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Is Smoking Behavior Culturally Determined? Evidence from British Immigrants*

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Abstract

We exploit migration patterns from the UK to Australia and the US to investigate whether a person's decision to smoke is determined by culture. For each country, we use retrospective data to describe individual smoking trajectories over the life-course. For the UK, we use these trajectories to measure culture by cohort and cohort-age, and more accurately relative to the extant literature. Our proxy predicts smoking participation of second-generation British immigrants but not that of non-British immigrants and natives. Researchers can apply our strategy to estimate culture effects on other outcomes when retrospective or longitudinal data are available.

Keywords

Culture; Immigrant health; Smoking

1 Introduction

A new strand of literature has emerged in economics to investigate the hypothesis that culture determines behavior. To identify culture effects, this literature typically examines immigrant groups in a host country and tests whether their behavior varies systematically

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with behavior of people living in their country of origin. The underlying assumption is that immigrants who arrive from different countries bring different inherited norms and values that may induce them to behave differently even though they face a common context. Because this literature assumes that immigrants are “infected” with the culture of their country of origin, its empirical strategy is sometimes termed the “epidemiological” approach (see reviews by Guiso et al. 2006 and Fernandez 2011). Evidence suggests that culture, measured in this way, significantly influences immigrant behavior. For example, Fishman and Miguel (2007) analyse the parking behavior by diplomats in New York City to examine the link between culture and corruption. They show that parking law violations by diplomats are strongly correlated with measures of corruption in their country of origin. Fernandez and Fogli (2009) investigate whether second generation immigrant women are influenced by their inherited culture when they decide whether or not to participate in the labor force or to bear children. They proxy for inherited culture using the labor force participation rate and fertility rate of the previous generation in the parents' country of origin. They too find evidence of a strong culture effect. Using similar strategies, other papers find a causal effect of culture on bilateral economic exchange (Guiso et al. 2009), economic growth (Algan and Cahuk 2010), preferences for redistribution (Luttmer and Singal 2011), and other outcomes.

We are the first in this growing body of economic research to elaborately address culture dynamics. Due to data limitations, the bulk of the research tests the influence of immigrant culture on outcomes of interest with measures of culture that do not vary over time. Typically used culture measures only vary across the different countries of origin. To our knowledge, Algan and Cahuc (2010) is the only study to date that exploits time variation. To proxy for culture, they use indicators of social attitudes of two cohorts of second generation Americans, whose parents immigrated in different periods. Therefore, they exploit differences between only two periods. However, observers argue that culture, or most its expressions, evolves in a highly dynamic way in reaction to forces associated with globalization, technical change, and general socioeconomic development (Inglehart and Welzel 2005). Further, when one uses a time-invariant measure of culture, one cannot determine whether differences associated with culture measure the effect of culture, the effect of omitted time-invariant factors that vary similarly across countries, or both. For example, Kapoor and Ravi (2012) show that the culture effect on corruption that Fishman and Miguel (2007) predict does not survive after controling for goverment effectiveness. Thus, to claim that they identify a culture effect, all existing studies had to maintain and defend the underlying assumptions that culture changes slowly over time and across the life-course, and that it is not shared across countries.

Our contribution is to observe that there is an important class of economic behaviors - those that are subject to culture dynamics - that cannot be accurately analysed by the standard cross-sectional approach. This class includes behaviors that have already been studied in the literature, such as fertility and labor force participation of women, which are closely tied to dynamic cultural forces like feminism. It also includes a range of behaviors tied to the production of health such as smoking, drinking, early sex, eating habits, driving practices, and exercise. Here, we use the example of smoking behavior to demonstrate how one can test for culture effects when there exists a behavior-specific culture that may be changing rapidly over time and across generations. Our technique relies on the fact that our smoking

data are retrospectively reported and allow us to track lifetime smoking histories. Researchers can generally apply our approach to study any type of behavior reported retrospectively or longitudinally.

We use a natural experiment design that exploits historical patterns of migration from Great Britain to Australia and the US. We use data from cross-sectional or panel surveys, administered in each host country, that collects information on each individual's smoking history and country of origin. With the retrospective information on smoking we describe individual smoking trajectories over the life-course. With the data on country of origin we define samples of British immigrants, immigrants from other countries, and natives. We then relate each group's smoking participation in every year of life to a proxy of British smoking culture that varies by cohort and year of life. We measure smoking culture of a British immigrant by the smoking prevalence rate of UK residents who belong to the same generation as the immigrants' parents. This measure lets us test whether and to what degree the smoking behavior of each group reflects the smoking behavior of their parents' generation in the UK when that generation was at the same point in their life-cycle. If culture matters, i.e. if British immigrant parents carried cultural beliefs with them when they emigrated from the UK and they transmitted those beliefs to their children, our proxy should predict the smoking behavior of their children. Our proxy should not predict the smoking prevalence rates of children of native-born parents and parents who immigrated from other countries.

This exercise is innovative in two ways. First, because our measure of smoking culture varies by cohort and year of life, it captures dynamically how attitudes about smoking changed. Thus, compared to studies that measure culture at the country level, we use a measure that suffers less from measurement error that arises when culture changes. Second, we measure culture as the outcome variable (smoking) in the UK only after we net out any determinants that are correlated across the UK and the host countries. Thus, relative to studies that measure culture as the (gross) outcome variable in the country of immigrant origin, we use a finer measure. Our measure avoids the bias that may arise when contextual factors that affect smoking behavior develop simultaneously in many countries. For example, countries often adopt and sustain policies that are informed by experiences of others; especially countries like the UK, US, and Australia, that have (had) close political and economic ties. Relative to studies that measure culture with variables that represent specific aspects of culture in the country of immigrant origin (religion, trust etc.), we use a more comprehensive measure. Our measure captures the aggregate effect of culture that is transmitted via all possible transmission channels to the extent that it drives smoking behavior and differs across home and host countries.

Apart from the novel technique, our paper also contributes evidence that suggests that culture causally affects smoking choices. We find that British smoking culture significantly predicts the smoking behavior of British immigrants. It does not predict the smoking behavior of the local population in the host countries or of non-British immigrants. We also find that the culture effects differ by gender, with women playing a more important role than men in the process of cultural transmission.

To proceed, we discuss our empirical strategy in section 2, we describe the data in section 3, and we present our results in section 4. A final section concludes the paper.

2 Empirical strategy

2.1 The model

Our empirical exercise relies on the following linear probability model of smoking participation:

$$S_{ict}^{HOST}=\alpha_1 S_{ic,t-1}^{HOST}+\alpha_2 X_t^{HOST}+\alpha_3 \tilde{S}_{c-5,t-25}^{UK}+\alpha_i+\varepsilon_{it}^{HOST} \quad (1)$$

Superscripts *HOST* and *UK* identify variables measured in the country of destination and origin, respectively S_{ict} is a binary variable that takes value one if individual *i* who belongs to five-year cohort *c* smokes at age *t*, and zero otherwise. X_t is a vector of time-varying variables that potentially determine smoking behavior, and α_1 captures a general persistence effect of smoking that reflects not only the cost of quitting but also the effect of other factors that persist over time and are correlated with smoking. Because our objective is to estimate a culture effect, we include our measure of smoking culture, $S^{\tilde{UK}}$, as a separate covariate from the other controls in *X*. Finally, we model the error term as consisting of a permanent individual-specific component α_i, and an individual- and age-varying term ε_{it}.

