we use as a control) encompasses

[10] In fact we only assume a household member *potentially* sees the article. From now on we use the terms "exposure" and "potential exposure" interchangeably.

the variation illustrated in the shaded area only. The 'typical' PCG in our sample has been exposed to about 12 anti-smoking articles since age 10, while the corresponding number for the 'typical' child is 4.

5.4 Indicators of the economic environment

In our regressions, we control for time-varying state-specific economic factors that may affect the child's probability to smoke or the PCG's socialization efforts. Specifically, we control for state and federal cigarette taxes and state per-capita income. We use the measure of 'full' taxes on cigarettes described in Lillard and Sfekas (2013). This measure is the sum of the state and federal cigarette tax and the per pack escrow payments that are required on the 1998 "Master Settlement Agreement" between the four major cigarette manufactures and the US states. Viscusi and Hersh (2011) and Lillard and Sfekas (2013) document that this payment is functionally equivalent to a per pack cigarette tax. We draw data on real per capita state income from the Regional Economic Information System of the Bureau of Economic Analysis in the U.S. Department of Commerce (SA05 series) and adjust them in units of real 2008 dollars. To illustrate the variation in these two variables, we plot them by state and year in Figures 4 and 5.

6 Results

6.1 Vertical versus horizontal transmission of the smoking trait

The naive way to think about the cultural transmission of the smoking trait would be to assume no reverse causality between parental socialization efforts and the childrens' decision to smoke. Table 2 presents estimates of equation (3) under this assumption; that is, by treating $Pr(socialization = 1)$ as exogenous. In column 1 we use the socialization data in their 'raw' form; i.e. in five categorical dummy variables: not in the past month (the reference category), once/twice a month, once a week, several times a week, and daily. In columns 2-5 we dichotomize the data into a single dummy to denote that children are socialized with 'at least' a given level of frequency (at least once/twice per month, at least once a week etc.). The resulting estimates vary across the combinations. In column 1, PCG socialization efforts are associated with a higher probability that the child ever smokes, even though that association decreases as the frequency of socialization increases. The estimates in column 2 also suggest a positive association, while columns 3-5 suggest a weak negative association (which in 3 and 5 is statistically insignificant). The probable cause of these counterintuitive results is the endogeneity of the socialization variable. Children may be less likely to smoke when their parents socialize them against it, but it is also likely that a PCG will discuss substance use more often if a child smokes or the parent suspects a child is likely to smoke. To isolate the former effect we abandon the naive approach.

Table 3 presents our baseline estimates of jointly determined equations (3) and (4), as described in Section

4.[11] To simplify the analysis, we dichotomize the socialization variable, $Pr(socialization = 1)$, to indicate the probability that the PCG socializes the child about the dangers of substance use at least once a week.[12] As we mentioned earlier, we instrument the PCG's socialization efforts with the parental exposure to smoking-related health information since age 10 and the infrequency of that exposure. In contrast to the results in column 3 of Table 2, the coefficient on the instrumented socialization variable is negative and statistically significant, suggesting that a parent's effort to socialize her child is effective. The performance of the instruments in the first-stage equation is satisfactory: they are statistically significant and the sign of their coefficients are in the direction described in Prediction 1. Those PCGs who are exposed to more anti-smoking articles on average also exert more effort to socialize their children. Holding the average level of exposure constant, PCGs socialize their children less if their exposure varies more over time. That is, PCGs socialize their children more when they are exposed to a constant stream of information about the health risks of smoking compared to PCGs who see the same number of articles on average but who see no articles in some years and many articles in others. The diagnostic test results corroborate the good performance of our instruments and of the baseline specification in general.

We should note that a factor contributing to instrument validity is that the baseline specification controls for the child's exposure to anti-smoking articles since age 10. Again, the estimated coefficient on this variable makes economic sense: a higher information exposure of the child is associated with a lower probability that the child ever smokes and with a lower socialization effort by the PCG (as s/he now relies on the external information to do the job). Although the statistical significance of these effects is somewhat weak, it is important to mention that removing the measure of child exposure from the estimation significantly impacts the ALN test of over-identification. In this case, the χ^2 statistic increases to 3.255 and the probability values drops to 0.071, so that we reject the hypothesis that the instruments are valid at the 10% level of significance.

In all regressions reported in Tables 2 and 3, the probability that the child ever smokes increases with the state-specific smoking prevalence rates of the role-model population, providing evidence for cultural conformity. In the first-stage of Table 3, the socialization effort of PCGs also increases with the smoking prevalence in the role model population and it is statistically unrelated to parental smoking status, providing evidence for cultural substitution. The co-existence of cultural substitution and conformity supports Prediction 2 of the model. It suggests that all parents wish to discourage smoking and, because they know that their children will conform to societal trends, they will increase their anti-smoking socialization efforts when smoking becomes more popular in society (we expand this discussion in Section 6.3).

[11] From this point onwards all regressions are estimated by IV probit. See Table 4 in the Appendix for a set of robustness test of the baseline estimates to alternative methods of estimation.

[12] We dichotomize the socialization variable because the coefficients on the different versions of the socialization variable presented in columns 2-5 of Table 2 suggest that socialization categories "once a week", "several times per week" and "every day" produce results that are similar among them but much different to the results produced by category "once/twice a month". This implies that the response distribution we observe could be a mixture of two distributions, each capturing a different kind of decision process. Nonetheless, our baseline results show low sensitivity to the definition of the dichotomized socialization variable. See Table 5 for the robustness analysis. In a similar exercise, Patacchini and Zenou (2011) use the frequency that a parent reads to a child to capture parental effort to cultivate interest in education to the child. They dichotomize their socialization variable the same way.

To compare the importance of the parental (vertical) and social (horizontal) channels of transmission of the smoking habit, we calculate marginal effects of 'equivalent' changes in parental socialization efforts and the smoking prevalence of the role model population on the probability that the child smokes. Clearly, defining changes of equivalent magnitude in two completely different variables is a challenge. We take a 'let the data speak' approach and allow both variables to increase by half their standard deviation. This corresponds to a 9.8 percentage point increase in the share of parents who socialize their children at least once a week (from 41.6% to 51.4%)[13], and to a 2.6 percentage point increase in the smoking prevalence rate of the role model population (from 26.3% to 28.9%). We find that these changes cause the likelihood that the child smokes to decrease by 3.9 percentage points and to increase by 2.8 percentage points, respectively. The implication is that parental influences are stronger than social influences in the determination of youth smoking participation. Note that we reach this conclusion without taking into account the direct effects of parental smoking behavior on the probability of youth smoking participation (e.g. due to genetics, mimicking, nicotine addiction from passive smoking, or easier access to cigarettes). Our results suggest that this channel of transmission is also important; the child is more likely to have ever smoked when the PCG has ever smoked. The reported marginal effects, therefore, understate the true effect of the parental channel of transmission.

All regressions in Tables 2 and 3 control for the PCG's education, family income, state cigarette tax, state income, and a wealth of demographic variables.[14] The signs of the estimated coefficients on all these variables are in the expected direction and consistent with empirical findings in the health economics literature. The child is more likely to have ever smoked when the PCG is less educated, when family and state income is low, and when cigarette taxes are low. The PCG is more likely to socialize the child at least once a week when s/he is highly educated, when family and state income is higher, and when cigarette taxes are higher (e.g. because, when taxes are high, smoking by the child entails a higher financial cost for the entire family). From this point on, we do not show estimated coefficients on the control variables, but rather focus on the variables of interest.

6.2 Robustness analysis

Although the baseline specification is already very conservative, there are reasons that induce us to test its robustness to new instruments and controls. First, the performance of our instruments may be impaired by their limited variation; e.g., our measure of PCG exposure to health information does not vary across PCGs who live in the same state and were born in the same year. We could benefit from an exposure measure that is parent-specific and adds individual-level variation to our instrument. Second, our definition of the exposure measure (i.e.

[13] Because the parental socialization variable is binary, we calculate its standard deviation (0.19) using its estimated value from the reduced-form regression. To induce an increase in this variable that corresponds to half of its standard deviation, we had to randomly shift PCGs from category 0 to category 1 so that the mean of the variable increases by 9.8 percentage points. This is mathematically equivalent to increasing the probability of socialization for those parents who do not socialize their kids at least once a week from 0 to 0.17 (=(0.19/2)/(1-0.416)).

[14] To save space, we do not present coefficients on the age fixed effects of the PCG and the age, sex, state, race, and religion fixed-effects of the child. However, full results for all models are available on request.

as the accumulated sum of anti-smoking articles) imposes the restrictive assumptions that information does not decay and that it has constant returns to scale. More flexible specifications of the exposure measure may be more appropriate. Third, in our baseline specification we do not account for differences in the personality of children or in parenting styles. Both of these have been shown in the psychology literature to vary with children's smoking behavior and the effectiveness of parental socialization efforts (e.g. Huver et al. 2007). Fourth, in our baseline specification we do not control for differences across PCGs in the cost of the parental socialization efforts. We next try to address these issues using a new set of variables which Table 6 presents along with some basic descriptive statistics.

Our first exercise aims at introducing individual-level variation in our set of instruments. To do this, we draw from the PSID-CDS-TA a variable that measures how often the PCG reads the newspaper during the week. Assuming that this variable is highly correlated with magazine readership, we interact it with the PCG's information exposure measure to generate a new variable that varies across PCGs. Column 1 of Table 7 tests the robustness of the baseline specification to this inclusion. The results are highly robust, and the new instrument appears with a positive and significant coefficient, suggesting that anti-smoking information is more effective at increasing parental socialization efforts when parents have the habit of reading the newspaper often. In comparison to the baseline results, the diagnostic test results are slightly improved (e.g. the probability value of the the ALN test statistic increases to 0.578).

