The role of culture in smoking behavior: evidence from British immigrants in Australia, South Africa, and the US*

Rebekka Christopoulou† and Dean R. Lillard

Cornell University

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Abstract

We exploit historical migration patterns from the UK to Australia, South Africa, and the US to investigate whether culture determines smoking habits. 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 culture proxy significantly predicts life-course smoking participation of second-generation British immigrants but not that of non-British immigrants and natives. Researchers can generally apply our strategy to measure culture when retrospective or longitudinal data are available.

Keywords: Immigrant health, Smoking, Culture

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†Email: rc448@cornell.edu

1 Introduction

Research documents large differences in whether and how much people smoke by ethnicity (Ellickson et al. 2004, Acevedo-Garcia et al. 2005, Wallace et al. 2003, 2009 etc.), and country of residence (Piko et al. 2005, Keaweaimoku et al. 2006, Agyemang et al. 2010). Here, we ask whether such differences in smoking behavior are influenced by culture. We use a natural experiment design that exploits historical patterns of migration from Great Britain to other Commonwealth countries (Australia, South Africa) and the US. If British immigrants assimilate completely in their new environment, their smoking behavior should depend on the national context alone; it should not be correlated with smoking habits in Britain.

Conceptually, culture is associated with norms or values that are shared by members of small groups such as families or larger groups defined by place of residence, religion, or ethnicity. Because of this vague definition, it is difficult to model and empirically measure culture with standard quantitative data. Therefore, economists are mostly skeptical about including it in empirical models of behavior (Guiso et al. 2006 review early studies). Despite this skepticism, they widely recognize that, both conceptually and intuitively, differences in culture potentially explain systematic variation in outcomes of interest.

A new strand of literature has emerged in economics that investigates 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 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 home country, its empirical strategy is sometimes termed the "epidemiological" approach (see Fernandez 2010 for a recent review). 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 violation by diplomats is

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 contribute to this growing body of economics research on culture. In each of the host countries, we use a cross-sectional or panel survey 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 information on country of origin we define samples of British immigrants, immigrants from other countries, and natives. We then relate smoking participation at every year of life of each group 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. Our proxy should be significant for British immigrants if parents carried cultural beliefs with them when they emigrated from the UK and if they transmitted those beliefs to their children. Our proxy should not be significant for other immigrants and natives.

This exercise is innovative in two ways. First, we measure culture as the outcome variable (smoking) in the UK only after we net out any non-cultural determinant factors 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, Australia, and South Africa 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. In his review, Nichter (2003) argues that smoking-related culture is a mix of gender roles, parenting styles, role models, types of peer relations, perceptions of the timing of adolescence, and aesthetics. Our measure captures all such (non-contextual) factors that drive smoking behavior and that differ across home and host countries.

Second, our strategy exploits changes in smoking-related attitudes across time. Due to data limitations, the bulk of the extant research tests the influence of immigrant culture on outcomes of interest with measures of culture that do not vary over time. They only vary across the different countries of origin. 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, if 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. 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. Because our measure of smoking culture varies by cohort and year of life, it captures the evolution in attitudes about smoking more fully.

One can generally apply these two methodological improvements to any data that allow one to construct life histories. In our case, the retrospective nature of our data allow us to track lifetime smoking histories. However, researchers can apply our approach to study any type of behavior reported retrospectively or longitudinally. For example, if Fernandez and Fogli (2009) had the complete fertility histories of their survey respondents, they could measure immigrant culture as the cohort-specific fertility rates in their country of origin, and purge it of any non-cultural determinant factors correlated across home and host countries. Educational attainment and age at first marriage

can also be studied in the same way.

Our exercise contributes new evidence that suggests that culture might causally affect behavioral 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 and country of immigrant destination. We argue that these differences may be explained by the influence of feminist smoking related attitudes; by the degree of ethnic homogeneity in the host countries; by gender-differences in health-based selection to migration; and by gender differences in the timing of migration.

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

To frame our empirical analysis, consider a model of smoking participation where the cost of quitting is fixed. An individual i who belongs to cohort c and is of age t smokes $(S_{ict} = 1)$ if current utility from smoking u_{ict} is greater than current costs c_{ict} of smoking minus a cost of quiting $\bar{q}$. One can think of this cost as a utility loss due to nicotine withdrawal that rises linearly with the amount one consumes:

$$S_{ict} = \begin{cases} 1 & if\ u_{it} > c_{ict} - \bar{q}S_{ict-1} \\ 0 & otherwise \end{cases} \tag{1}$$

To implement the model statistically, we follow the standard latent variable approach that specifies the observed data as a binary outcome which is observed if an index passes a critical value. The index is posited to be linear in a set of observed covariates. Under standard assumptions we

estimate the relationship of the covariates using a reduced relationship of the form:

$$S_{ict} = \begin{cases} 1 & if\ \alpha_1 S_{ict-1} + \alpha_2 X_{ict} + \eta_{it} > 0 \\ 0 & otherwise, \end{cases} \tag{2}$$

where X_{ict} is a vector of time-varying variables that potentially determine smoking behavior, and α_1 captures a more general persistence effect of smoking that reflects not only the cost of quitting, $\bar{q}$, but also the effect of other factors that persist over time and that are correlated with smoking.