To measure $S^{\tilde{UK}}$, we follow the epidemiological literature by using data from the country of immigrant origin, but we also advance the literature by refining our measure further. Specifically, we proxy British smoking culture by cohort-specific smoking prevalence rates in the UK net of persistence effects and the effect of any causal contextual factors common with the host countries.[1] To do this, we estimate a model of smoking participation of British natives for each of the host countries. In each model, we control for the average smoking prevalence of the corresponding native-born cohort in the host country, $S_{ct}^{HOST}\left(=\sum\left(S_{ict}^{HOST}\right)/N_{ct}\right)$ if *i* is native-born, which we treat as an endogenous variable. Similar to the model in equation (1), we allow for persistence effects (β_1) and individual fixed effects (β_i). The model we estimate is given by:

$$S_{ict}^{UK}=\beta_1 S_{ic,t-1}^{UK}+\beta_2 S_{ct}^{HOST}+\beta_i+\varepsilon_{it}^{UK} \quad (2)$$

We use the time-varying residual in (2) to construct our proxy of British smoking culture. We first average ε_{it} across all members of each five-year birth cohort. The resulting time series, $\varepsilon_{ct}^{UK}\left(=\sum\left(\varepsilon_{ict}^{UK}\right)/N_{ct}\right)$, consists of that cohort's smoking prevalence rate in every year of life, purged of persistence and shared determinant factors. Then, to every individual in our host country samples, we assign the value of ε_{ct} of the cohort to which her parents

[1] Absent this adjustment, the estimated effect of culture will be biased upwards when the contextual factors that determine smoking are highly correlated across home and host countries and over time. For example, let tax, τ be an element of *X* in (3) so that $X_{ict} = x_{ict} + \tau_t$ and $X_{\tilde{c}-5,t-25} = x_{\tilde{c}-5,t-25} + \tau_{\tilde{t}-25}$. Also, let $\tau_t^{HOST}=\rho\tilde{\tau}_{t-25}^{UK}$. Substitution shows that the culture effect is a function of $\alpha_3\tilde{x}_{c-5,t-25}^{UK}+(\alpha_3/\rho)\,\tau_t^{HOST}$. The latter term is the bias from the common context.

belong, assuming (for now) that parents and offspring are born twenty five years apart. For example, individuals who are 10-15 years old have parents who are 35-40 years old. Similarly, individuals who are 15-20 years old have parents who are 40-45 years old, and so on. Finally, we lag this value by twenty five years. That is, we use the year when the parents' cohort in the UK was the same age as their children currently are.[2] Formally, we set:

$$\tilde{S}^{UK}_{c-5,t-25}=\tilde{\varepsilon}^{UK}_{c-5,t-25} \quad (3)$$

With this variable, we test whether a person of a given age who lives in one of the host countries smokes like his/her parents' cohort smoked when that cohort was the same age and living in the UK.[3] That is, we estimate:

$$S^{HOST}_{ict}=\alpha_1 S^{HOST}_{ic,t-1}+\alpha_2 X^{HOST}_{t}+\alpha_3 \tilde{\varepsilon}^{UK}_{c-5,t-25}+\alpha_i+\varepsilon^{HOST}_{it} \quad (4)$$

By construction, our culture proxy aids identification of a "culture effect" in four ways. First, it measures all determinants of smoking of the parent generation that differ across countries. Thus, it captures all cultural differences that may affect smoking rather than individual aspects of culture, such as religion or a particular social image. Second and following from the above, the proxy is orthogonal to non-cultural determinants of smoking decisions in the host countries ($\tilde{S}^{UK}_{c-5,t-25} \perp X^{HOST}_{t-25}$ and therefore $\tilde{S}^{UK}_{c-5,t-25} \perp X^{HOST}_{t}$).[4] Third, it reflects differences in smoking-related attitudes across generations and over time. Finally, our proxy measures cohort-specific smoking behavior in the UK both before and after the immigrant parents left the country. Thus, although it is likely correlated, it does not exactly match with the parental smoking behavior (which is not reported in our data). Our proxy tests for a correlation between the smoking habits of immigrants and their parents' generation in the UK and not for a correlation between the smoking habits of immigrants and the smoking behavior of their own parents (which could be driven by common unobserved factors unrelated to culture, like family income). The underlying hypothesis is that, independently of their own smoking behavior, immigrant parents transmit smoking-related attitudes or values to their children. For example, if an immigrant parent carries a culture that tolerated (or even condoned) smoking, she will transmit those values (consciously or subconsciously) to her child, increasing the probability that her child smokes.

[2]As an example, consider how we match a UK smoking proxy to a person in Australia who, in the survey year, is 15-20 years old. We assume her parents are 25 years older. That means her cohort is separated by 5 five-year cohorts from her parent's cohort. So, her parent's cohort comprises those who are 40-45 years old in the survey year. We find the cohort of people in the UK sample who are 40-45 years old in that same year. Then we find the value of ε^{UK} of that cohort in the year they were 15-20 years old (t-25).

[3]As an alternative to this specification, one could estimate a reduced-form of equation (1) using $S^{UK}_{c-5,t-25}$ as the culture proxy while controlling for $S^{UK}_{c-5,t-26}$ (persistence) and $S^{HOST}_{c-5,t-25}$ (common context). We prefer to use the two-step approach because of its intuitive appeal, because it allows us to address the potential endogeneity between $S^{UK}_{c-5,t-25}$ and $S^{HOST}_{c-5,t-25}$, and because it enables us to isolate and graphically depict the identifying variation of our "net" culture effect.

[4]Note that from this it does not immediately follow that for children of native-born parents $\tilde{S}^{UK}_{c-5,t-25} \perp S^{HOST}_{ict}$, since British smoking culture may independently affect whether children of native-born parents smoke (e.g. it transmits through vintage British movies).

Due to the restrictive model specification, it is plausible that α_3 underestimates true culture effects. Because they condition on individual fixed effects and past smoking behaviour, the residuals of equation (2) may exclude variation which intuitively would reflect smoking culture. The fixed effects clean out cultural traits related to smoking that are constant over the life-course (e.g. a fixed proclivity to start smoking) and the lagged dependent variable cleans out culturally-driven smoking persistence. We opt to control for fixed effects because these could also reflect unobserved time-invariant individual characteristics unrelated to culture, such as fixed personality traits, completed schooling, sex, race, etc. We opt to include the lagged dependent variable because this could also reflect persistence effects unrelated to culture, such as addiction to smoking driven by a genetic predisposition. Thus, our estimate of α_3 reflects exclusively culture effects that vary over the life-course, across generations, or across calendar years, such as the attitudes about starting and quitting smoking at a given age (e.g. some cultures may equate smoking with masculinity and may explicitly or implicitly celebrate when boys start to smoke at a young age. Other cultural norms may sanction women who smoke, especially after they marry and expect to bear children).