As a further robustness test, we define information exposure to be the average number (instead of the accumulated sum) of anti-smoking articles that the PCG or the child potentially read since age 10. This specification essentially decreases the contributing value of each anti-smoking article by a factor proportional to the age of the PCG, so that (older) PCGs who see a given number of articles over a longer life-span end up with a lower information exposure score than (younger) PCGs who see the same number of articles over a shorter life-span. In other words, the new exposure measure allows the value of information to decay over time. Columns 2 and 3 of Table 7 show how the baseline estimates change when we replace the original exposure measure with this new measure, and when we interact it with the frequency the PCG reads the newspaper. Although the results are qualitatively robust to this change and the instrument coefficients are positive and statistically significant, the tests of instrument identification reject the hypothesis that the instruments are valid.

Finally, in Table 8 we check the robustness of the baseline estimates to controls that capture personality traits of the child and parenting styles of the PCG (column 1), to controls that capture the cost of PCG socialization efforts (column 2), and to both set of controls together (column 3).

The first set of controls includes: (i) an indicator of whether the child has a tendency to "break rules", which we create by combining a selection of variables documenting "problematic" past behavior (see note of Table 6 for the exact definition); (ii) an indicator of high and strict parental control, which we create by combining responses of the PCG to questions about the number of rules s/he impose on the child, whether these rules are strictly

enforced, and whether s/he discusses these rules with the child; (iii) an indicator of violent parenting, which flags whether the child is spanked more than 3 times per week; and (iv) an indicator of complete lack of communication among the PCG and the child. Controlling for these variables is potentially important because it may further address the problem of reverse causality between socialization efforts of the PCG and the smoking behavior of the child. Rebellious children or children subject to authoritative parenting may react against parental anti-smoking pressure and may be more likely to smoke when the socialization efforts of the PCG are more frequent. We find that the these controls significantly predict the dependent variables in both the structural and reduced-form equation, while leaving the remaining coefficients almost unaffected. As expected, children who are "rule-breakers" and children who are spanked regularly are more likely to ever smoke and more likely to be socialized by PCGs. In contrast, children who are under strict parental control are less likely to ever smoke and are less often socialized by parents. Finally, children who never discuss any subject with their parents are more likely to ever smoke.

Our measures of the parental socialization cost include: (i) the number of individuals younger than 18 in the family unit; (ii) an indicator of whether the PCG is employed; (iii) the weekly hours that the PCG spends at work; (iv) an indicator of whether the PCG works a regular daytime schedule; and (v) an indicator of whether it takes the PCG over an hour to get to work each way. Once more, we find that, when included in the baseline regression, these variables significantly predict the dependent variables and only result in small quantitative changes in the estimated coefficients of the other variables. Children who live in households with many other children are less likely to smoke and more likely to be socialized by PCGs. Children of working parents are less likely to smoke but also less likely to be socialized, whereas children whose parents work more and regular hours are more likely to smoke and more likely to be socialized.

When we insert in the baseline specification all the new controls together we obtain similar results. All three specifications of Table 8 pass the diagnostic tests. In fact, the probability values of the ALM test for specifications (1) and (3) are higher in comparison to that of the baseline specification (0.860 and 0.648, respectively), suggesting that the addition of the controls aids identification.

6.3 The mechanisms underlying the transmission process

In the previous sections we found a positive coefficient on the smoking prevalence of the role-model population in both the reduced-form and the structural-form equations. We interpreted these findings as evidence for cultural substitution and conformity. In this section, we scrutinize these results by carrying out two exercises. First, we interact parental smoking status with the prevalence rate of the role-model population to inform our discussion of cultural substitution. Second, we use alternative definitions of the role-model population to inform our discussion on cultural conformity. Both exercises allow us to confirm the links between substitution and conformity and between complementarity and distinction. We conclude this section by further exploring differences in the socialization process between smoker and non-smoker parents.

6.3.1 Substitution versus complementarity

Assuming that both smoker and non-smoker parents wish to discourage smoking, cultural substitution entails that all parents should increase anti-smoking socialization efforts in response to the smoking rate in society. The first-stage estimates in columns 1 and 2 of Table 9 show that this prediction is supported by the data. We find positive and significant coefficients on all interactions of smoking prevalence and parental smoking status. We read this result to suggest that our extension of the Bisin and Verdier model is more appropriate to use when studying the transmission of traits like smoking than the Bisin and Verdier model in its original version. If smoker parents wished to transmit the smoking culture to their children, like the Bisin and Verdier model would assume, then we would expect to find evidence of cultural complementarity for smoker parents and cultural substitution for non-smoker parents (i.e. positive coefficients on the interaction terms in Table 9 for non-smoker parents, and negative coefficients for smoker parents). However, our results suggest otherwise.

As our model predicts, we find similar behavior across smokers and non-smoker parents because there is an exogenous force (anti-smoking information) that has changed people's perceptions of smoking from a 'good' trait to a 'bad' trait. As a result, parents wish to socialize children to be non-smokers, even if they themselves smoke. We follow this logic to develop another implication: we should find evidence of culture complementarity among smoker PCGs exposed to little (or very low) anti-smoking information because, absent other information, these would still consider smoking to be a good trait. In column 3 of Table 9 we attempt to test this hypothesis by interacting the parental smoking status with smoking prevalence in the role model population at different quantiles of parental information exposure (quantiles 0-10, 10-50, 50-90, and 90-100).

We note that, in our data there is no PCG who is subject to no anti-smoking information. All PCGs are exposed to some non-negligible level of information. In fact, the PCG at the 10th percentile of the distribution of our exposure variable saw 8 articles, relative to a mean of 12. Therefore, it comes as no surprise that we do not find evidence of cultural complementarity in our results (none of the estimated coefficients carries a negative sign). Nonetheless, there is enough variation in the data to reveal two important patterns that corroborate our theoretical set-up. First, when the smoking prevalence of the role model population increases, all parents respond by increasing their socialization efforts, and their response is larger the more exposed they are to information about the health risks of smoking. Second, this response is not statistically different from zero for smoker parents who are exposed to very low levels of information. These findings encourage us to speculate that, had we observed in our data parents with no or negligible information exposure, we would be able to document a switch in the direction of the relationship between parental socialization and the proportion of smokers in society from positive to negative.

6.3.2 Conformity versus distinction

To this point, all the results we have reported are based on the assumption that children derive their role-models from the population that is 20-29 years older. Next, we test whether the probability that the child ever smokes is associated with the smoking prevalence rate of the population of individuals 0-9 and 10-19 years older than the child, and whether that association differs by gender.[15] We present our results in Table 10. Estimates in columns 1-4 are based on the total sample, while estimates in columns 5 and 6 are based on data on male and female children, respectively.

The structural form estimates suggest that the probability that the child ever smokes increases with the smoking prevalence rates of all population sub-groups, except for males who are 0-9 years older. The implication is that, in relation to the bulk of the population, children form their smoking decisions based on their needs to achieve assimilation, inclusiveness, and cultural conformity. In relation to the sub-group of young boys, however, the childrens' motive is the exact opposite. In this case, it is their need to generate a sense of distinctiveness from individuals that are part of that group that motivates their smoking decisions. To put it bluntly, our results suggest that young boys operate as anti-role-models. Although this result might seem surprising, one should note that health economic studies on peer effects have not reached a consensus on gender differences in social influence. For example, Nakajima (2007) study peer effects of smoking among school-mates in the US and finds that these are positive and significant within the same gender but statistically negligible across genders. In contrast, Clark and Loheac (2007) study peer effects on different types of risky behavior among friends and school-mates in the US and find significant cross-gender interactions for alcohol use, with young males being more influential than young girls. While we are the first to provide evidence on cultural distinction using smoking data, our evidence complements those presented in the study of ethnic identity formation by Bisin et al.(2013). These authors find that, in neighborhoods in which the share of a given ethnic group is high, the association between the share of the ethnic group and individual ethnic identities is negative.

6.3.3 The link between substitution (complementarity) and conformity (distinction)

In both Tables 9 and 10, the smoking prevalence rates significantly predict the PCGs socialization efforts in the reduced form and the probability that the child smokes in the structural form. Importantly, these effects always run in the same direction, a result which reconfirms the inter-connection between cultural substitution and conformity and between cultural complementarity and distinction, and corroborates Prediction 2 of the model. This result suggests that, because all parents wish to discourage smoking, they will increase their anti-smoking socialization efforts when smoking becomes more popular among all societal groups to which their children will conform, and they decrease their anti-smoking socialization efforts when smoking becomes more popular among young boys

[15] Naturally, the alternative measures of smoking prevalence rates are correlated with each other, but there is still independent variation in the smoking prevalence rates across the different groups. See Table 11 for correlation coefficients and Table 6 for means and standard deviations.

from which their children will want to distinguish themselves.

It is worth noting that both our theory and findings contradict those produced by Patacchini and Zenou (2011), although they apply a comparable exercise to identify the cultural transmission mechanisms of preferences for education. Like our assumption that both smoker and non-smoker parents wish to transmit preferences against smoking to their children, Patacchini and Zenou assume that both educated and uneducated parents wish to transmit preferences in favor of education to their children. However, unlike our prediction that cultural complementarity is tied with cultural distinction, these authors predict that complementarity is tied with conformity. As we briefly discuss in section 3, this prediction relies on their assumption that a high prevalence of education in society creates positive externatilities in the effectiveness of parental socialization efforts by decreasing socialization costs (e.g. because more educated neighbors can help a less educated parent to better socialize the children). Their results support their theory. They find that all parents socialize their kids in favor of education, that their socialization effort increases with the prevalence of educational attainment in the population residing in the same neighborhood and, at the same time, the neighborhood education level increases the probability that children acquire education.