We further modify (2) in two ways. First, in choosing an estimation strategy for this binary-choice framework, one needs to decide whether unobserved individual heterogeneity is better approximated by fixed or random effects. To use random effects the individual effects must be uncorrelated with the regressors. Because this condition is unlikely to hold for smoking participation, we specify a model that includes individual fixed effects.[1] Thus, we model the error term η_{it} as consisting of a permanent individual-specific component α_i, and an individual- and age-varying term ε_{it}. Second, because our objective is to estimate a culture effect, we include our measure of British smoking culture, $\widetilde{S}^{UK}$, as a separate covariate from the other controls in X. For each host country, we estimate a linear probability model of the form:

$$S_{ict}^{HOST} = \alpha_1 S_{ic,t-1}^{HOST} + \alpha_2 X_{ict}^{HOST} + \alpha_3 \widetilde{S}_{c-5,t-25}^{UK} + \alpha_i + \varepsilon_{it}^{HOST} \tag{3}$$

where c comprises a 5-year range of birth-years and the label $HOST$ (UK) identifies variables measured in the country of destination (origin).

To construct $\widetilde{S}^{UK}$, we follow the epidimiological literature and measure culture in the country of immigrant origin. That is, we proxy for British smoking culture using cohort-specific smoking prevalence rates in the UK. However, we also advance the literature by further refining our proxy; namely, we purge it of persistence effects and the effect of any causal contextual factors common in the home and host countries.[2] To do this, we estimate three models of smoking participation of British natives - one for each of the host countries that we study. In each model,

we control for the average smoking prevalence of the corresponding cohort in the host country, $S_{ct}^{HOST}\left(=\sum(S_{ict}^{HOST})/N_c\right)$. Similar to the model in equation (3), we allow for persistence effects (β_1) and individual fixed effects (β_i). The model we estimate is given by:

$$S_{ict}^{UK} = \beta_1 S_{ic,t-1}^{UK} + \beta_2 S_{ct}^{HOST} + \beta_i + \varepsilon_{it}^{UK} \tag{4}$$

We use the time-varying residual in (4) 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, $\varepsilon_{ct}^{UK}\left(=\sum(\varepsilon_{ict}^{UK})/N_c\right)$, consists of that cohort's smoking prevalence rate in every year of life, purged of persistence and shared contextual factors. Then, to every individual in our host country samples, we assing the value of ε_{ct} of the cohort to which her parents 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.[3] Formally, we set:

$$\widetilde{S}_{c-5,t-25}^{UK} = \varepsilon_{c-5,t-25}^{UK} \tag{5}$$

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.

By construction, our culture proxy aids identification of a "culture effect" in four ways. First, it measures all non-contextual determinants of smoking that differ across countries. Thus, it captures all cultural differences that may affect smoking rather than individual aspects of culture, such as religion, style etc. Second and following from the above, the proxy is orthogonal to other (non-cultural) determinants of current smoking decisions in the host countries ($\widetilde{S}_{c-5,t-25}^{UK} \perp X_{ict}^{HOST}$). Third, it reflects the variation of smoking-related attitudes across generations and over time. Fi-

nally, 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). As such, our proxy tests for a correlation in smoking habits of immigrants and their parent generation in the UK and not for a correlation in smoking habits of immigrants with their own parents. The underlying hypothesis is that smoking-related values travel from immigrants parents to their kids independently of whether or not the immigrant parents themselves smoke. If an immigrant parent grew up in a culture that tolerated (or even condoned) smoking, she will carry and transmit those values (consciously or unconsiously) to her child, thus, increasing the probability that the child becomes a smoker.

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. education. Therefore, 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.[4]

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 and South African samples, 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.[5] Further, as noted previously, random effects models are valid only under the restrictive (and we believe invalid) assumption that all regressors are uncorrelated with the random effects.

For each host country, we estimate (3) separately for the children of British immigrants, im-

migrants from other countries, and native-born parents. We focus on offspring of immigrants themselves 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). Lillard and Christopoulou (2011) provide evidence of a healthy immigrant effect with data on 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. As we describe below, the precision with which we are able to define each sample varies with the information available in each country's survey.

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 South Africa we use the 2008 wave of the National Income Dynamics Study (NIDS); 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, and 2007 waves of the Tobacco Use Supplement of the Current Population Survey (TUS-CPS).[6] All surveys ask respondents to report four types of smoking behavior: whether respondents ever smoked regularly, whether they currently smoke, what age they 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. 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. For ex-smokers the indicator 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 fact that our analysis relies on retrospective reports on smoking behavior may raise concerns of bias due to bad recall or due to the lack of information on temporary quits. While we acknowledge both possibilities, we are confident that the size of bias will not be significant. Ample evidence suggests that retrospectively reported data more generally (Berney & Blane, 1997; Koenig et al., 2009), and on smoking behavior in particular (Christopoulou et al. 2011; Brigham et al., 2010; Kenkel et al., 2003), are both valid and reliable. Another source of concern is that retrospective data do not measure the past smoking behavior of all members of a given cohort because some have died by the time of interview. This issue is important because we use the data to characterize the smoking behavior of a given cohort in past years and 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. To correct for this bias we use cause, age, and sex-specific mortality data and the algorithm described in Christopoulou et al. (2011). The adjustment doesn't change smoking rates by much except for the oldest cohorts.[7]

The surveys that generate our data also collect information on country of origin. With this information we define immigrant and native subsamples but with varying degrees of precision. For example, 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 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. In South Africa the NIDS does not ask respondents to report their mothers' or fathers' country of birth. However, it collects information about each respondent's country of birth, language, and race. We use those data to label a respondent as of British-origin if he was born in South Africa, speaks English as a mother tongue, and is "white" or "colored."