Despite the restrictive specification, our definition of culture still reflects a “black box” whose internal workings are not readily understood. In essence, our estimate of α_3 captures the gross effect of all factors and characteristics that co-vary across the life-course of British immigrants and earlier non-migrant cohorts, which we cannot include in X due to lack of data. Were these factors and characteristics observable, we would obtain a clearer idea about how smoking culture develops and diffuses. However, like other empirical studies that follow the epidemiological approach to estimate culture effects, we cannot identify the mechanisms of cultural transmission. Our priors of these mechanisms are shaped by the available economic theory which predicts that all forms of culture transmit either via societal influences or via parental socialization efforts in the family (Bisin and Verdier 2001). In his review of transmission channels specific to smoking, Nichter (2003) lists parenting styles, role models, peer relations, social perceptions of the timing of adolescence, gender roles, and aesthetics. More specific examples of how culture transmits dynamically include, among others, the release of influential movies, TV series, or advertisements that promote smoker role-models; goverment campaigns and parental socialization efforts that discourage smoking; and the influence of peers in education and the labor market.[5]

Although it goes beyond the scope of this paper to formally model these channels of transmission, our data allow us to test for gender-specific links. In the family environment, it is mothers who are traditionally the primary care-givers[6] and, thus, their parental socialization efforts may play a more important role relative to that of the fathers. In society, feminist movements and the tobacco industry have used propaganda to present smoking by women as an act of liberation, independence, and gender equality (Kaufman and Nichter

[5]For instance, the offspring of British immigrants may exhibit more UK-like age-specific smoking patterns because their parents enroll them in schools that are more similar to the UK educational environment. Since this choice will also determine the children's peers, the estimated effect of the UK culture could reflect peer-effects. We thank one of our anonymous referees for making this point.

[6]The Panel Study of Income Dynamics (PSID) - Child Development Supplement/Transition to Adulthood (CDS-TA) surveys provides data for the US. The CDS-TA surveys interviews the person in the PSID household who identifies herself/himself as a child's ‘primary’ care-giver (PCG). In 2002 and 2007, over 90 percent of the self-identified care-givers were biological, adoptive, or step mothers.

2010). Both these processes suggest that cultural transmission of smoking may be gendered - a hypothesis that we test empirically with US data.[7]

2.2 Methods

We consider two methods to estimate our models: the Arellano and Bond (1991) difference GMM and its augmented version, the Arellano and Bover (1995) system GMM. The difference GMM purges fixed effects by taking first differences, and instruments the (differenced) lagged dependent variable and other endogenous regressors using their lags in levels. Because the lagged levels of the regressors may be poor instruments for the first-differenced regressors, the system GMM estimator consists of two simultaneously determined equations: one differenced and one in levels. The variables in levels in the second equation are instrumented with their own first differences. This specification increases efficiency (Roodman, 2006). While both methods have characteristics that recommend them, we reject the system GMM estimator because one of its key identifying assumptions is probably violated in our data. In particular, the system GMM estimator requires that individual deviations from the long-run mean of the dependent variable (conditional on the independent covariates) be uncorrelated with the fixed effects (Blundell and Bond, 1998; Roodman, 2009). In our data on smoking participation, we expect the deviation from the long run mean of S_{it} to be zero for individuals who never smoke, and non-zero for individuals who do smoke for some period in their lives. We also expect these deviations to be correlated with the factors that our fixed effects capture, e.g. completed education. We use the difference GMM estimator because it is not sensitive to this correlation.

In estimating the models, we have paid special attention to the choice of instruments for our endogenous variables. The literature warns that, depending on the time dimension (T) of the panel, difference GMM can generate an instrument count that is large relative to the sample size (Roodman, 2009). For $T = 3$, the method produces one instrument per endogenous variable, but as T grows the instrument count can explode. When one uses too many instruments it is easier to overfit the endogenous variable and thereby fail to account for its endogeneity. Moreover, the Hansen tests of overidentifying restrictions are vulnerable to instrument proliferation. In this situation, they more often fail to detect overfitting. To avoid this problem we significantly restrict the number of lags that we use as instruments. In doing so, we aim to choose lags that capture, on average, the time of smoking initiation of the individuals in our sample, which is where most of the variation in our dependent variable is concentrated. Because women typically initiate smoking later and over a longer time period than men, we generally use smaller lags for women than men. The exact number of lags we use differs by sub-sample and is guided by the Hansen tests for instrument validity.[8] Finally, to avoid spurious precision from implausibly small standard errors, we apply the Windmeijer (2005) correction in all our estimations.

[7]Elsewhere, we build a formal model that describes the transmission process of smoking culture by adapting the existing theory to smoking behavior and extending it further (Christopoulou et al. 2013).

[8]When we include large numbers of instruments the Hansen tests of overidentifying restrictions produce probability values that are equal to 1, which is the classic symptom of instrument proliferation.

One can also estimate our model with the dymanic random effects probit because it controls for both persistence and individual heterogeneity. An advantage of that estimator is that it produces predictions strictly within the 0-1 range. However, we do not use it because it relies on assumptions that are more difficult to defend. In particular, this estimator requires the assumption that the initial observations, S_{ic1}, of the individuals are exogenous to the random effects. This assumption would hold if our sample included the year of birth for every individual irrespective of individual characteristics (i.e. when $S_{ic1} = 0$). In our Australian sample, we lack tax data for the early years of life of the oldest cohorts and, therefore, we cannot include those years in our estimation. Thus, those data do not satisfy the initial conditions assumption.[9] Further, random effects models are valid only under the restrictive assumption that all regressors are uncorrelated with the random effects.

For each host country, we estimate (4) separately for the children of British immigrants, immigrants from other countries, and native-born parents. We focus on smoking behavior of children of immigrants who moved from the UK. We exclude first-generation immigrants because their decision to smoke and their decision to immigrate may be simultaneously determined. Because second-generation immigrants did not themselves choose to be born in the destination country, bias due to selective migration will be weaker (and will derive from their parents). To the degree that selection bias affects our results, we expect it to work in the opposite direction to the culture effect. This would be consistent with the hypothesis that it is healthy people who select to migrate because they are more able to move, to manage the difficulties of transition, and to undertake often physically demanding work in the destination country. As a result of this selection, immigrants are reportedly healthier upon arrival to the host countries in comparison to the native population (e.g. McDonald and Kennedy 2004; Newbold 2005; Akresh and Frank 2008). Lillard and Christopoulou (2013) provide evidence of a healthy immigrant effect with data specific to the smoking behavior of British immigrants in the US. Given this evidence, we expect that in the presence of selection bias, our evidence of the culture effect will be underestimated.