6.3.4 Other mechanisms

Also relevant is that the theoretical predictions of Patacchini and Zenou rely on the assumption that educated parents are more effective in socializing their children than uneducated parents because they face lower cost of socialization. Our model necessitates no particular assumption about the mechanisms underlying the vertical transmission process of smoker versus non-smoker parents. On the contrary, it allows many mechanisms to be at work at the same time. For example, smoker and non-smoker parents may or may not differ in the effectiveness of their socialization efforts, in their tolerance of having children who smoke, in their perception of the health-risks of smoking, and other dimensions. Whether or not each of these scenaria is true is an empirical question.

The evidence we present in Table 9 already shed some light on this issue. We find that never-smoker parents respond to the popularity of smoking in society by increasing their socialization efforts both more than ever-smokers (column 1) and more than current and ex-smokers (column 2). Further, we find that this difference persists at all levels of information exposure (column 3). We present more evidence in Table 12. There we show that never-smoker parents also have a higher responsiveness to health information both relative to ever smokers (column 1) and relative to current and ex-smokers (column 2). Both these findings suggest that smoker and non-smoker parents evaluate differently the health risks that their children face when they smoke, and are consistent with existing empirical evidence that non-smokers tend to overestimate the impact of smoking on health (e.g. Viscusi and Hakes 2008). In Table 12 we also show that never-smoker parents lower their socialization efforts when they work and increase their socialization efforts when their child is a rule-breaker less than ever-smokers (column 3 and 5, respectively). This may be because, unlike parents who smoke, never-smoker parents are more

effective at socializing their kids to be non-smokers by setting the right example and can, therefore, afford to lower their socialization efforts when socialization cost increases or when they have reactive children. Somewhat at odds with this interpretation is our finding that the responsiveness to the socialization cost does not statistically differ between never-smokers and current smokers (column 4), whereas the responsiveness to having a reactive child does not statistically differ between never-smokers and ex-smokers (column 6).

6.4 Forward projections of the share of children who ever smoke

As a final exercise, we simulate the dynamics of youth ever-smoking rates over time as described by equation (2) in section 3.5. To do this we use the estimated structural parameters from column 3 of Table 8 (i.e. our most restrictive specification), and the mean probability that a child ever smokes that we observe in our sample as the initial condition. This exercise serves two purposes. First and foremost, it shows that the smoking trait persists in the steady state of the population dynamics, it provides an estimate of the smoking rates at the steady state, and it provides an estimate of the time that smoking rates would need in order to adjust to that steady-state. Second, this exercise provides a framework that we can use to test how different policy regimes can affect the speed of adjustment of the youth smoking rates to equilibrium. We draw the projections in Figure 6.

The solid line shows how the youth ever-smoking rates would fare in future generations all else equal. It indicates that the share of youth ever-smokers converges to a steady-state value just above 11 percent.[16] This evidence confirms the long term persistence of the smoking trait, and is consistent with Predictions 3 and 4. Full convergence to that steady-state occurs within five generations (which are on average 25 years apart); i.e. for individuals that are born between 2108-2121. However, 90 percent of the adjustment occurs within only three generations; i.e. for individuals who are born between 2058-2071.

This trajectory can be altered by a social planner via several policy instruments. Here we give the examples of 'reasonable' increases in cigarette taxes and in parental socialization efforts during the lifetime of the children observed in our data. The dotted line shows how the smoking rates would fare under half a standard deviation increase in cigarette taxes (i.e. under a 0.27 dollar increase); and the dashed line shows how they would fare under half a standard deviation increase in the share of parents who socialize their children at least once a week (i.e. an increase of 9.8 percentage points). The former is an example of a policy that targets children directly, while the latter is an example of a policy that targets children indirectly, via parental behavior.

We find that the proposed increase in taxes accelerates the rate of adjustment of youth ever-smoking rates so that 90 percent of the adjustment is achieved within two generations. In comparison, the proposed increase in socialization accelerates the rate of adjustment so that 90 percent of the adjustment is achieved within only one generation. Similarly, the increase in taxes reduces the rate of young smokers at the steady-state by 0.8

[16] Because we use our most restrictive specification to carry out the projections, our results are based on a sample size of 2074. The probability that the child ever smokes in this sample equals 19.6% (as opposed to the 18% reported in Table 1). Our results suggest that, in order to reach their equilibrium value, youth smoking rates need to drop by 8.6 percentage points (from 19.6% to 11%).

percentage points, whereas the increase in socialization reduces it by 1 percentage point. By no means, do we present these results to suggest that subsidizing parental socialization is a preferred tobacco control policy relative to a tax increase. Such a claim would require evidence on the cost of each policy regime. Rather, this evidence demonstrates that a policy maker has the tools to significantly change both the steady-state smoking rates of young people and the speed of adjustment to that steady state. For example, *Healthy People 2020* reports that 19.5 percent of adolescents in grades 9 through 12 smoked cigarettes in 2009 and sets the objective to reduce that percentage to 16 percent by 2020. The results we present in Figure 6 suggest that, by using the right policy regime, the government can achieve and even surpass that goal.

7 Conclusion

Building on the literature on cultural transmission, we develop a model of smoking dynamics that focuses on the role of parents and social norms; we use novel data to test its theoretical predictions; and we find empirical support. Our paper advances the literature in several ways.

On the theory front, we extend the seminal work by Bisin and Verdier (2001) to provide a rationale for why traits first emerge, why parents may change the traits they prefer to transmit to their children, and how long-term cultural heterogeneity can be achieved when that occurs. We argue that a cultural trait may emerge when a profit-maximizing industry promotes it, and the way people perceive that trait can be influenced by the flow of related information. Thus, relative to the existing theory, we contribute a framework of analysis to study the transmission mechanisms of a wider variety of cultural traits. Specifically, our model can be used to study (i) cultural traits that already exist and the way people perceive them does not change over time (e.g. preferences on education; trust; religion); (ii) cultural traits that already exist but the way people perceive them changes over time (e.g. preferences on smoking, polluting the environment, or women's rights); and (iii) cultural traits that are brand new (e.g. the culture of social networking). To date, the cultural transmission theory has focused on the traits in the first category.

In developing our model, we introduce new mechanisms to characterize the cultural transmission process. Specifically, we relax a standard assumption in the cultural transmission theory that, when children adopt their traits from society, this happens via a (random or non-random) matching process. This matching process entails that the probability of acquiring a trait always increases with the prevalence of that trait in society. In our model, we allow the industry to affect the direction of this relationship. Borrowing terminology from the literature on identity formation, we formally define the positive relationship between the probability of adopting a trait and the prevalence of that trait in society as cultural conformity. We show that conformity in the social channel of transmission is always tied to substitution in the parental channel of transmission, which posits that parents increase their socialization efforts the more prevalent their preferred cultural trait is in society, and is a cornerstone

assumption in the literature. Conformity and substitution will co-exist because parents will lower their socialization efforts when their preferred trait becomes more popular among societal groups to which they know that their children will conform. Correspondingly, we formalize the assumption of cultural distinction to predict the opposite of cultural conformity; i.e. a negative relationship between the probability of adopting a trait and the prevalence of that trait in society. We show that distinction in the social channel of transmission is always tied to complementarity in the parental channel of transmission, which posits that parents decrease their socialization efforts the more prevalent their preferred cultural trait is in society. Distinction and complementarity will co-exist because parents will increase their socialization efforts when their preferred trait becomes more popular among societal groups from which they know that their children will distinguish themselves. We take all our theoretical predictions to U.S. data and we find supporting evidence.

To test our model, we carefully account for the endogeneity of the parental socialization efforts using a novel measure of parental exposure to anti-smoking information as an instrument. We also avoid selection, simultaneity, and exogenous correlation bias in the estimated social effects by measuring smoking prevalence of the role-model population at the state-level, and including a full set of fixed effects in our regression models. Thus, our empirical analysis contributes to the health economics literature causal estimates of parental and social influences on youth smoking participation. Whether smoking behavior is transmitted through parents, role-models, or peers is relevant for designing tobacco control policy and anti-smoking campaigns. If children primarily pick up smoking from their parents, policies that target parental behavior may be more effective at preventing smoking onset than policies that target young people directly. If children primarily pick up smoking from the society, then this implies externalities that can lead to large differences in smoking behavior through social-multiplier effects. On the one hand, social pressure can cause consumption to be sticky in the face of policy instruments; on the other hand, social influences can complement government interventions to prevent smoking initiation among young people. Our results suggest that parental influences are more important than social influences in the transmission of the smoking trait, and they showcase the spread of anti-smoking information as a key instrument to lower smoking rates among young people.

Finally, we demonstrate how the cultural transmission theory can provide an analytical framework which policy makers can use to evaluate the long-term effects of tobacco control policies. Specifically, we use our empirical results to project what will be the steady state rate of youth smoking in future generations. We show that the rate converges to a steady state in which smoking persists. But we also show that a policy maker can affect both the level of smoking at that steady state and the speed of adjustment to that steady state. Our projections suggest that, under the right policy regime, it is possible to achieve the youth smoking rate objectives set by *Healthy People 2020*.

References

[1] Algan, Yann, and Pierre Cahuc. 2010. "Inherited Trust and Growth." American Economic Review, 100(5): 2060-92.

[2] Aquilino, W. S. 1994. "Interview mode effects in surveys of drug and alcohol use: a field experiment." Public Opinion Quarterly, 58: 210-240.