These rules select samples in Australia and the US that precisely fit our objective - all respondents are native born and all have at least one parent who was born in the UK - but only imprecisely for the South African sample. Our British immigrant subsample in South Africa will include children or descendants of English-speaking immigrants who do not meet our preferred selection rule. The South African sample will include native-born individuals whose UK heritage flows from grandparents or older ancestors. It will also include people whose mother tongue is English (i.e. those who "speak English at home" or "prefer to speak English" or "read and write English very well"), but who come from other countries where English is spoken (e.g. the US, Canada, India, Pakistan). The first type of person has a direct (but presumably weakening link to UK culture). The second type of peson has (presumably) even weaker links to British culture.

The presence of such people will potentially introduce two sources of downward bias into the coefficient on culture. First, if immigrants increasingly adopt local culture in successive generations then the smoking behavior of parents will reflect less of UK culture when the historical connection is temporally more distant. Second, for respondents whose relatives were locals or

immigrated from an English-speaking country other than the UK, we expect UK culture to matter less. Both sources of contamination bias our coefficient on the culture proxy towards zero. If we do find evidence of an effect of culture on smoking behavior in South Africa, it is likely to be a lower bound estimate.

Similar issues arise in defining the "native" and "non-British immigrant" subsamples because we use the same information in our selection rules. For Australia and the US, we label a person as of native-origin if she and both of her parents are native-born. We label a person as a non-British immigrant if she is native-born and one or both of her parents were born in any country other than the UK. In the South African sample, we label a person as "native" if she is not white, was born in the country, and her preferred language is not English. Again, this selection rule is restrictive. Because we cannot distinguish between parents who were native-born and those who were born abroad, we do not define a non-British immigrant sample for South Africa. In this case, non-British immigrants are included in the "native" group.

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 US where taxes vary by state. The per capita GDP data are from Barro and Ursúa (2008) while the tax data are from various national sources (see Table 1).

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 three interesting patterns. First, overall and among all sub-groups, the average smoking prevalence rate in the UK is higher than the rate in the host countries. Second, the smoking prevalence rate is almost identical for children of native-

born parents and children of British immigrants in Australia. For those groups it differs little in the US. But in South Africa, the difference is sizable, especially for women. The proportion of South African women who smoke is almost six times higher among women with a UK heritage than it is among South African women with some other heritage. Third, in both the US and in Australia, children of immigrant parents from countries other than the UK appear to smoke less than children of parents who were both born in the country or children who have at least one parent who immigrated from 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 the smoking behavior of different birth cohorts. To do this we first identify members of the same sex who were born in different calendar periods; namely, those who were ages 15-19, 20-24, 25-29, ... , 70-74 in 2002.[8] Then, we compute each cohorts' smoking prevalence rate (by sex) over their whole life-course. We computed this rate as the mean smoking status in each year (weighted by sampling 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. Because we observe 13 cohorts overall in the UK and because we assume that parents and children generations are 25 years apart, we can only define the cultural variable for those in the host countries that 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 declines. While we do not investigate formally here, we speculate that this pattern is consistent with the pattern of increasing awareness and dissemination of information on the health impact of smoking over time.[9] For men, smoking prevalence increases once more for those cohorts that came of age during the period that the Thatcher administration drastically cut income taxes. In all cohorts of men that followed, lifetime smoking prevalence trajectories are lower. Among women, the peak rate of smoking prevalence increases and falls more sporadically. Some of the rise and fall of the life-course smoking trajectories may be associated with changes in social norms that are associated with the feminist movement.

For each gender and cohort of Figure 1, Table 2 reports the corresponding sample sizes and 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 essentially fixed for every cohort; that is, they are directly comparable across genders and birth cohorts. Because we do not observe the full life-course of each cohort, 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.

Figure 2 plots the ratio of the summary measures of (Table 3) of British immigrant descendants to their native-born counterpart by cohort, gender, and host country. Our data again show that the most interesting differences appear in South Africa. The smoking habits of British immigrants in Australia and the US are close to those of natives; ratios vary within the 0.5 to 1.5 range for all genders and cohorts (apart from teenagers and young adults in the US who show ratios closer to 2 or more). In contrast, children of British immigrants in South Africa smoke more and longer than South African natives. Again, this pattern is particularly striking for women, especially young cohorts (notice the scale of the y-axis).

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 3 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. The correlation may arise because there is a strong biological force that gives rise to similar patterns of smoking across individuals. A candidate for such a force is a genetic predisposition to nicotine addiction (Ding et al 2006).