We also want to exclude immigrants whose connection to the UK operates through a third or higher generation (grandparents, great-grandparents etc) because they are more likely to have assimilated to local culture than second-generation immigrants. Excluding them assures a certain degree of homogeneity of cultural assimilation in our immigrant samples.

3 Data and descriptive evidence

3.1 Sources and definition of panels and sub-samples

To describe life-course smoking trajectories in the home and host countries we use retrospective smoking questions from national cross-sectional and panel surveys. For Australia, we use the 2007 wave of the Household, Income and Labour Dynamics in Australia (HILDA) survey; for the UK we use the 1999 and 2002 waves of the British Household Panel Study (BHPS); and, for the US, we use the 1995, 1996, 1998-2003, 2006,

[9]Heckman (1981) proposes a procedure where one can instrument for the initial values. However, the procedure requires instruments that affect the smoking status in the initial observation of each individual (i.e at birth), but that does not affect his smoking status in subsequent years. We cannot think of any variable that satisfies these requirements.

and 2007 waves of the Tobacco Use Supplement of the Current Population Survey (TUS-CPS).[10] All surveys ask respondents to report four types of smoking behavior: whether respondents ever smoked regularly, whether they currently smoke, what age smokers started to smoke, and when ex-smokers quit.

Together with information on the date of each survey, we use these data to create a smoking status indicator for each respondent in each year of her life (from birth to the survey year). The indicator equals 0 in every year a person did not smoke and it equals 1 in every year she smoked. The indicator "turns on" in the year (at the age) a current or former smoker reported she began to smoke. It stays on (i.e. remains equal to 1) in all subsequent years (up to and including the survey year) if the person currently smokes. For ex-smokers, the indicator "turns off" in the year she said she quit and remains off (i.e. equal to 0) in all subsequent years (up to and including the survey year). Thus, our smoking data follow a panel of individuals over all years of their lives.

The distinct advantage of the long behavioral history from retrospective reports is tempered somewhat by the potential for bias due to bad recall or due to the lack of information on temporary quits. We recognize both possibilities but expect that they pose few challenges to our analysis. Ample evidence suggests that retrospectively reported data more generally, and on smoking behavior in particular, are both valid and reliable (see the literature review in the supplementary material of Lillard et al. 2014). Researchers in the health economics literature increasingly rely on retrospective smoking data, e.g. to examine how individual smoking behaviors vary with price (Douglas and Hariharan 1994, Douglas 1998, Forster and Jones 2001, Nicolas 2002, Kenkel et al. 2009, Lillard et al. 2013).

Another source of concern is that retrospective data do not measure the past smoking behavior of all members of a given cohort because smokers die sooner than non-smokers. Due to this differential mortality, in any given sample of people who survive to answer a survey, one may underestimate past smoking prevalence rates. This issue is especially important for our study because we rely on reconstructed historical smoking trajectories by birth-cohort. We use cause, age, and sex-specific mortality data and the algorithm described in Christopoulou et al. (2011) to correct for this bias. The adjustment doesn't change smoking rates by much except for the oldest cohorts.[11]

The surveys that generate our data also collect information on country of origin, with which we define immigrant and native subsamples. The HILDA and TUS-CPS collect information about each person's mother tongue and the country of birth of himself, his mother, and his father. For those samples, we label a person to be of British "origin" only if he is native-born, speaks English as his mother tongue, and at least one of his parents was born in the UK. The TUS-CPS sample is large enough for us to estimate our models on three subsamples of children of British immigrants - individuals whose parents were both born in

[10]We pool multiple TUS-CPS surveys to maximize the size of the British immigrant sample. However, this strategy results in enormous samples of natives and non-British immigrants. To speed model estimation, we draw random subsamples of these groups, which we use throughout the analysis.

[11]At the very peak of each cohort's lifetime smoking prevalence, the correction adds 2, 5, and 14 percentage points respectively for US men ages 60-69, 70-79, and over 80. The correction adds 1, 2 and 6 percentage points for US women; 0.4, 1, and 8 percentage points for Australian men; and 0.1, 0.4, and 1 percentage point for Australian women (for the same cohorts respectively).

the UK, individuals whose father is the only parent born in the UK, and individuals whose mother is the only one born in the UK.

Our samples include immigrants whose parent's migrated at different ages. Admittedly, some parents migrated as children, had the opportunity and the time to assimilate their host country's culture and, therefore, have weaker links to the British smoking culture compared to parents who migrated as adults. These parents qualify more as second- than as first-generation immigrants, which makes their offspring more similar to third-generation immigrants. Because of the presence of such parents in our samples, the coefficient on culture could be biased towards zero.

To define the “native” and “non-British immigrant” subsamples we use similar selection rules. For Australia and the US, we label a person as of native-origin if he and both of his parents are native-born. We label a person as a non-British immigrant if he is native-born and one or both of his parents were born in any country other than the UK. Finally, when using the UK data to select the people who represent “British” culture, we also apply a very strict selection rule. In particular, we define the “British” sample as UK residents who report their race to be “white British”, were born in the UK, and both their parents were born in the UK.

With each of the sub-samples, we regress the smoking status of each individual on the cultural proxy, cigarette tax, GDP per capita, age and age squared. Taxes and GDP per capita vary only temporally, apart from the US where taxes also vary by state. The per capita GDP data are from Barro and Ursúa (2008), while the tax data are from the “Commonwealth of Australia Gazette” for Australia, and from the “Tax Burden on Tobacco” and state statutes for the US.

3.2 Smoking habits by country and immigrant status

Table 1 provides sample means of smoking participation and our control variables by country, immigrant status, and sex. The smoking data reveal two interesting patterns. First, overall and among all sub-groups, the average smoking prevalence rate in the UK is higher than the corresponding rates in the host countries. Second, the children of British immigrants in both host countries smoke in equal or higher proportion compared to the children of native-born parents and they, in turn, smoke in higher proportion compared to children of immigrant parents from countries other than the UK. These patterns suggest that smoking behavior differs significantly across countries and sub-samples; variation we exploit in the empirical analysis.

It is also informative to examine differences and similarities in the smoking behavior of different birth cohorts. To do this, we create 12 sex-specific birth cohorts of people born in the same five-year period. The cohorts include those who were ages 15-19, 20-24, 25-29, …, 70-74 in 2002.[12] For each cohort of men and women, we compute the smoking prevalence rate in each year (as the mean smoking status indicator weighted by sampling and

[12]We define birth-cohorts in each country that are standardized by calendar and chronological year. We use 2002 as the reference year because it is the latest survey year common to all of the surveys we use.

differential-mortality weights). Figure 1 plots the resulting data for the UK sample. Our measure of British culture will be based on the smoking prevalence rates among the eight oldest British cohorts in the UK, as shown in this figure. Although we observe 12 UK cohorts, because we assume that the average parent is 25 years older than the average child, we can only define the cultural variable for children in the host countries who were younger than 55 in 2002; i.e. for the 8 youngest cohorts.