[3] Avenevoli S. and Ries Merikangas K. 2003. Familial influences on adolescent smoking. Addiction 98(Suppl. 1): 1-20.

[4] Becker G. S. and Murphy K. M. 1988. A Theory of Rational Addiction, Journal of Political Economy, 96 (4): 675-700.

[5] Bisin, Alberto and Thierry Verdier, 2000. "Beyond the Melting Pot: Cultural Transmission, Marriage and the Evolution of Ethnic and Religious Traits." *Quarterly Journal of Economics, 115: 955-988.*

[6] Bisin, Alberto and Thierry Verdier, 2001. "The Economics of Cultural Transmission and the Dynamics of Preferences. *Journal of Economic Theory*, 97: 298-319.

[7] Bisin, Alberto and Thierry Verdier, 2011. "The Economics of Cultural Transmission and Socialization." in *Handbook of Social Economics, Jess Benhabib, Alberto Bisin, and Matt Jackson (eds), Elsevier Science.*

[8] Bisin, A., Topa,G., Verdier,T., 2004.Religious intermarriage and socialization in the United States.Journal of Political Economy.112,615–664.

[9] Bisin Alberto, Patacchini Eleonora, Verdier Thierry and Zenou Yves. 2013. "Bend It Like Beckham: Ethnic Identity and Integration," Unpublished manuscript (available at: http://people.su.se/~yvze0888/Beckham%2001-23-2013.pdf)

[10] Borjas, George J. 1992. "Ethnic Capital and Intergenerational Mobility." The Quarterly Journal of Economics, 107(1):123-50.

[11] Chern, Wen S, Loehman, Edna T, and Yen, Steven T, 1995. "Information, Health Risk Beliefs, and the Demand for Fats and Oils," The Review of Economics and Statistics, 77(3): 555-564.

[12] Christopoulou, R., Lillard, D. R. 2013. Is Smoking Behavior Culturally Determined? Evidence from British Immigrants. NBER Working Paper No. 19036.

[13] Clark, A.E., and Etile, F., 2006. Don't give up on me baby: spousal correlation in smoking behavior. Journal of Health Economics 25 (5), 958–978.

[14] Clark, Andrew E. and Youenn L. 2007. “It wasn’t me, it was them!” Social Influence in risky behavior by adolescents. Journal of Health Economics. 26: 763-784.

[15] Dohmen Thomas, Armin Falk, David Huffman, Uwe Sunde, 2012. "The Intergenerational Transmission of Risk and Trust Attitudes," Review of Economic Studies, 79(2): 645-677.

[16] Fernandez, Raquel, and Alessandra Fogli. 2009. “Culture: An Empirical Investigation of Beliefs, Work, and Fertility.” American Economic Journal: Macroeconomics, 1(1): 146–77.

[17] Fletcher Jason, M. 2010. “Social Interactions and Smoking: Evidence using multiple student cohorts, instrumental variables, and school fixed effects”, Health Economics, 19: 466-484.

[18] Freedman David A. and Jasjeet S. Sekhon. 2010. “Endogeneity in Probit Response Models.” Political Analysis, 18(2): 138-150.

[19] Gaviria, Alejandro and Raphael, Steven. 2001. "School-Based Peer Effects And Juvenile Behavior," The Review of Economics and Statistics, 83(2): 257-268.

[20] Göhlmann, Silja, Christoph M. Schmidt, and Harald Tauchmann. 2010. “Smoking Initiation in Germany: The Role of Intergenerational Transmission.” Health Economics, 19(2): 227- 242.

[21] Harris, Jeffrey E. and Gonzalez Lopez-Valcarcel B. 2008. “Assymetric peer effects in the analysis of cigarette smoking among young people in the United States, 1992-1999” Journal of Health Economics. 27:249-264.

[22] Heckman James J. 1978. “Dummy Endogenous Variables in a Simultaneous Equations System.” Econometrica, 46(6): 931-959.

[23] Huver Rose M. E., Engels Rutger C. M. E, Van Breukelen Gerard, De Vreis Hein. 2007. “Parenting style and adolescent smoking cognitions and behavior.” Psychology and Health, 22(5): 575-593.

[24] Jellal Mohamed and Wolff Francois-Charles. 2002. “Cultural evolutionary altruism: theory and evidence”, European Journal of Political Economy, 18: 241-262.

[25] Lillard, Dean. 2011. "Keeping it in the Family? If Parents Smoke Do Children Follow?" Journal of Applied Social Science Studies. 131: 277-286

[26] Lillard Dean R. and Sfekas Andrew. 2013. “Just passing through: the effect of the Master Settlement Agreement on estimated cigarette tax price pass-through.” Applied Economics Letters, 20(4): 353-357

[27] Loureiro, Maria L., Anna Sanz-de-Galdeano, and Daniela Vuri. 2010. “Smoking Habits: Like Father, Like Son, Like Mother, Like Daughter?” Oxford Bulletin of Economics and Statistics, 72(6): 717-743.

[28] Lundborg, P., 2006. Having the wrong friends? Peer effects in adolescent substance use. Journal of Health Economics 25 (2), 214–233.

[29] Manski, C.F., 1993. Identification of endogenous social effects: the reflection problem. Review of Economic Studies 60 (3), 531–542.

[30] Manski, C.F., 2000. Economic analysis of social interactions. Journal of Economic Perspectives 14 (3), 115–136.

[31] Nakajima, Ryo. 2007. "Measuring Peer Effects on Youth Smoking Behaviour," Review of Economic Studies, 74(3): 897-935.

[32] Namoro Soiliou and Rania Roushdy. 2008. "Intergenerational Transmission of Fertility Preferences: A Test of the Cultural Substitution Assumption," Working Paper 352, University of Pittsburgh, Department of Economics.

[33] Newey W. K. 1987. "Efficient estimation of limited dependent variable models with endogenous explanatory variables." Journal of Econometrics, 36: 231-250.

[34] Nichols Austin. 2011. "Causal Inference for Binary Regression with Observational Data." CHI11 Stata Conference 6, Stata Users Group.

[35] Orphanides, A., Zervos, D., 1998. Myopia and addictive behavior. The Economic Journal 108 (446), 75–91.

[36] Orphanides, A., Zervos, D., 1995. Rational addiction with learning and regret. The Journal of Political Economy 103 (4), 739–758.

[37] Patacchini Eleonora and Zenou Yves. 2011. "Neighborhood Effects and Parental Involvement in the Intergenerational Transmission of Education." The Journal of Regional Science, 51(5): 987-1013.

[38] Pollak, R. A. 1970. Habit formation and dynamic demand functions. Journal of Political Economy. 78: 745-763.

[39] Pollak, R. A. 1976a. Habit formation and long-run utility functions. Journal of Economic Theory, 13: 272-297.

[40] Pollak, R.A., 1976b. Interdependent preferences. American Economic Review 66 (3), 309–320.

[41] Poutvaara P. and Siemers L-H.R. 2008. "Smoking and Social Interaction". Journal of Health Economics. 27:1503-1515.

[42] Powell L. M. and Chaloupka F.J. 2005. Parents, public policy, and youth smoking, Journal of Policy Analysis and Management, Volume 24, Issue 1, p. 93112.

[43] Powell Lisa M., John A. Tauras, Hana Ross, 2005. The importance of peer effects, cigarette prices and tobacco control policies for youth smoking behavior, Journal of Health Economics, Volume 24, Issue 5, September 2005, Pages 950-968

[44] Robson, Arthur and Larry Samuelson. 2011. "The Evolutionary Foundations of Preferences." in *Handbook of Social Economics, Jess Benhabib, Alberto Bisin, and Matt Jackson (eds), Elsevier Science.*

[45] Royal College of Physicians of London (RCPL), 1962. Smoking and Health: Summary and Report of the Royal College of Physicians of London on Smoking in Relation to Cancer of the Lung and other Diseases, London: Pitman Medical Publishing Co. Ltd

[46] Sáez-Martí María & Sjögren Anna, 2008. "Peers and Culture," Scandinavian Journal of Economics, 110(1): 73-92.

[47] Stigler G. J. and Becker G. S. 1977. De Gustibus Non Est Disputandum, American Economic Review, 67 (2):76-90.

[48] U.S. Department of Health, Education and Welfare (USDHEW). 1964. "Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service", Public Health Service Publication No. 1103.

[49] Viscusi, W. Kip & Hakes, Jahn K. 2008. "Risk Beliefs And Smoking Behavior," Economic Inquiry, 46(1): 45-59.

[50] Viscusi, W. Kip and Hersch Joni. 2011. "Tobacco Regulation Through Litigation: The Master Settlement Agreement." in Regulation versus Litigation: Perspectives from Economics and Law. Daniel P Kessler (editor), University of Chicago Press, 71-101.