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). In doing so, 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 4. In all models the coefficient on the lagged dependent variable is high and for South African males it even exceeds 0.98. These results suggest a significant persistence in the smoking habit of British natives. The smoking prevalence rates in the host countries are also significant predictors. The estimated coefficient on the this variable is positive for Australia and the US and negative for South Africa. This result is consistent with the fact that smoking diffused more or less simultaneously in Australia, the UK and the US, whereas the majority of the South African population took up smoking later as incomes rose more slowly. Thus, while smoking prevalence in Australia, the UK and the US generally decreases across the generations in our sample, smoking prevalence in South Africa increases across generations.

The residuals 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.

To further demonstrate the variation in our measure of culture, we report the correlation matrix of the estimated residuals across genders and host countries in Table 5. 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. In contrast to the coefficients in Table 3, the new correlation coefficients are more heterogeneous across groups and in ways that appeal to intuition. For example, the highest correlation is between Australian and American females. This result implies that the smoking behavior of native females in Australia differs from smoking behavior of native UK females in much the same way that smoking behavior of native US females differs from the smoking behavior of native females in the UK. Given the overall similarity of smoking patterns in the two countries, this similarity is unsurprising. Also, the lowest correlation in smoking culture is between South African females and American males. This result implies that the deviation of smoking behavior of native females in South Africa and the UK is substantially different from the deviation in smoking behavior of native males in the US and the UK. This result also fits our expectations.

In general, our prior is that British migrants and natives are ethnically very different in South Africa, and less different in the US and Australia where more of the population originates from Europe. For Australia in particular, the 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 essentially constitute schemes for preferential migration from the UK. The correlation coefficients in Table 5 are consistent with this evidence; they suggest that the British smoking culture deviates most from the local culture in South Africa, less in the US, and least in Australia.

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

In Table 6, we report results from regressions of smoking participation that include our culture proxies.[10] As in Table 4, the lagged dependent variable absorbs a lot of the variation in smoking participation. In most cases, coefficient differences between immigrants and natives are statisti-

cally 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 immigrant men from Britain than for native men and immigrant men from other countries.

Our cultural proxy significantly predicts smoking participation of sons and daughters of British immigrants in Australia and the US, and of daughters of British immigrants in South Africa. The result implies that these groups have not fully assimilated their host country's culture - at least with regards to that part of UK culture that predicts smoking habits. Instead, their choices are influenced by smoking preferences that they inherited from a parent born in the UK.

Contrary to our expectations, the cultural proxy does not significantly predict smoking participation of sons of British immigrants in South Africa. This could be due to several reasons. First, health may play a bigger role in the decision of men to migrate to South Africa than to the other two countries (e.g. because of country-differences in living standards and health care quality), and this selection effect may transmit to second-generation immigrants. Second, the diversity in smoking culture among the native and the immigrant population may cause the cultural assimilation of immigrants to accelerate, whereas the relatively higher homogeneity between British immigrants and natives in Australia and the US may facilitate the preservation of British cultural habits. Third, the effect of ethnic diversity in South Africa may be inflated by its geographical remoteness from the UK. The more culturally different British immigrants are in the host country, and the more remote that country is from Britain, the more difficult it should be for those immigrants to sustain their cultural habits and their ties with Britain. This should be especially true for periods when means of communications were scarce and underdeveloped, which is the case for many years that our sample covers for South Africa.

Part of the above interpretation is also consistent with the fact that our proxy of British smoking culture predicts the smoking behavior of women but not the smoking behavior of men whose parents migrated to South Africa. Women are typically not the bread-earners of the family, and less often compete for physically demanding jobs in the host countries in comparison to men. Therefore, health selection to migration may not be as significant for women as for men. However,

a potentially more important reason is that womens' smoking habits are driven by changes in social norms and attitudes brought about by the feminist movement. An extensive womens' studies literature supports this hypothesis (Graham, 1994; Tinkler, 2006; Elliot, 2008). 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 to their daughters. Finally, patterns of migration between the UK and South Africa may differ by sex in ways that weaken our assumption that the average child was born when the average father was 25 years old. We explore this possibility in Section 4.3.

Our assertion that we capture a true culture effect finds further support when we fit the model on data from the people whose parents were born in Australia and the US. For those groups, in both countries, the culture proxy is uncorrelated with smoking participation. Because these groups are potentially of British and European origin, the result suggests that immigrants fully assimilate with respect to smoking culture from the third generation and higher. Finally, our proxy does not significantly predict smoking participation of people whose parents migrated to Australia and the US from a country other than the UK and it does not predict the smoking participation decisions of people whose parents were born in Australia. This result in line with the higher ethnic diversity between these groups and British immigrants.

As an additional exercise, and to further explore the significant culture effect for British immigrants, we test whether the culture 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.[11]

Table 7 reports results for different model specifications estimated on these samples. In the first three columns we report results for three different specifications, each of which is estimated on the sample of individuals whose parents were both born in the UK. The underlying model for column 1 measures smoking culture as the average culture 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. Thus, the coefficients in columns 1-3 respectively test whether smoking behavior of children of two UK-born immigrants varies with the mean smoking culture of UK men and women, the culture of UK men only, or the culture of UK women only. Column 4 and column 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. Thus, the coefficient estimate in column 4 and tests whether smoking behavior of children with a UK-born father systematically varies with the smoking culture of UK men. The coefficient estimate in column 5 tests whether smoking behavior of children with a UK-born mother systematically varies with the smoking culture of UK women.