It is clear in Figure 1 that there is substantial variation in the smoking prevalence rate across cohorts, sex, and calendar years. The prevalence of smoking in each cohort generally follows a bell-shaped pattern over time, reflecting a common pattern of smoking initiation that occurs in a fairly narrow chronological window - during puberty and early adolescence - and a longer period that stretches over the decades of adulthood during which smokers quit (at a much lower annual rate). While the general life-course pattern is similar for men and women in different cohorts, the peak prevalence rates differ. In older cohorts, men smoke more than women, but this difference fades among younger cohorts. For both men and women, smoking prevalence reaches its highest peak for the oldest cohort that entered puberty during World War II. In subsequent cohorts, the peak smoking prevalence rate of men generally declines. Smoking prevalence increases once more for the cohort whose members came of age in the mid- to late-1980s. While we do not investigate formally here, these patterns are consistent with the pattern of increasing awareness and dissemination of information on the health impact of smoking over time[13] and a sharply lower income tax enacted during the Thatcher administration. Among women, the peak rate of smoking prevalence increases and falls more sporadically. Researchers have suggested that some of the rise and fall of the life-course smoking trajectories may be associated with changes in social norms caused by the feminist movement (e.g. see Christopoulou 2015, and studies cited therein).

For each gender and cohort of Figure 1, one can derive three summary measures of the smoking trajectories: the peak smoking prevalence rate, mean age at peak prevalence, and average years of smoking. These measures reflect the popularity, timing, and duration of the smoking habit, respectively. The first two of these measures are directly comparable across genders and birth cohorts. Because we do not observe the full life-course of each cohort (since smoking trajectories stop at the time of interview), one cannot directly compare smoking duration across all cohorts. However, one can compare the level of these variables from a given cohort across genders and countries. We explore this comparability in Figure 2, where we plot the ratio of the three summary measures of British immigrant descendants to their native-born counterparts by cohort, gender, and host country. Our data show that the smoking habits of British decendants in Australia are somewhat closer to those of natives relative to British descendants in the US, especially in younger cohorts. Ratios vary within the 0.5 to 1.5 range for all groups, apart from teenagers and young adults in the US who show ratios closer to 2 or higher.

[13]The first Royal College of Physicians (RCP) report on Smoking and Health was published in the UK in 1962. It received massive publicity. The main recommendations were: restrict tobacco advertising; increase taxes on cigarettes; more strictly regulate sales of cigarettes to children, limit smoking in public places; and provide information on the tar/nicotine content of cigarettes.

These differences fit our expectation that British migrants and natives are culturally more different in the US compared to Australia. The Australian group that we call natives (those who are native-born with two native-born parents) likely includes a big share of British immigrants of third or higher generation. We estimate that the grandparents of the 'native' group (or a large share of them) arrived in Australia over the period 1920-1970. This period overlaps with the 'Big Brother Movement' founded in the Australia in early 1920s (Gill 2005) and the formalization of this movement via the 'Populate or Perish' policy over 1945-1980 (Jupp 2004; Hamerton and Thomson 2005), both of which constitute schemes for preferential migration from the UK.

4 Results

4.1 Measuring smoking culture of British immigrants

Our smoking data provide a clear demonstration of the bias that can arise when one measures immigrant culture with a lagged value of the outcome of interest. Table 2 reports correlation coefficients between smoking participation of immigrants and natives in the host countries (at the individual level) and smoking prevalence rates of the parent generation living in Britain twenty five years earlier. The coefficients are positive, sizable and statistically significant for all sub-samples.

This positive correlation derives from four potential sources. First, a common process may have caused the economies in home and host countries to develop in similar ways. Second, information about health risks of smoking flows freely and (probably) simultaneously to people in all countries; especially when they all speak English. Third, there are good reasons to suspect that countries may have adopted similar tobacco control policies at similar times. For example, for many years the World Health Organization has lobbied countries to adopt a standard set of tobacco control policies to reduce smoking prevalence worldwide. Even independent of that effort, tobacco control policies of the UK, US, and Australia follow similar trends (though states in the US tend to have adopted regulations earlier). Both processes (economic and regulatory) could cause cohort-specific contextual factors to be correlated across countries. Further, to the extent that contextual factors decay slowly over time, this correlation across countries for the parents' cohort will persist and be correlated with contextual factors in the childrens' generation in destination countries. Finally, the smoking participation rate might be correlated across cultures and generations due to factors that are neither contextual nor cultural. As we mentioned above, the correlation may arise because there is a strong biological force that gives rise to similar patterns of smoking across individuals, such as a genetic predisposition to nicotine addiction.

As section 3 describes, we exploit the time dimension of our data to purge contextual factors and persistence effects from smoking prevalence rates in the UK. Specifically, we estimate dynamic models of smoking participation in the UK (at the individual/age level) on smoking prevalence rates of natives in each host country (at the cohort/age level). We remind the reader that we treat smoking prevalence of natives in the host countries as endogenous and, thus, we instrument these variables using lagged values, just like we do for the lagged dependent variable. We average the resulting residuals at the cohort level and treat them as the respective cultural proxy for each host country. Essentially, the residuals represent

differences in smoking behavior of natives in the host countries and in the UK, which are either due to cultural differences or uncorrelated contextual differences.

We present the results of this exercise in Table 3. In all models the coefficient on the lagged dependent variable is high, suggesting a significant persistence in the smoking habit of British natives. The smoking prevalence rates in the host countries are also significant predictors. The estimated coefficients on these variables are positive - a result that is consistent with the fact that the popularity of smoking in Australia, the UK and the US spread, peaked, and started declining at roughly the same time.

The residuals that result from these estimations vary substantially across host countries, genders, cohorts, and years of age. To demonstrate this variation, we plot them in Figure 3. Compared to the values of the dependent variable (i.e. the prevalence rates of natives in the UK presented in Figure 1), the estimated residuals are very low in value and often negative. Negative values indicate that natives in the host country smoke more than natives in the UK, while positive values indicate that natives in the host countries smoke less than natives in the UK. Further, the distribution of the residuals across calendar years is less skewed than that of the gross smoking prevalence rates in Figure 1. This pattern is no surprise considering that the residuals are purged of addiction effects. The most impressive difference is in the year-by-year variation, which is admittedly high in the residual values. A priori, we are agnostic as to what degree this variation represents noise or actual time-varying factors and characteristics that shaped British smoking culture. This is the empirical question which we test in section 4.2.

To further assess the variation in our measure of culture, we report the correlation matrix of the estimated residuals across genders and host countries in Table 4. The residuals are significantly correlated in all cases (as they should be - after all, each one measures the same smoking culture). They differ to the extent that each one has been purged of its common correlation with smoking behavior in a different country. These correlation coefficients already provide a clear indication that culture is gendered. Our estimated residuals are highly correlated between women across countries, less so between men and women within each country, even less between men across countries, and the least between different genders across countries.