Appendix I: Proofs

Proof of Proposition 1: The parent invests an amount $\lambda_i^* \equiv \arg\max_{\lambda_0} (1 - v(\lambda_i)) S(q) u_i(H) - c(\lambda_i)$ into anti-smoking culturalization. The assumptions on $v(.)$ and $c(.)$ ensure the maximization problem is concave, and so the solution solves the First Order Condition $-v'(\lambda_i) S(q) u_i(H) - c'(\lambda_i) = 0$, which can be rewritten as (??). ⊠

Proof of Proposition 2: By total differentiation of (??), we get $\frac{d\lambda_i^*}{dH} = \frac{-u_i'(H)}{(u_i(H))^2} \frac{1}{S(q)} \frac{\left[c'(\lambda_i^*)\right]^2}{c''(\lambda_i^*)v'(\lambda_i^*) - v''(\lambda_i^*)c'(\lambda_i^*)} > 0$ (Prediction 1). We then always have $\frac{d\lambda_{-i}^*}{dq} = \frac{S'(q)}{[S(q)]^2} \frac{1}{-u_i(H)} \frac{\left[c'(\lambda_i^*)\right]^2}{c''(\lambda_i^*)v'(\lambda_i^*) - v''(\lambda_i^*)c'(\lambda_i^*)} > 0$ iff $S'(q) > 0$, and the effect of an increase in smoking prevalence on the likelihood a child becomes a smoker can be expressed as $(1 - v(\lambda_i^*)) S'(q)$ (Prediction 2). ⊠

Proof of Proposition 3: In a subgame perfect equilibrium, the firm's objective function can be written as:

$$\max_{\theta} Q(q, \theta) - \kappa(\theta),$$

where $Q(q, \theta) \equiv [q(1 - a(q, \theta)) + (1 - q)(1 - d(q, \theta))] S(q, \theta)$ denotes the proportion of children who become smokers given a proportion q of adult smokers. whose solution $\theta^*(q)$ solves the FOC:[17]

$$[q(1 - a(q, \theta)) + (1 - q)(1 - d(q, \theta))] \frac{\partial S(q, \theta)}{\partial \theta} = \kappa'(\theta)$$

In order to understand when cultural distinction vs. conformity arise, we need to study the sign of: $\left.\frac{dS}{dq}\right|_{\theta^*} = \frac{\partial S}{\partial q} + \left.\frac{\partial S}{\partial \theta}\right|_{\theta^*} \frac{\partial \theta^*}{\partial q}$, where $\frac{\partial \theta^*}{\partial q} = \frac{\left[d(q) - a(q) - qa'(q) - (1-q)d'(q)\right] \frac{\partial S(q,\theta)}{\partial \theta} + [q(1-a(q)) + (1-q)(1-d(q))] \frac{\partial^2 S(q,\theta)}{\partial \theta \partial q}}{c''(\theta) - [q(1-a(q)) + (1-q)(1-d(q))] \frac{\partial^2 S(q,\theta)}{\partial \theta^2}}$. While the denominator of the expression for $\frac{\partial \theta^*}{\partial q}$ is always positive, the numerator can admit both signs. Hence cultural substitution obtains if and only if the numerator takes a large enough negative value. ⊠

Proof of Proposition 4: When $q = 0$, we have $\dot{q} = (1 - d(0)) S(0)$. Under cultural substitution we have $d(0) = 0$ and so $\dot{q} > 0$ iff $S(0) > 0$. Under cultural complementarity we have $d(0) > 0$ and so $\dot{q} > 0$ iff $S(0) > 0$ and $d(0) < 1$. When $q = 1$, we have $\dot{q} = (1 - a(1)) S(1) - 1$. Under cultural substitution we have $a(1) > 0$ and so $\dot{q} < 0$ always. Under cultural complementarity we have $a(1) = 0$ and so $\dot{q} < 0$ iff $S(1) < 1$. By Proposition 2, we have $S' < 0$ under cultural complementarity, and so $S(0) > 0$ and $S(1) < 1$. ⊠

[17] Our assumptions ensure that the second order condition satisfies

$$SOC : [q(1 - a^1(q)) + (1 - q)(1 - d^0(q))] \frac{\partial^2 S(q, \theta)}{\partial \theta^2} - \kappa''(\theta) < 0$$

and so we have a concave problem whose solution can be recovered through the FOC.

Appendix II: Tables

Table 1: Weighted means and frequencies of selected variables

Variable	$\bar{x}/\%$	[s.d]
Child		
Female	0.50	
Age	13.83	[2.25]
Race		
White, non Hispanic	0.66	
Black, non Hispanic	0.16	
Other	0.18	
Ever smoked regularly	0.18	[0.39]
Acc. sum of anti-smoking articles potentially read since age 10	4.07	[0.05]
Primary Care Giver (PCG)		
Age	41.99	[6.10]
Education	13.02	[2.75]
Relationship to child		
Biological mother	0.93	
Biological father	0.06	
Adoptive mother	0.01	
Stepmother	0.00	
Socializes child against substance use		
Never	0.21	
Once or twice a month	0.42	
Once a week	0.14	
Several times a week	0.13	
Every day	0.10	
Ever smoked regularly	0.45	[0.50]
Acc. sum of anti-smoking articles potentially read since age 10	12.2	[2.78]
St.deviation of anti-smoking articles potentially read since age 10	0.42	[0.07]
Family unit		
Real family income/10000	4.31	[5.22]
State		
Real cigarette tax	1.59	[0.54]
Real state income/10000	3.66	[0.50]
Sm. prevalence of total population 20-29 years older than child	0.26	[0.05]

Observations: 2246.

Table 2: Probit regression of the probability that the child has ever smoked

	All categories	Dichotomized: 1='At least'			
	(1)	(2)	(3)	(4)	(5)
Child socialized against substance use					
Once or twice per month	0.351***	0.288***			
	(0.026)	(0.024)			
Once a week	0.256***		-0.025		
	(0.033)		(0.020)		
Several times a week	0.187***			-0.050**	
	(0.033)			(0.022)	
Every day	0.192***				-0.031
	(0.037)				(0.031)
Sm. prevalence of role-model population	3.399***	3.117***	3.598***	3.579***	3.522***
	(1.048)	(1.047)	(1.043)	(1.041)	(1.041)
PCG has ever smoked	0.329***	0.333***	0.337***	0.336***	0.338***
	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)
Education of PCG	-0.044***	-0.038***	-0.041***	-0.042***	-0.041***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Family income	-5.453***	-5.433***	-5.121***	-5.100***	-5.127***
	(0.388)	(0.387)	(0.385)	(0.385)	(0.385)
State cigarette tax	-0.930***	-0.919***	-0.900***	-0.902***	-0.901***
	(0.074)	(0.074)	(0.074)	(0.074)	(0.074)
State income per capita	-2.404***	-2.450***	-2.372***	-2.374***	-2.384***
	(0.171)	(0.172)	(0.171)	(0.170)	(0.170)
Pseudo R-squared	0.257	0.256	0.252	0.252	0.252

Controls: Dummies for age of PCG, and age, sex, state, race, and religion of child. Observations: 2246. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 3: IVprobit estimates of baseline specification

	Prob(child ever smokes)	Prob(child socialized at least 1/week)
Child socialized at least once/week	-1.461***	
	(0.159)	
Sm. prevalence of role-model population	4.929***	1.309***
	(0.856)	(0.197)
PCG has ever smoked	0.251***	-0.003
	(0.026)	(0.005)
Education of PCG	-0.085***	-0.037***
	(0.005)	(0.001)
Family income	-3.504***	0.471***
	(0.468)	(0.048)
State cigarette tax	-0.656***	0.045***
	(0.083)	(0.0110)
State income per capita	-1.684***	0.148***
	(0.205)	(0.022)
Child info exposure since age 10	-0.025*	-0.012**
	(0.014)	(0.005)
PCG info exposure since age 10		0.018***
		(0.006)
Infrequency of PCG info exposure since age 10		-1.160***
		(0.101)
Amemiya-Lee-Newey minimum X^2 statistic	0.82 [0.365]	
Hausman X^2 statistic	252.0 [0.000]***	
Wald X^2 statistic	40.4 [0.000]***	
X^2 for joint significance of instruments	165.4 [0.000]***	
Change in prob(child ever smokes) after half s.d increase in:		
Socialization	-0.039	
Sm. prevalence of role-model population	0.028	

Controls: Dummies for age of PCG, and age, sex, state, race, and religion of child. Obs: 2246. Standard errors in parenthesis; p-values in brackets. *** p<0.01, ** p<0.05, * p<0.1.

Table 4: Robustness of baseline estimates to alternative methods of estimation

	Linear probability model			Bivariate	Probit +
	2SLS	IVLIML	IVGMM	probit	Ordered probit
	(1)	(2)	(3)	(4)	(5)
Second-stage: Prob(child ever smokes)					
Child socialization	-0.187***	-0.207***	-0.173**	-1.501***	-0.415***
	(0.063)	(0.066)	(0.071)	(0.019)	(0.147)
Sm. prevalence of role-model population	1.215***	1.246***	1.153***	4.171***	4.982***
	(0.187)	(0.191)	(0.176)	(0.721)	(1.164)
PCG has ever smoked	0.072***	0.072***	0.073***	0.223***	0.338***
	(0.004)	(0.004)	(0.004)	(0.016)	(0.018)
Education of PCG	-0.017***	-0.018***	-0.016***	-0.086***	-0.077***
	(0.003)	(0.003)	(0.003)	(0.004)	(0.014)
Family income	-0.346***	-0.337***	-0.354***	-3.054***	-4.779***
	(0.048)	(0.049)	(0.048)	(0.297)	(0.406)
State cigarette tax	-0.028***	-0.027***	-0.028***	-0.534***	-0.821***
	(0.009)	(0.009)	(0.008)	(0.059)	(0.079)
State income per capita	-0.072***	-0.070***	-0.072***	-1.487***	-2.344***
	(0.019)	(0.020)	(0.015)	(0.134)	(0.171)
Child info exposure since age 10	0.034***	0.034***	0.035***	-0.022	-0.014
	(0.003)	(0.003)	(0.003)	(0.013)	(0.016)
First-stage: Prob(child socialization)					
Sm. prevalence of role-model population	1.305***	1.305***	1.305***	4.438***	2.984***
	(0.200)	(0.200)	(0.204)	(0.657)	(0.487)
PCG has ever smoked	-0.002	-0.002	-0.002	-0.014	0.011
	(0.005)	(0.005)	(0.005)	(0.015)	(0.012)
Education of PCG	-0.036***	-0.036***	-0.036***	-0.115***	-0.090***
	(0.001)	(0.001)	(0.001)	(0.003)	(0.003)
Family income	0.473***	0.473***	0.473***	1.400***	1.145***
	(0.048)	(0.048)	(0.047)	(0.137)	(0.113)
State cigarette tax	0.046***	0.046***	0.046***	0.180***	0.231***
	(0.011)	(0.011)	(0.011)	(0.033)	(0.027)
State income per capita	0.148***	0.148***	0.148***	0.440***	0.257***
	(0.023)	(0.023)	(0.023)	(0.071)	(0.056)
Child info exposure since age 10	-0.011**	-0.011**	-0.011**	-0.067***	-0.011
	(0.005)	(0.005)	(0.005)	(0.014)	(0.010)
PCG info exposure since age 10	0.016***	0.016***	0.016***	0.081***	0.057***
	(0.006)	(0.006)	(0.006)	(0.016)	(0.011)
Infrequency of PCG info exposure since age 10	-1.161***	-1.161***	-1.161***	-3.717***	-2.505***
	(0.105)	(0.105)	(0.114)	(0.275)	(0.210)