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 male 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. Again, one can read this result to reflect the role of the feminist movement as a shaping and transmission mechanism of female smoking culture.

4.3 Testing robustness to an alternative definition of generation gap

To this point, 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 for two reasons. First, our data do not allow us to observe each individual's birth order (or whether she has siblings). Second, our data contain significant heterogeneity by gender and birth-cohort. 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.

The cohort gap changes over time and across genders for several reasons. Generally, older cohorts of parents gave birth at younger ages than did later cohorts. Older cohorts of parents also had more children. Finally, within a given cohort, men tend to have children at older ages than women. Because men and women may have migrated to each country at different rates over time, these sorts of differences might lead to a generational gap that varies across host countries and over time. If early cohorts of British immigrants to a given country were predominantly men, then the average British immigrant man in our samples may have more children and have had them at younger ages than the average British immigrant woman. Although we recognize the significant variation in the generation gap, we lack the data to precisely calculate it for each gender and birth cohort.

As a robustness check and to produce partial evidence on whether results vary with the assumed gap, we reestimated the models in Table 6 but assume a generational gap of 20 years. Note, that this assumption significantly increases our sample size, as we can now match 9 cohorts of children and parents, as opposed to 8 cohorts that we could match before. The new results, which we present in Table 8, are very robust to this change. Qualitatively, the estimated culture effects are exactly the same for all coutries and sub-groups. Our culture proxy significantly predicts the smoking behavior of Australian and the US people whose parents are British immigrants, and the smoking behavior of daughters of British immigrants in South Africa. In all other cases, the culture effects are statistically insignificant. Quantitatively, the results for Australia and the US are also robust; the differences in the culture coefficients reported in Tables 6 and 8 are statistically insignificant for these countries.

There are, however, some interesting quantitative changes in the South African results. When

we allow parent cohorts to be 20 instead of 25 years older than children cohorts, the culture coefficient for British immigrant women falls from .5 to .14, and the respective coefficient for British immigrant men increases from -.1 to .24 (with a p-value of 0.122). The implication is that the smoking habits of British immigrant women are closer to the smoking habits of UK cohorts that are 25 years older, while the habits of British immigrant men are closer to those of UK cohorts that are 20 years older. We interpret this result to reflect the fact that early waves of British immigrants in South Africa consisted of more men than women. Historical accounts report that UK women were willing to emigrate to the Commonwealth since the early 1900s. The UK government officially promoted female emigration to address an increasing gender imbalance in the home population resulting from male migration and WWI losses and also to facilitate endogamy in the population abroad. However, these efforts had very limited success in the 1920s and were eventually terminated by the 1930s depression (Blakeley 1988). To provide evidence specific to South Africa, we combined data from the 1996 and 2001 South African census (available at the IPUMS international online database). Using those data, we find that, in 2002, the fraction of men age 45-69 who were born in the UK was 11.5 percentage points higher than was the fraction of similarly aged women who were born in the UK. All together, this evidence suggests that, in South Africa, the culture effect for men is driven by older cohorts, who had children at younger ages (either with British immigrant women or with native women). In contrast, the culture effect for women is driven by the younger cohorts, who had children at relatively older ages.

5 Conclusion

A growing economic literature argues that culture predicts important economic and social outcomes. In this paper, we show that culture also predicts health outcomes like smoking participation. We find that culture matters for the smoking behavior of sons and daughters of British immigrants in Australia and the US, and daughters of British immigrants in South Africa. We find this result even though we employ a very conservative estimation technique and despite some data

limitations. 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. Our culture proxy does not significantly predict the smoking behavior of sons of British immigrants in South Africa, who thus appear to have culturally assimilated in their host countries. Although we acknowledge that the South African results are susceptible to noise, we argue that culture may matter less in this country because the native population is much more heterogenous and ethnically different from the population of Great Britain. The greater ethnic mix likely accelerates the rate of immigrant assimilation. We attribute differences in the estimated culture effects between sons and daughters of British immigrants in South Africa to the spread of feminist smoking-related attitudes and to differential degrees of health-based selective migration by gender. Finally, and more encouragingly, we find that our cultural proxy does not significantly predict the smoking behavior of natives in the host countries or of children of immigrants from countries other than Britain.

Our results have policy relevance. Worldwide, governments are devoting resources to try to influence individual decisions to smoke. These efforts reflect concerns about the annual (premature) deaths of more than 5 million people that are linked to tobacco consumption and projections that the number of deaths will grow in the near term. Already, governments spend a total of 965 million US dollars on anti-smoking policies per year, while annual tobacco tax revenues amount to more than 167 billion US dollars (WHO 2009). Our results suggest that the gains from these policies stretch beyond current generations and across national borders. Efforts to reduce smoking prevalence spill over from one generation to the next, even across geographic distances. Specifically, our results imply that, every 1 percentage point reduction in smoking prevalence in the parent generation in the UK decreases the probability of smoking participation among the children of British immigrants by between .09 to .50 percentage points.