4.2 Testing the explanatory power of the culture proxy in the host countries

In Table 5, we report results from regressions of smoking participation in the host countries that include our culture proxies. For British immigrants, we assign sex-specific residuals if a person has only one UK-born parent. For example, we assign children of UK-born fathers the residual of UK men. If both parents are UK-born, we assign the average of the UK residuals of the two sexes. In all other cases, we assign men the residuals of UK-born males and women the residuals of UK-born females.

As in Table 3, the lagged dependent variable absorbs a lot of the variation in smoking participation. In most cases, coefficient differences between immigrants and natives are statistically insignificant, suggesting that the persistence of the smoking habit is independent of immigrant status. Australian males are an exception; the estimated persistence effects are

significantly lower for second-generation immigrant men from Britain than for native men and second-generation immigrant men from other countries.

Our cultural proxy significantly predicts smoking participation of both sons and daughters of British immigrants in Australia and the US. We estimate that, every 1 percentage point increase in the "residual" smoking prevalence of the parent generation in the UK increases the probability of smoking participation among the children of British immigrants by between 0.1 to 0.4 percentage points.[14] This result implies that immigrants have not fully assimilated their host country's culture - at least with regards to that part of culture that predicts smoking habits. Instead, their choices are influenced by smoking preferences that they inherited from a parent born in the UK.

Our assertion that we capture a true culture effect finds further support when we fit the model on data from the people whose parents migrated to Australia and the US from a country other than the UK. For those groups, in both countries, the culture proxy is uncorrelated with smoking participation. This result is in line with the higher ethnic diversity between these groups and British immigrants. Finally, our proxy does not significantly predict smoking participation of people whose parents were native-born in each of the host countries. Encouragingly, this result suggests that there is no link between the smoking behavior of the previous generation who lived in Britain and of the current generation of natives in the host countries, other than through the common context shared by the previous generations in the host countries and the UK.

As an additional exercise, and to further explore the significant culture effect for British immigrants, we test whether this effect is significant and similar for people with only a UK-born mother, only a UK-born father, or with both. We limit this exercise to the US sample because only the TUS-CPS has the data and a large enough sample to test whether smoking culture travels through gender-specific channels.[15] Table 6 reports results for different model specifications estimated on these samples. In the first three columns we report results for specifications estimated on the sample of individuals whoseparents were both born in the UK. The underlying model for column 1 measures smoking culture as the average culture of UK men and UK women (in the appropriate cohorts). The underlying model for column 2 includes the smoking culture of UK men only. The underlying model for column 3 includes the smoking culture of UK women only. Columns 4 and 5 present results for the samples with a UK-born father only and a UK-born mother only. Their underlying models use the sex-specific measures of smoking culture in the UK.

[14]To get a better idea about the magnitude of this effect, consider the case of British migrant women in Australia, for whom we find the largest coefficient (0.395). Our culture proxy for this group has a mean of 0.005 and a standard deviation of 0.028. Given the linearity assumption of the linear probability model, the marginal effect of every one-standard deviation increase in the culture proxy (i.e. an increase of 2.8 percentage points) is a 1.11 percentage point increase in the smoking rates of British migrant women. As shown in Table 1, the mean rate of smoking prevalence for this group is 21.9%. The marginal effect would increase this rate to 23.01%, which constitutes a 5.1% increase.

[15]A related empirical literature examines whether children are more likely to smoke if their parent smokes or ever smoked. This literature is rife with statistical weaknesses and 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.

We find that all measures of culture have significant and equal predictive power for British immigrant men. Differences in the estimated culture effects between columns (2) and (3) and columns (4) and (5) are statistically insignificant. The implication is that the smoking culture of mothers' and fathers' cohorts equally affect the smoking participation of sons. For British immigrant women, however, the results are different. The mean culture of males and females significantly predicts the smoking participation of daughters of immigrants when both parents were born in the UK. However, when we control for female and male smoking culture separately, only the female culture appears significant. Consistently, female culture predicts the smoking participation of daughters of immigrants whose mothers were born in the UK and fathers were born elsewhere, but male culture does not predict the smoking participation of women whose fathers were born in the UK if their mothers were born elsewhere. These results suggest that daughters inherit the smoking culture of their mothers' cohort but not the culture of their fathers' cohort.

Together, the estimates in Table 6 support our prior that women have a special role as bearers of smoking culture. Because mothers typically spend more time at home taking care of the children, it is plausible that they transmit their smoking culture via the parental socialization channel to both sons and daughters. The paternal influence is weaker because fathers spend less time with their children and/or because they only discuss matters related to smoking with sons, since smoking has long been customary for men and a taboo for women. Womens' smoking habits may also transmit strongly though the societal channel due to changes in norms and attitudes about gender roles brought about by the feminist movement. It is plausible that, because of feminist influences, women who migrated from the UK had an additional incentive to preserve their smoking culture after arrival (which men did not have), and they also had an incentive to transmit that culture equally to their daughters and sons.

All the results we have reported are based on the assumption that parent and children generations are 25 years apart. Other studies in the literature have used a more conservative generation gap of 20 years (e.g. Fernandez and Fogli 2009). We chose the 25 year gap because our data contain significant heterogeneity by gender and birth-cohort and do not allow us to observe each individual's birth order, whether she has siblings, or her parents' ages. Our aim was to define a gap that better approximates the temporal distance between the average child and the average parent in the average cohort represented in our data. In estimates not shown here, we tested whether the results in Table 5 change when we set the generational gap to 20 years. We find that the estimated culture effects are both qualitatively and quantitatively robust to this change (the differences between the new culture coefficients and those reported in Table 5 are statistically insignificant for all groups).

5 Conclusion

A growing economic literature argues that culture predicts important economic and social outcomes, but the bulk of the available evidence derive from cross-sectional data under the assumption that culture changes little over time. Our paper highlights the fact that there are a class of economically important behaviors that are plausibly influenced by cultural factors that are not time-invariant. Social scientists are keenly interested in understanding how

cultural attitudes develop with respect to women, labor markets, and health. We propose a technique that exploits time-variation, made available by retrospective or longitudinal data, to measure a changing culture. This technique can be applied and extended to utilize similar data, as these are becoming increasingly available and as they increasingly measure a larger variety of behaviors.

By relying exclusively on temporal and intergenerational variation, in this paper we show that culture is a significant determinant of smoking participation. Specifically, we show that culture matters for the smoking behavior of sons and daughters of British immigrants in Australia and the US. With US data, we also find that the smoking culture of women is gendered; while sons of British immigrants adopt the smoking culture of both parents' cohorts, daughters of British immigrants adopt the smoking culture from their mothers' cohort only. Finally, we find that our cultural proxy does not significantly predict the smoking behavior of natives in the host countries, and more encouragingly, it does not predict that of children of immigrants from countries other than Britain.

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Highlights

We show that culture determines smoking decisions.

Our measure of culture varies by age, calendar year, and across birth-cohorts.

Our measure is net of any shared variation in smoking patterns across countries

When life-course data are available, our approach can be applied to other outcomes.