Controls: Dummies for age of PCG, and age, sex, state, race, and religion of child. Observations: 2246. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The child socialization variable in (1)-(4) is the Prob(child socialized at least once/week) and in (5) it is the original ordinal variable that takes values 1-5 to measure how often PCGs socialize children. In (5) we estimated the 1st stage by ordered probit, and the 2nd stage by probit using the hat values from the 1st stage as the socialization variable. We did not adjust the errors.

Table 5: Robustness of baseline estimates to alternative definitions of the socialization variable

	Dichotomized: 1='At least'		
	Once or twice/month	Several times a week	Every day
Second-stage: Prob(child ever smokes)			
Child socialization	-1.465***	-2.215***	-3.249***
	(0.324)	(0.150)	(0.183)
Sm. prevalence of role-model population	3.397***	3.809***	5.218***
	(0.862)	(0.718)	(0.652)
PCG has ever smoked	0.266***	0.136***	0.195***
	(0.0345)	(0.0343)	(0.031)
Education of PCG	-0.049***	-0.097***	-0.085***
	(0.004)	(0.004)	(0.003)
Family income	-3.625***	-2.369***	-2.415***
	(0.695)	(0.528)	(0.514)
State cigarette tax	-0.618***	-0.334***	-0.302***
	(0.121)	(0.100)	(0.097)
State income per capita	-1.835***	-1.420***	-1.088***
	(0.282)	(0.219)	(0.234)
Child info exposure since age 10	0.005	0.002	-0.057***
	(0.014)	(0.012)	(0.011)
First-stage: Prob(child socialization)			
Sm. prevalence of role-model population	0.345**	0.752***	1.057***
	(0.175)	(0.168)	(0.120)
PCG has ever smoked	0.012**	-0.024***	0.010***
	(0.004)	(0.004)	(0.003)
Education of PCG	-0.012***	-0.034***	-0.020***
	(0.001)	(0.001)	(0.001)
Family income	0.353***	0.329***	0.083***
	(0.042)	(0.041)	(0.029)
State cigarette tax	0.061***	0.090***	0.048***
	(0.010)	(0.009)	(0.007)
State income per capita	0.033*	-0.007	0.040***
	(0.020)	(0.019)	(0.014)
Child info exposure since age 10	0.030***	-0.0004	-0.019***
	(0.005)	(0.004)	(0.003)
PCG info exposure since age 10	-0.025***	0.015***	0.011***
	(0.006)	(0.004)	(0.003)
Infrequency of PCG info exposure since age 10	-0.214**	-0.620***	-0.379***
	(0.089)	(0.087)	(0.064)

All controls and remaining information, as in Table 3.

Table 6: Weighted means and frequencies of new controls and instruments

	$\bar{x}/\%$	[s.d]	N
Indicators of child personality and parenting styles			
Child is a "rule-breaker"	0.31	[0.46]	2232
Child subject to many rules, strictly enforced, rarely discussed	0.10	[0.31]	2232
Child spanked more than 3 times per week	0.01	[0.09]	2232
PCG never discusses any subject with child	0.01	[0.08]	2232
Measures of socialization cost			
Number of children in the family unit	2.10	[1.15]	2088
PCG has a job	0.71	[0.45]	2088
Hours per week PCG typically works	27.5	[20.5]	2088
PCG usually works a regular daytime schedule	0.57	[0.49]	2088
Typically takes PCG over an hour to get to work each way	0.05	[0.21]	2088
Alternative measures of information exposure			
Mean anti-smoking articles PCG potentially read per year since age 10	0.88	[0.13]	2236
Days per week the PCG reads the newspaper	1.96	[1.94]	2236
Alternative definitions of role-mode sm. prevalence			
State-specific smoking prevalence of:			
Total population 0-9 years older than child	0.19	[0.07]	2246
Total population 10-19 years older than child	0.26	[0.06]	2246
Females 0-9 years older than child	0.17	[0.07]	2246
Females 10-19 years older than child	0.23	[0.06]	2246
Females 20-29 years older than child	0.24	[0.06]	2246
Males 0-9 years older than child	0.21	[0.07]	2246
Males 10-19 years older than child	0.30	[0.05]	2246
Males 20-29 years older than child	0.29	[0.05]	2246

Rule breaker: the child did something dangerous, damaged public property, got in a fight, drove drunk or high over 10 times the last 6 months or that the child has been arrested or put in jail more than once to date or that the child has a lot of secrets or hides a lot of things from parents.

Table 7: Robustness of the baseline specification to new instruments

	(1)	(2)	(3)
Second-stage: Prob(child ever smokes)			
Child socialized at least once/week	-1.465***	-1.436***	-1.576***
	(0.138)	(0.174)	(0.132)
Sm. prevalence of role-model population	4.376***	4.527***	3.952***
	(0.861)	(0.889)	(0.850)
First-stage: Prob(child socialization)			
Sm. prevalence of role-model population	1.231***	1.245***	1.186***
	(0.197)	(0.210)	(0.211)
PCG info exposure since age 10 (acc. sum)	0.017***		
	(0.006)		
* frequency PCG reads the newspaper	0.0005***		
	(0.0000)		
PCG info exposure since age 10 (mean)		0.290***	0.273***
		(0.082)	(0.079)
* frequency PCG reads the newspaper			0.006***
			(0.001)
Infrequency of PCG info exposure since age 10	-1.167***	-1.206***	-1.181***
	(0.099)	(0.104)	(0.103)
Observations	2,236	2,246	2,236
Amemiya-Lee-Newey minimum X^2 statistic	1.095	17.0***	17.1***
Hausman X^2 statistic	252.0***	253.9***	295.0***
Wald X^2 statistic	53.3***	33.8***	57.6***
X^2 for joint significance of instruments	216.5***	163.6***	194.0***

Controls: for (1) as in baseline specification; (2) and (3) control for child info exposure since age 10 (mean) instead of (acc. sum).

Table 8: Robustness of the baseline specification to new controls

	(1)	(2)	(3)
Second-stage: Prob(child ever smokes)			
Child socialized at least once a week	-1.424***	-1.843***	-1.836***
	(0.179)	(0.095)	(0.108)
Sm. prevalence of role-model population	4.248***	3.535***	2.773***
	(0.894)	(0.760)	(0.791)
Child is a 'rule-breaker'	0.417***		0.389***
	(0.024)		(0.025)
Child subject to many rules, strictly enforced, rarely discussed	-0.593***		-0.562***
	(0.031)		(0.035)
Child spanked more than 3 times per week	0.546***		0.707***
	(0.115)		(0.093)
PCG never discusses any subject with child	0.611***		0.395***
	(0.102)		(0.093)
No. of kids below 18 in the family unit		-0.032***	-0.027***
		(0.010)	(0.010)
PCG has a job		-0.303***	-0.374***
		(0.039)	(0.039)
Hours per week PCG typically works		0.002**	0.003***
		(0.001)	(0.001)
PCG works a regular daytime schedule		0.089***	0.086***
		(0.021)	(0.022)
Takes PCG over an hour to get to work each way		-0.062*	-0.004
		(0.036)	(0.037)
First-stage: Prob(child socialized at least once/week)			
Sm. prevalence of role-model population	1.312***	1.006***	0.979***
	(0.197)	(0.208)	(0.208)
PCG info exposure since age 10	0.017***	0.033***	0.032***
	(0.006)	(0.006)	(0.006)
Infrequency of PCG info exposure since age 10	-1.121***	-1.205***	-1.171***
	(0.101)	(0.105)	(0.105)
Child is a 'rule-breaker'	0.054***		0.060***
	(0.005)		(0.005)
Child subject to many rules, strictly enforced, rarely discussed	-0.109***		-0.100***
	(0.007)		(0.008)
Child spanked more than 3 times per week	0.367***		0.368***
	(0.026)		(0.026)
PCG never discusses any subject with child	-0.038		-0.047*
	(0.028)		(0.027)
No. of kids below 18 in the family unit		0.015***	0.014***
		(0.003)	(0.003)
PCG has a job		-0.177***	-0.184***
		(0.011)	(0.011)
Hours per week PCG typically works		0.003***	0.002***
		(0.000)	(0.000)
PCG works a regular daytime schedule		0.045***	0.046***
		(0.007)	(0.007)
Takes PCG over an hour to get to work each way		0.015	0.025**
		(0.011)	(0.011)
Observations	2,232	2,088	2.074
Amemiya-Lee-Newey minimum X^2 statistic	0.045	1.299	0.209
Hausman X^2 statistic	242.7***	358.1***	350.1***
Wald X^2 statistic	31.0***	90.1***	71.9***
X^2 for joint significance of instruments	157.8***	139.6***	133.4***

Controls: as in baseline specification.