Further, our results suggest that anti-smoking policies should be country-specific. To limit smoking, the World Health Organization (WHO) actively recommends that governments further monitor behavior, establish smoke-free environments, fund and promote smoking cessation pro-

grams, issue health warnings, ban tobacco advertising, and tax the sale of tobacco. These recommendations are collected under the acronym MPOWER. WHO staff suggest that "these six policies, if implemented in each country as a comprehensive package, would transform public health" (WHO 2008, p. 41). We show here that it may make sense to advocate an one-size-fits-all guideline and that anti-smoking policies should consider and reflect culture-specific patterns. After all, culture informs product promotion practices of tobacco companies, that target cigarette advertising to specific ethnic groups (Landrine et al. 2005, Primack et al. 2007). Our evidence informs policy makers about the potential efficiency (in terms of effects) that might be available if policies, such as taxation or anti-smoking campaigns, account for or use information about culture-specific smoking habits in their designs.

Our results are also scientifically relevant. Researchers believe that smoking proxies for or is highly related to rates of time preference and risk aversion (Fuchs 1982, Khwaja et al. 2007, Anderson and Mellor 2008 etc.). In fact, researchers often use smoking indicators as a predictors of discount rates (e.g. DellaVigna and Paserman, 2005) or as instruments to predict levels of schooling (e.g. Fersterer and Winter-Ebmer 2003; Dickson 2009) and other outcomes. We show here that observed patterns in smoking reflect culture-specific factors. While this does not speak directly to whether culture shapes an individual's rate of time preference or aversion to risk, it points to the possibility that it does, and/or that the use of smoking indicators as proxies for time preference/risk aversion need to be adjusted for the influence of cultural factors.

Finally, this paper also adds a methodological improvement to the extant literature on effects of culture. We propose a technique that exploits time-variation, made available by retrospective or longitudinal data, to produce a more precise test for culture effects. This technique can be applied and extended to utilize such types of data, as these are becoming increasingly available and as they increasingly measure a larger variety of behaviors.

Notes

[1]In X_{ict} we include regressors (age, cigarette taxes, and income) that are probably correlated with individual and contextual factors that we do not observe in our data (e.g. education, unemployment rates etc). A random effects specification would capture those associations.

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

[3]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 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).

[4]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.

[5]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.

[6]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.

[7]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). Because we lack detailed South African mortality data by age, race, sex, and cause, we cannot correct the South African prevalence rates. However, we suspect that any bias will be minimal because South African mortality rates are relatively high and mostly due to causes unrelated to smoking. For South African women in particular we expect that the adjustment would leave estimated rates unchanged because so few older South African women smoked.

[8]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.

[9]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.

[10]For our Australian and US samples, we assign sex-specific residuls 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.

[11]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.

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Tables and Figures

Table 1: Sample means

		Smoking prevalence		Age at interview		Real tax per pack	Real GDP per
		Males	Females	Males	Females	(local currency)	capita (2006=100)
Australia	All	25.2	21.5	38.0	37.6	2.34	73.7
	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		
South Africa	All	17.8	4.7	36.3	37.4	2.08	87.2
	British	28.8	18.4	40.1	39.7		
	Native/Other	15.8	2.5	35.6	37.0		
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	0.77	67.9
	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*: Smoking data: Australia - HILDA (2007), South Africa - NIDS(2009), UK - BHPS (1999, 2002), US - TUS-CPS (1995, 1996, 1998-2003, 2006, 2007); Tax data: Australia - Commonwealth of Australia Gazette (2005), South Africa - van Walbeek (2005), US - Tax Burden on Tobacco (2008) and state statutes; GDP per capita - Barro and Ursua (2008).

Table 2: Summary indicators of smoking trajectories of natives in the UK by sex and birth-cohort

Cohort	Males				Females			
(Age in 2002)	Peak prevalence	Age at peak	Years smoking	Obs.	Peak prevalence	Age at peak	Years smoking	Obs.
10-19	0.15	16	1	191	0.19	17	1	208
20-24	0.42	18	3	135	0.45	18	4	185
25-29	0.51	20	7	179	0.42	21	5	205
30-34	0.41	26	7	193	0.48	20	8	235
35-39	0.45	20	9	213	0.44	23	9	293
40-44	0.47	21	11	220	0.47	25	11	278
45-49	0.48	22	12	186	0.42	22	11	201
50-54	0.60	23	18	162	0.47	23	15	216
55-59	0.59	25	17	196	0.56	22	18	216
60-64	0.65	28	22	146	0.39	32	14	160
65-69	0.73	29	29	112	0.37	28	14	137
70-74	0.74	24	25	111	0.53	25	22	143

Table 3: Correlation coefficients among cohort-specific smoking prevalence in the UK and smoking participation in each host country by sex and immigrant status

	Australia		South Africa		USA	
	Males	Females	Males	Females	Males	Females
British	0.4627*	0.4358*	0.1876*	0.3982*	0.4103*	0.3468*
Native	0.4543*	0.4018*	0.4636*	0.5118*	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 4: Difference GMM estimation of smoking participation of British in the UK on cohort-specific smoking prevalence of natives in the host countries