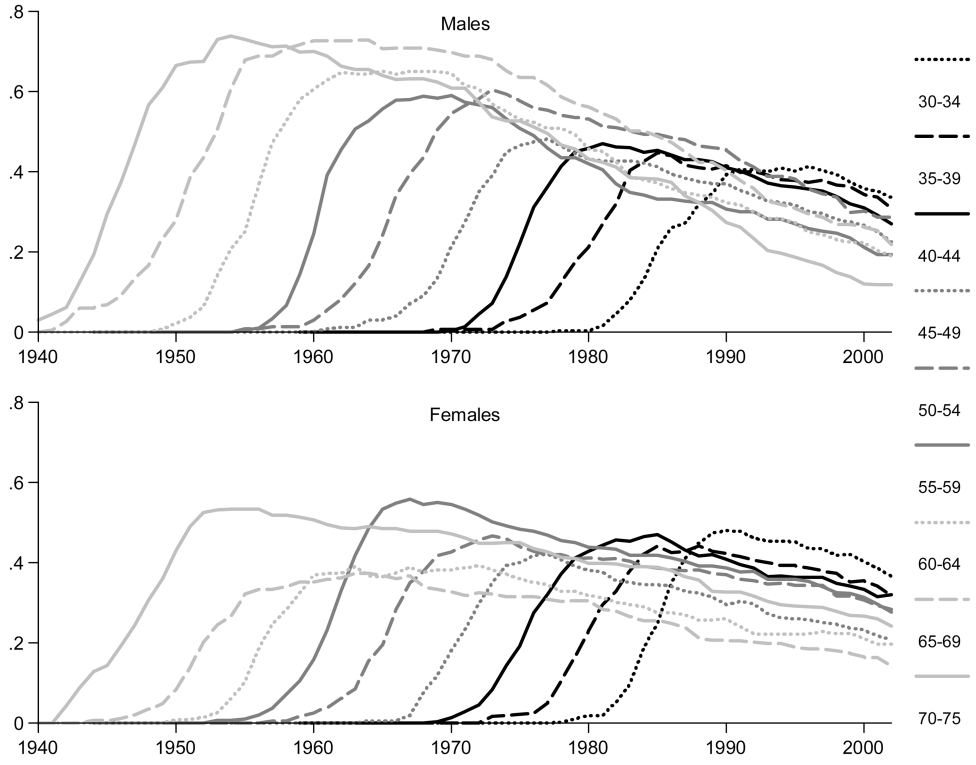

Figure 1. Life-course smoking prevalence of natives in the UK by birth cohort (age in 2002)

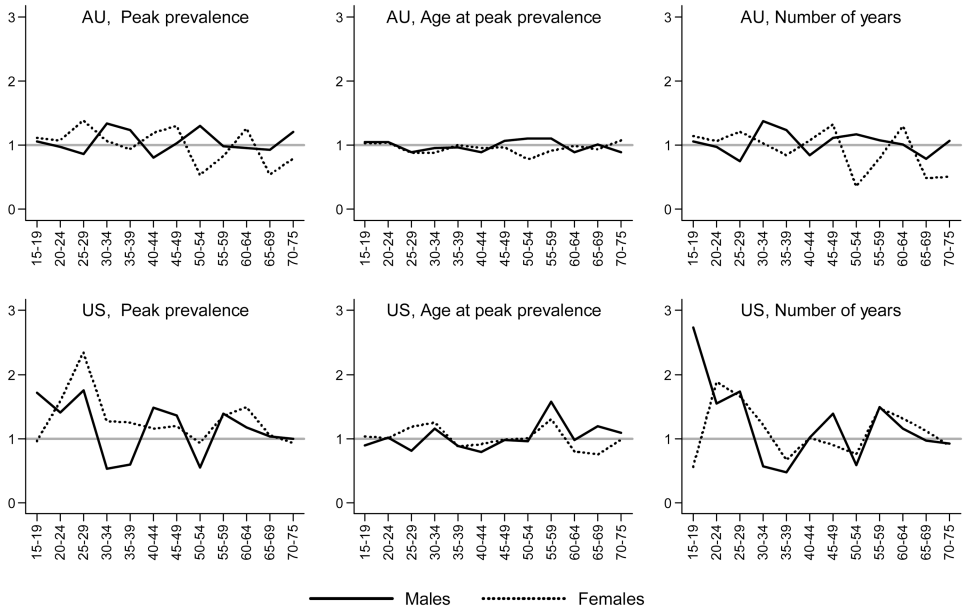

Figure 2. Summary indicators of smoking trajectories in relative terms between British immigrants and natives across birth-cohorts

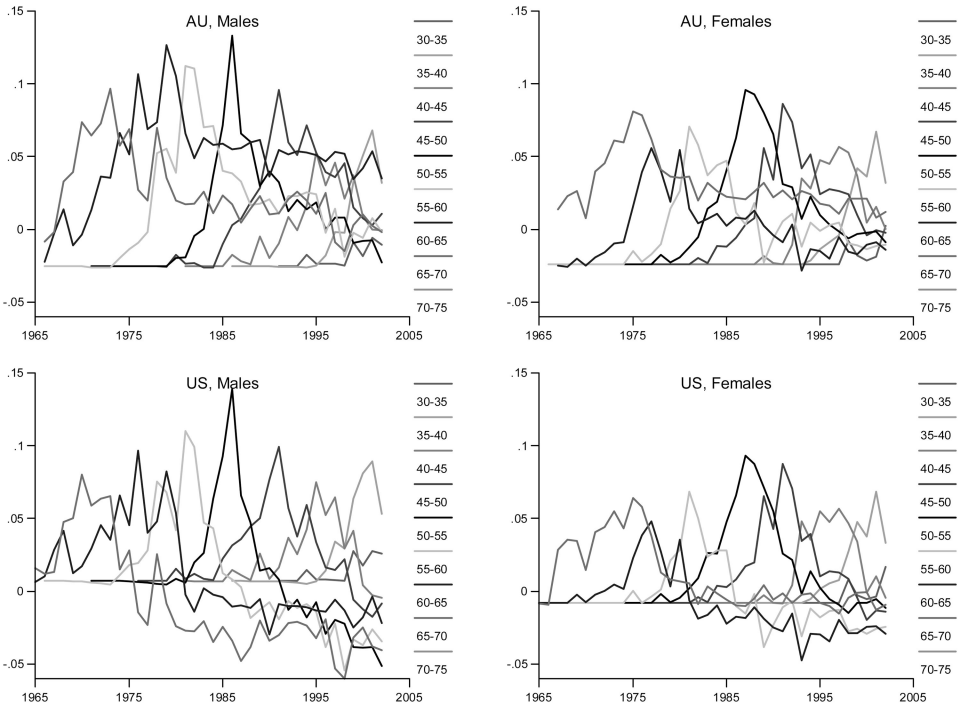

Figure 3. Residuals by host country, sex, and birth cohort

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Table 1

Sample means of smoking participation and age at interview

		Smoking prevalence		Age at interview	
		Males	**Females**	**Males**	**Females**
Australia	All	25.2	21.5	38.0	37.6
	British	24.4	21.9	35.2	35.6
	Native	25.8	21.9	38.6	38.2
	Other	21.6	18.2	36.8	35.5
UK	All	31.8	27.7	48.5	48.6
	British	30.4	26.8	49.0	49.1
USA	All	18.8	16.1	33.7	34.1
	British	22.6	20.8	36.1	36.2
	Native	21.3	18.3	35.1	35.4
	Other	12.4	9.3	29.8	30.4

Notes: Sampling weights applied in all cases. *Sources*: Australia - HILDA (2007), UK - BHPS (1999 BHPS (2002), US - TUS-CPS (1995, 1996, 1998-2003-2006-2007).