Table 9: Probit estimation of the probability that the child is socialized at least once/week

	(1)	(2)	(3)
Second-stage: Prob(child ever smokes)			
Child socialized at least once/week	-1.462***	-1.457***	-0.998***
	(0.159)	(0.161)	(0.208)
Prev. of role model pop. * PCG never smoked	4.455***	4.154***	
	(0.875)	(0.881)	
Prev. of role model pop. * PCG ever smoked	5.158***		
	(0.867)		
Prev. of role model pop. * PCG currently smokes		4.938***	
		(0.870)	
Prev. of role model pop. * PCG used to smoke		3.966***	
		(0.878)	
Prev. of role model pop. * PCG never smoked * PCG info exposure q0-10			0.461***
			(0.080)
Prev. of role model pop. * PCG never smoked * PCG info exposure q10-50			0.295***
			(0.062)
Prev. of role model pop. * PCG never smoked * PCG info exposure q50-90			0.252***
			(0.056)
Prev. of role model pop. * PCG never smoked * PCG info exposure q90-100			0.318***
			(0.0541)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q0-10			0.573***
			(0.083)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q10-50			0.540***
			(0.066)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q50-90			0.485***
			(0.057)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q90-100			0.478***
			(0.057)
First-stage: Prob(child socialized at least once/week)			
Prev. of role model pop. * PCG never smoked	1.367***	1.361***	
	(0.200)	(0.200)	
Prev. of role model pop. * PCG ever smoked	1.195***		
	(0.206)		
Prev. of role model pop. * PCG currently smokes		1.197***	
		(0.206)	
Prev. of role model pop. * PCG used to smoke		1.119***	
		(0.208)	
Prev. of role model pop. * PCG never smoked * PCG info exposure q0-10			0.053***
			(0.019)
Prev. of role model pop. * PCG never smoked * PCG info exposure q10-50			0.078***
			(0.017)
Prev. of role model pop. * PCG never smoked * PCG info exposure q50-90			0.094***
			(0.016)
Prev. of role model pop. * PCG never smoked * PCG info exposure q90-100			0.124***
			(0.016)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q0-10			0.030
			(0.020)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q10-50			0.061***
			(0.017)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q50-90			0.096***
			(0.016)
Prev. of role model pop. * PCG ever smoked * PCG info exposure q90-100			0.086***
			(0.016)
X^2 test of the equality of the coefficients in first-stage	3.538*	11.64***	237.5***

Observations: 2246. Controls: as in baseline specification, excluding sm. prevalence of role model population.

Table 10: Baseline specification under alternative definitions of the role-model population

	Total population				Males	Females
	(1)	(2)	(3)	(4)	(5)	(6)
Second-stage: Prob(child ever smokes)						
Child socialized at least once/week	-1.439***	-1.442***	-1.424***	-1.424***	-2.153***	-1.842***
	(0.163)	(0.163)	(0.161)	(0.165)	(0.183)	(0.094)
Sm. prevalence of:						
total population 0-9 years older	-0.083					
	(0.594)					
total population 10-19 years older	5.434***					
	(0.892)					
total population 20-29 years older	5.366***					
	(0.851)					
males 0-9 years older		-1.088**		-5.766***	-3.917***	-4.909***
		(0.513)		(0.666)	(1.010)	(0.912)
males 10-19 years older		5.543***				
		(0.707)				
males 20-29 years older		3.690***				
		(0.746)				
females 0-9 years older			1.328**	5.114***	7.787***	2.268***
			(0.549)	(0.661)	(2.179)	(0.866)
females 10-19 years older			1.375**			
			(0.689)			
females 20-29 years older			4.145***			
			(0.683)			
First-stage: Prob(child socialized at least once/week)						
Sm. prevalence of:						
total population 0-9 years older	-0.023					
	(0.159)					
total population 10-19 years older	1.272***					
	(0.208)					
total population 20-29 years older	0.153					
	(0.178)					
males 0-9 years older		-0.186		-0.552***	-0.824***	-0.003
		(0.137)		(0.160)	(0.239)	(0.237)
males 10-19 years older		0.087				
		(0.138)				
males 20-29 years older		1.247***				
		(0.173)				
females 0-9 years older			0.329**	0.711***	1.286***	0.594**
			(0.142)	(0.164)	(0.266)	(0.249)
females 10-19 years older			0.428***			
			(0.164)			
females 20-29 years older			0.466***			
			(0.166)			
Observations	2,246	2,246	2,246	2,246	1,058	1,045

Controls: as in baseline specification.

Table 11: Correlation coefficients of alternative measures of the sm. prevalence of the role-model population

Measure 1:	Measure 2:	Correlation
Smoking prevalence in total population:		
0-9 years older than child	10-19 years older than child	0.4839
0-9 years older than child	20-29 years older than child	0.6679
10-19 years older than child	20-29 years older than child	0.8355
Smoking prevalence in male population:		
0-9 years older than child	10-19 years older than child	0.3405
0-9 years older than child	20-29 years older than child	0.5592
10-19 years older than child	20-29 years older than child	0.7127
Smoking prevalence in female population:		
0-9 years older than child	10-19 years older than child	0.5732
0-9 years older than child	20-29 years older than child	0.7106
10-19 years older than child	20-29 years older than child	0.8505
Smoking prevalence in population 0-9 years older than child:		
Males	Females	0.9170

Table 12: Probit estimation of the probability that the child is socialized at least once/week (first-stage)

	PCG info exposure		PCG has a job		Child is a rule-breaker	
	(1)	(2)	(3)	(4)	(5)	(6)
Interacted with:						
PCG never smoked	0.020***	0.020***	-0.066***	-0.065***	0.046***	0.046***
	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)
PCG ever smoked	0.012**		-0.044***		0.068***	
	(0.006)		(0.008)		(0.007)	
PCG currently smokes		0.013**		-0.056***		0.087***
		(0.006)		(0.009)		(0.009)
PCG used to smoke		0.012*		-0.032***		0.035***
		(0.006)		(0.010)		(0.011)
Observations	2,246	2,246	2,101	2,101	2,238	2,238
X^2 test of the equality of the coeffs	17.7***	22.0***	3.9**	11.0***	4.7**	22.5***

Controls: as in baseline specification, excluding interacted variable.

Appendix III: Figures

Figure 1: Smoking prevalence of role-model population of 14 year old children by sex, state, and year