	Australia		South Africa		USA	
	Males	Females	Males	Females	Males	Females
Lagged dependent variable	0.745	0.830	0.981	0.902	0.826	0.858
	[0.025]*	[0.020]*	[0.008]*	[0.024]*	[0.023]*	[0.015]*
Sm. prevalence in host country	0.208	0.136	-0.161	-0.890	0.234	0.169
	[0.039]*	[0.019]*	[0.060]*	[0.426]*	[0.033]*	[0.022]*
Person-year observations	93624	112207	87559	105985	88674	107322
Number of persons	2380	2874	2380	2874	2380	2874
Instruments	704	1415	1274	624	1036	1461
Hansen test	722.8	1465.4	1305.4	640.8	1060.6	1464.7
	(0.285)	(0.162)	(0.251)	(0.292)	(0.276)	(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 5: Correlation matrix of estimated residuals by host country and sex

		Males			Females		
		Australia	South Africa	USA	Australia	South Africa	USA
Males	Australia	1.000					
	South Africa	0.5833	1.000				
	USA	0.6241	0.3228	1.000			
Females	Australia	0.7253	0.5491	0.5112	1.000		
	South Africa	0.2552	0.8178	-0.1213	0.4986	1.000	
	USA	0.5596	0.4077	0.7715	0.8581	0.2513	1.000

Table 6: Difference GMM estimation of smoking participation by host country, sex, and immigrant status

	Australia			South Africa		USA		
	British	Native	Other	British	Native/Other	British	Native	Other
Males								
Culture	0.233	0.035	-0.009	-0.097	-0.041	0.144	0.082	0.020
	[0.104]*	[0.022]	[0.090]	[0.220]	[0.070]	[0.030]*	[0.042]	[0.056]
Lagged sm. participation	0.356	0.592	0.717	0.764	0.931	0.877	0.793	0.801
	[0.023]*	[0.098]*	[0.209]*	[0.087]*	[0.052]*	[0.031]*	[0.047]*	[0.064]*
Person-year observations	10237	70281	8281	12002	91516	56792	84070	54468
No. of persons/Instruments	367/33	2378/199	275/29	462/94	3964/257	1874/180	2906/209	2304/103
Hansen test	22.7	202.8	20.1	76.0	260.5	170.1	151.7	85.7
(for lagged instruments)	(0.703)	(0.300)	(0.634)	(0.816)	(0.328)	(0.570)	(0.997)	(0.787)
Difference-in-Hansen test	5.5	7.0	4.6	8.9	2.7	6.6	7.5	6.7
(for independent variables)	(0.356)	(0.222)	(0.461)	(0.112)	(0.751)	(0.251)	(0.186)	(0.240)
Females								
Culture	0.395	0.069	-0.046	0.495	0.015	0.093	-0.015	-0.014
	[0.113]*	[0.062]	[0.131]	[0.184]*	[0.013]	[0.037]*	[0.018]	[0.033]
Lagged sm. participation	0.515	0.857	0.618	0.812	0.843	0.966	0.883	0.950
	[0.177]*	[0.029]*	[0.125]*	[0.029]*	[0.032]*	[0.019]*	[0.034]*	[0.054]*
Person-year observations	10717	79807	8014	17217	145946	59017	93556	59956
No. of persons/Instruments	380/48	2710/291	272/29	645/34	5897/184	1926/251	3160/253	2437/105
Hansen test	46.2	287.4	20.0	36.9	174.9	234.1	229.3	91.9
(for lagged instruments)	(0.302)	(0.450)	(0.640)	(0.121)	(0.551)	(0.681)	(0.785)	(0.682)
Difference-in-Hansen test	4.5	3.6	9.2	8.6	5.4	0.5	5.3	2.1
(for independent variables)	(0.484)	(0.608)	(0.102)	(0.127)	(0.367)	(0.993)	(0.378)	(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, and for British in South Africa, culture is sex-specific. For British in Australia and the US 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 7: 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	0.335	0.552	0.202	0.097
	[0.213]*	[0.167]*	[0.204]*	[0.092]*	[0.049]*
Lagged sm. participation	0.869	0.892	0.900	0.969	0.934
	[0.416]*	[0.349]*	[0.307]*	[0.025]*	[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	8.2	8.8	6.9	72.0	129.1
(for lagged instruments)	(0.613)	(0.361)	(0.643)	(0.287)	(0.840)
Difference-in-Hansen test	5.1	4.8	3.0	5.1	5.1
(for independent variables)	(0.403)	(0.437)	(0.701)	(0.404)	(0.410)
Females					
Culture	0.376	0.168	0.442	0.0012	0.173
	[0.188]*	[0.163]	[0.174]*	[0.054]	[0.087]*
Lagged sm. participation	0.9013	0.931	0.9433	0.976	0.918
	[0.120]*	[0.098]*	[0.433]*	[0.072]*	[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	10.7	14.6	11.0	79.0	122.3
(for lagged instruments)	(0.222)	(0.264)	(0.444)	(0.169)	(0.327)
Difference-in-Hansen test	7.4	8.6	6.0	5.0	9.1
(for independent variables)	(0.190)	(0.125)	(0.309)	(0.417)	(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 6.