Table 2

Correlation coefficients among cohort-specific smoking prevalence in the UK and smoking participation in each host country by sex and immigrant status

	Australia		USA	
	Males	**Females**	**Males**	**Females**
British	0.4627*	0.4358*	0.4103*	0.3468*
Native	0.4543*	0.4018*	0.4100*	0.3572*
Other	0.4542*	0.3931*	0.3348*	0.2878*

Notes:

*significantly differs from zero with a probability value<0.05

Table 3

Difference GMM estimation of smoking participation of British in the UK on cohort-specific smoking prevalence of natives in the host countries

	Australia		USA	
	Males	**Females**	**Males**	**Females**
Lagged dependent variable	0.745 [0.025]*	0.830 [0.020]*	0.826 [0.023]*	0.858 [0.015]*
Sm. prevalence in host country	0.208 [0.039]*	0.136 [0.019]*	0.234 [0.033]*	0.169 [0.022]*
Person-year observations	93624	112207	88674	107322
Number of persons	2380	2874	2380	2874
Instruments	704	1415	1036	1461
Hansen test	722.8 (0.285)	1465.4 (0.162)	1060.6 (0.276)	1464.7 (0.453)

Notes: Windmeijer-corrected standard errors are in brackets. Probability values are in parentheses.

*significantly differs from zero with a probability value<0.05

Table 4

Correlation matrix of estimated residuals by host country and sex

		Males		Females	
		Australia	**USA**	**Australia**	**USA**
Males	Australia	1.000			
	USA	0.6241	1.000		
Females	Australia	0.7253	0.5112	1.000	
	USA	0.5596	0.7715	0.8581	1.000

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Table 5

Difference GMM estimation of smoking participation by host country, sex, and immigrant status

	Australia			USA		
	British	**Native**	**Other**	**British**	**Native**	**Other**
Males						
Culture	0.233 [0.104]*	0.035 [0.022]	-0.009 [0.090]	0.144 [0.030]*	0.082 [0.042]	0.020 [0.056]
Lagged sm. participation	0.356 [0.023]*	0.592 [0.098]*	0.717 [0.209]*	0.877 [0.031]*	0.793 [0.047]*	0.801 [0.064]*
Person-year observations	10237	70281	8281	56792	84070	54468
No. of persons/Instruments	367/33	2378/199	275/29	1874/180	2906/209	2304/103
Hansen test (for lagged instruments)	22.7 (0.703)	202.8 (0.300)	20.1 (0.634)	170.1 (0.570)	151.7 (0.997)	85.7 (0.787)
Difference-in-Hansen test (for independent variables)	5.5 (0.356)	7.0 (0.222)	4.6 (0.461)	6.6 (0.251)	7.5 (0.186)	6.7 (0.240)
Females						
Culture	0.395 [0.113]*	0.069 [0.062]	-0.046 [0.131]	0.093 [0.037]*	-0.015 [0.018]	-0.014 [0.033]
Lagged sm. participation	0.515 [0.177]*	0.857 [0.029]*	0.618 [0.125]*	0.966 [0.019]*	0.883 [0.034]*	0.950 [0.054]*
Person-year observations	10717	79807	8014	59017	93556	59956
No. of persons/Instruments	380/48	2710/291	272/29	1926/251	3160/253	2437/105
Hansen test (for lagged instruments)	46.2 (0.302)	287.4 (0.450)	20.0 (0.640)	234.1 (0.681)	229.3 (0.785)	91.9 (0.682)
Difference-in-Hansen test (for independent variables)	4.5 (0.484)	3.6 (0.608)	9.2 (0.102)	0.5 (0.993)	5.3 (0.378)	2.1 (0.833)

Notes: Regressions control for log taxes, log GDP per capita, age and age squared. Windmeijer-corrected standard errors are in brackets. Probability values are in parentheses.

* significantly differs from zero with a probability value<0.05. In all specifications estimated for Natives and Others culture is sex-specific. For British immigrants culture corresponds to the gender of each individual's British parent when only one parent was born in the UK, and is the mean of male and female cohorts for individulas with both parents born in the UK.

Table 6

Difference GMM estimations of sm. participation of British sub-groups in the US by sex

	Both parents born in UK			Only father born in UK	Only mother born in UK
	(1)	(2)	(3)	(4)	(5)
Males					
Culture	0.542 [0.213]*	0.335 [0.167]*	0.552 [0.204]*	0.202 [0.092]*	0.097 [0.049]*
Lagged sm. participation	0.869 [0.416]*	0.892 [0.349]*	0.900 [0.307]*	0.969 [0.025]*	0.934 [0.020]*
Person-year observations	10019	10019	10059	15608	31165
No. of persons/Instruments	314/16	314/14	314/15	545/72	1015/152
Hansen test (for lagged instruments)	8.2 (0.613)	8.8 (0.361)	6.9 (0.643)	72.0 (0.287)	129.1 (0.840)
Difference-in-Hansen test (for independent variables)	5.1 (0.403)	4.8 (0.437)	3.0 (0.701)	5.1 (0.404)	5.1 (0.410)
Females					
Culture	0.376 [0.188]*	0.168 [0.163]	0.442 [0.174]*	0.0012 [0.054]	0.173 [0.087]*
Lagged sm. participation	0.9013 [0.120]*	0.931 [0.098]*	0.9433 [0.433]*	0.976 [0.072]*	0.918 [0.026]*
Person-year observations	11267	11267	11340	15983	31767
No. of persons/Instruments	359/14	359/18	359/17	542/74	1025/122
Hansen test (for lagged instruments)	10.7 (0.222)	14.6 (0.264)	11.0 (0.444)	79.0 (0.169)	122.3 (0.327)
Difference-in-Hansen test (for independent variables)	7.4 (0.190)	8.6 (0.125)	6.0 (0.309)	5.0 (0.417)	9.1 (0.107)

Notes: In specification (1) culture is calculated as a mean of male and female cohorts; in (2) and (4) it corresponds to that of male cohorts; and in (3) and (5) it corresponds to that of female cohorts. Because for British with both parents born in the UK the sample size is relatively small, we have collapsed the instruments to avoid over-fitting (i.e. we have allowed one instrument per lag distance, rather than one instrument per time period and lag distance). All other information, as for Table 5.