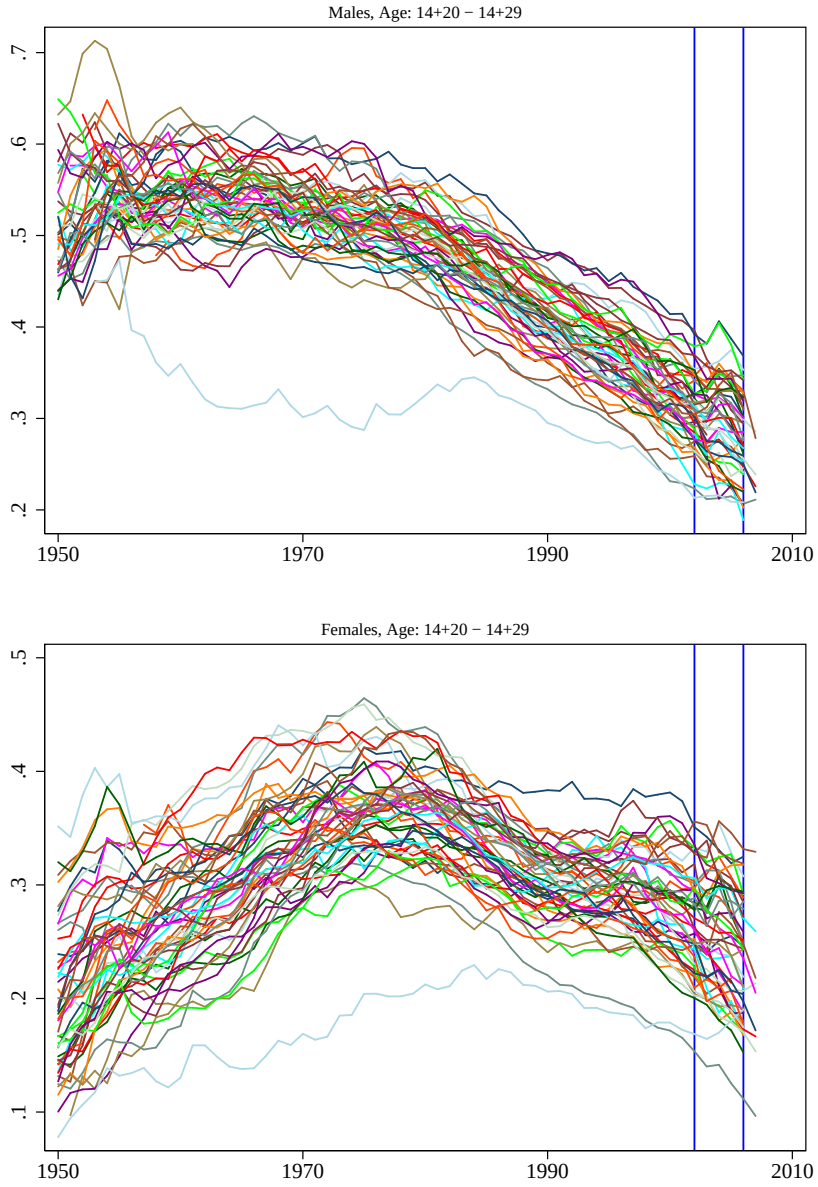

Figure 2: Smoking prevalence of role-model population by sex, age, and state

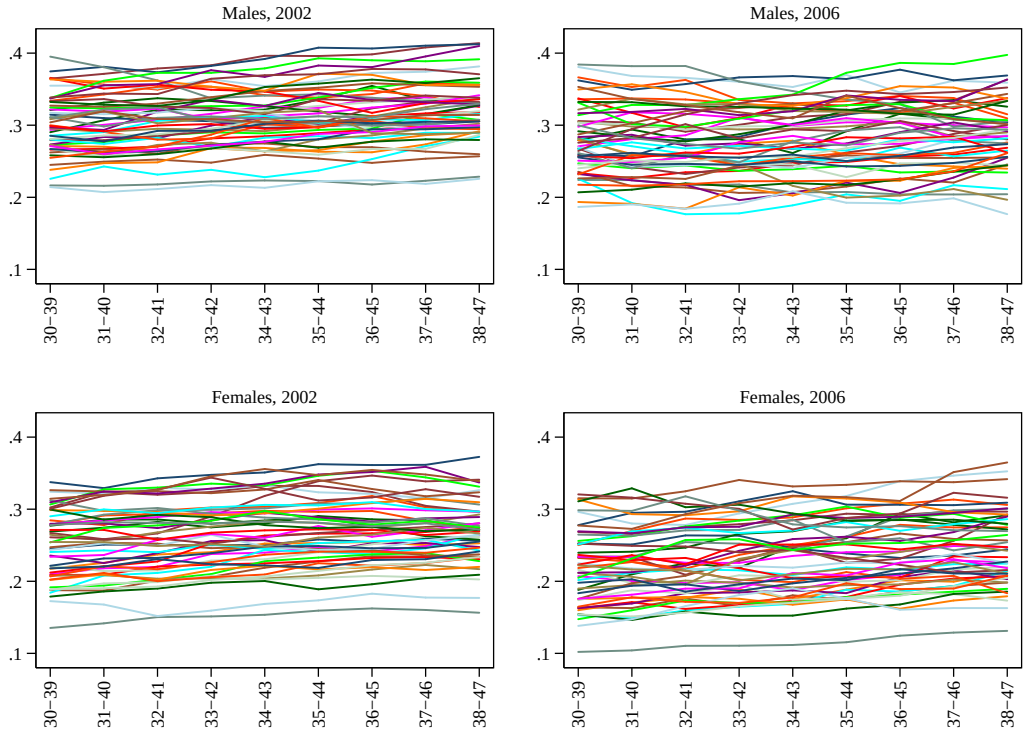

Figure 3: Number of published magazine anti-smoking articles by state and year (weighted by state subscription rate to each magazine)

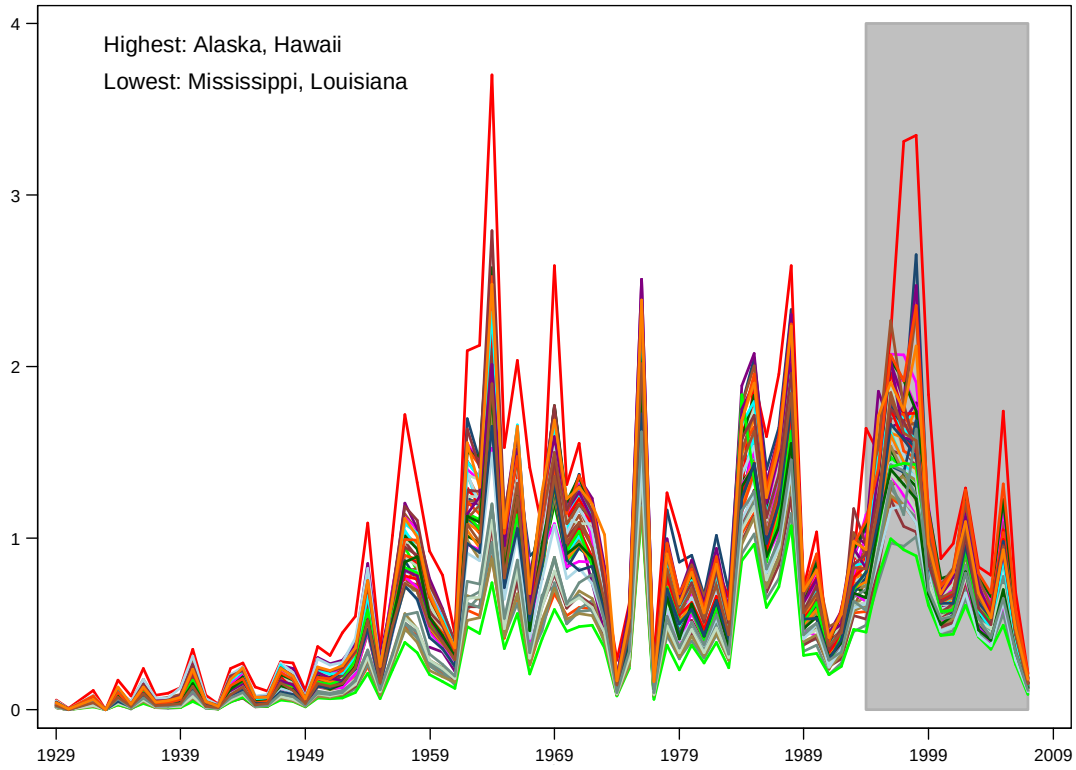

Figure 4: Cigarette taxes, by state and year ($)

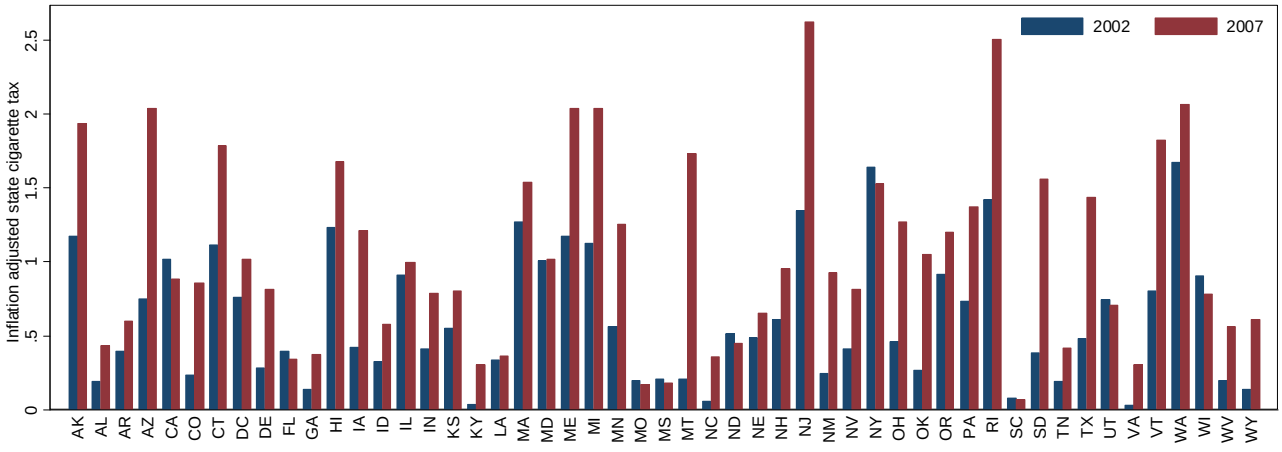

Figure 5: Real income per capita, by state and year ($)

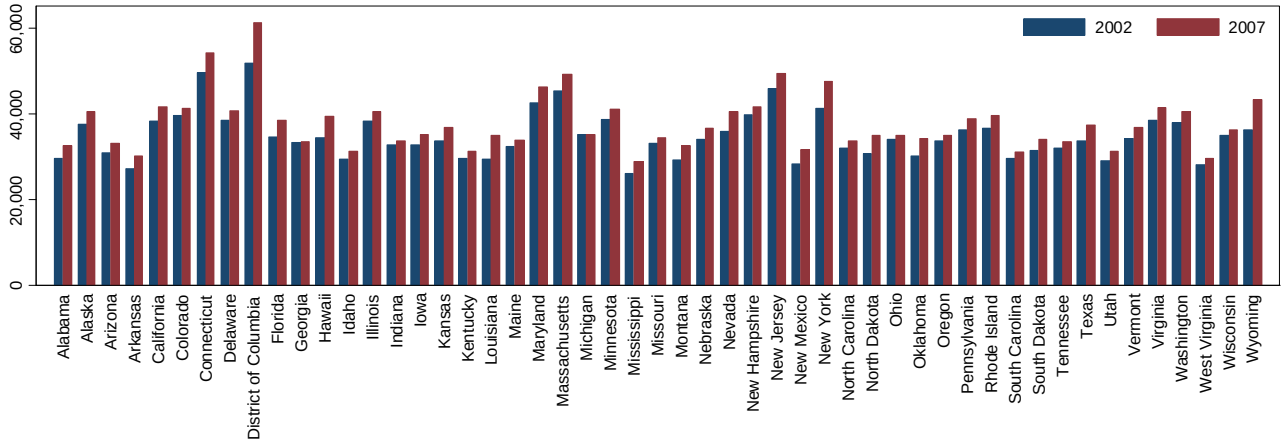

Figure 6: Forward projections of youth ever-smoking rates