Table 8: Estimates of the culture effect when assuming a 20 year generation gap

	Australia			South Africa		USA		
	British	Native	Other	British	Nat./Other	British	Native	Other
Males	0.335*	0.086	0.126	0.236	0.053	0.129*	0.061	0.086
Females	0.323*	0.096	0.060	0.142*	0.022	0.135*	0.093	0.019

Notes: Estimation method is the difference GMM. All regressions pass the Hansen test that instruments are valid and the difference-in-Hansen test for the exogeneity of the independent variables. All other information as for Table 6.

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

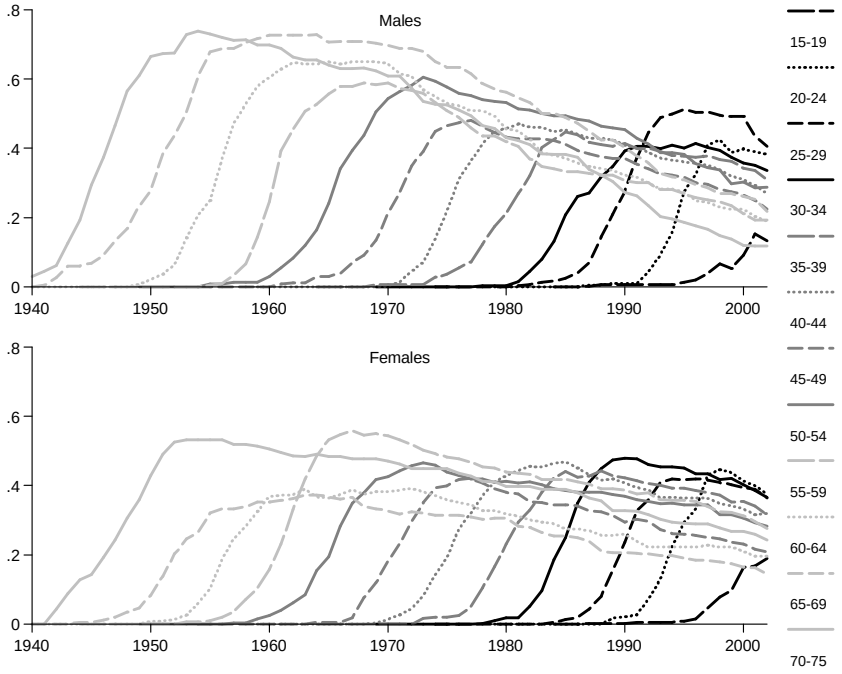

Figure 2: Summary indicators of smoking trajectories in relative terms between British immigrants and natives

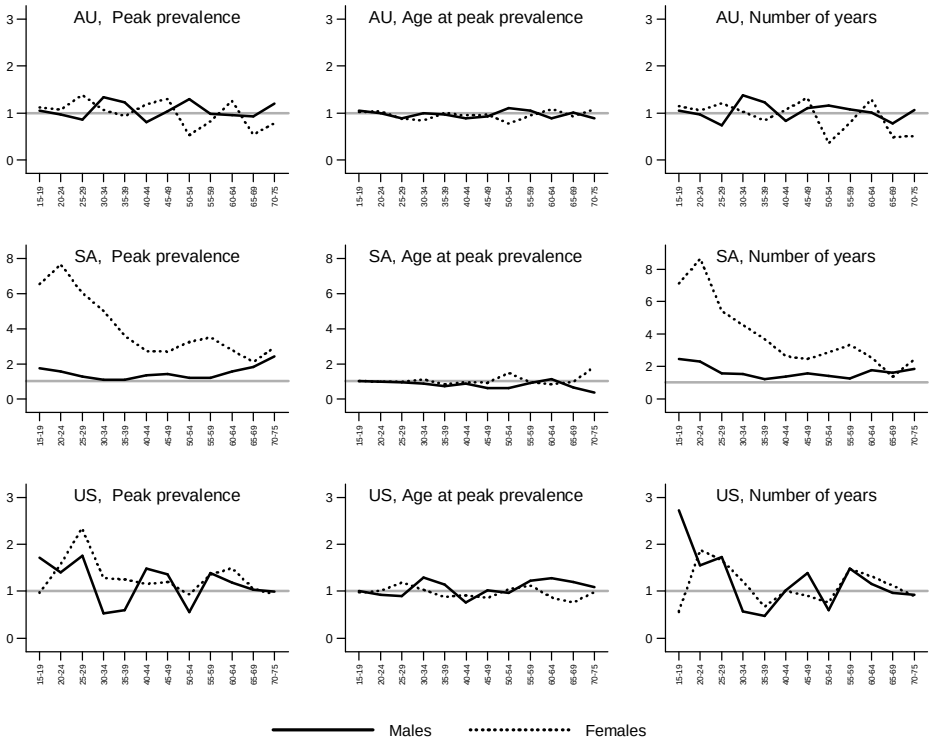

Figure 3: Residuals by host country, sex, and birth cohort