WORKING PAPER

working paper **number 146**
august, 2016

ISSN 1812-108x

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Alberto Palloni, University of Wisconsin
Laetícia De Souza, International Policy Centre for Inclusive Growth (IPC-IG)
Letícia Junqueira Marteleto, University of Texas

international policy centre for inclusive growth

International Policy Centre for Inclusive Growth (IPC-IG)
United Nations Development Programme

SBS, Quadra 1, Bloco J, Ed. BNDES, 13º andar
70076-900 Brasília, DF - Brazil
Telephone: +55 61 21055000

ipc@ipc-undp.org ▪ www.ipc-undp.org

The International Policy Centre for Inclusive Growth is jointly supported by the United Nations Development Programme and the Government of Brazil.

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Print ISSN: 1812-108X

FAMILIAL EFFECTS ON YOUTH SMOKING IN BRAZIL

Alberto Palloni,[1] Laetícia De Souza[2] and Letícia Junqueira Marteleto[3]

As with many other addictions and risk behaviours, smoking initiation is an early affair: most who adopt it at all do so early on during adolescence, rarely beyond their mid-20s. It is known that the cumulative health and mortality effects of smoking take a long time to unfold (Gajalakshmi et al. 2000; WHO 2008) and that they are a direct result of all three components of the behaviour: age of initiation, duration and intensity. Because age of initiation influences the other two components, the cumulative physiological damage induced by smoking depends on events that take place early in life and over a very small period of time. Protecting this sensitive period could not only remove the risk of ever smoking altogether but might also weaken the intensity and improve chances of desistance among those who do start smoking. For the majority of young people, most of the exposures that contribute to early smoking uptake are strongly associated with familial conditions and environments.

In this paper, we use a unique data set from Brazil and seek to identify effects of parental and sibling smoking on adolescent smoking behaviour. We estimate sibling models that account for common shared conditions within households and nearby environments, thus enabling us to estimate direct effects of parental and sibling behaviours. We identify the effects of birth order and sex among siblings and find that while the influence of older siblings on younger ones is very strong, the reverse is not true. Similarly, same-sex sibling effects are much more powerful than opposite-sex effects. We find that maternal smoking has a greater impact than paternal smoking, regardless of the sex of their offspring. Finally, we examine, albeit with fragile data, whether or not the effect of maternal smoking could be produced by in utero exposures but find no support for the idea. Whatever the mechanism, exposure to parental and sibling smoking exerts a considerable influence on youth smoking uptake and is a non-trivial conduit that reproduces smoking across successive generations.

This paper comprises four sections. Section 1 provides background information, reviews existing knowledge about determinants of early smoking onset, and introduces a set of conjectures. Section 2 summarises the state of the smoking epidemic in Brazil and describes the data used. Section 3 describes sibling models and statistical procedures, and summarises results. Section 4 concludes the study.

1. University of Wisconsin – Madison.

2. International Policy Centre for Inclusive Growth (IPC-IG).

3. University of Texas.

1 BACKGROUND INFORMATION, EXISTING KNOWLEDGE AND HYPOTHESES

1.1 BACKGROUND

It is entirely possible that continued progress towards longevity in low- and middle-income countries will encounter strong resistance as a result of lagged effects of the smoking epidemic. In particular, countries in the Latin American and Caribbean (LAC) region are more likely to face this challenge earlier in the 21st century, as they were first exposed to massive efforts by tobacco companies to broaden markets for cigarette consumption (Bianco et al. 2005). Recent assessments for a handful of countries in Latin America and the Caribbean, including Brazil, suggest that the relative number of years of life lost over age 50 due to smoking could be as high as 15–20 per cent of observed life expectancy at age 50, representing five to eight years of life lost (Palloni et al. 2012; 2013).

Any uncertainty is not about whether this 'health cliff' will be reached at all but, rather, about the timing of its onset, duration and magnitude. It is known that the aggregate health and mortality toll of smoking takes a long time to express itself, approximately 20 to 30 years after a cohort takes up smoking (WHO 2008). Much of the uncertainty about the future of health and mortality in a population exposed to smoking is rooted in the actual size of the smoking epidemic—the history of smoking in cohorts that will begin to enter vulnerable age groups. As a result, the overall mortality prospects for the next 20 years depend on events we can hardly alter, namely the smoking history of cohorts born between 1950 and 1970. Once these cohorts are extinct, ripple effects of smoking may continue to be felt. Their magnitude and duration will depend on patterns of smoking uptake, smoking intensity and smoking desistance among the younger cohorts born after 1980–1990. The exact configuration of these younger cohorts regarding smoking behaviour could depend in non-trivial ways on the influence of the parental generation.

1.2 EARLY START AND THE ROLE OF FAMILIAL INFLUENCES

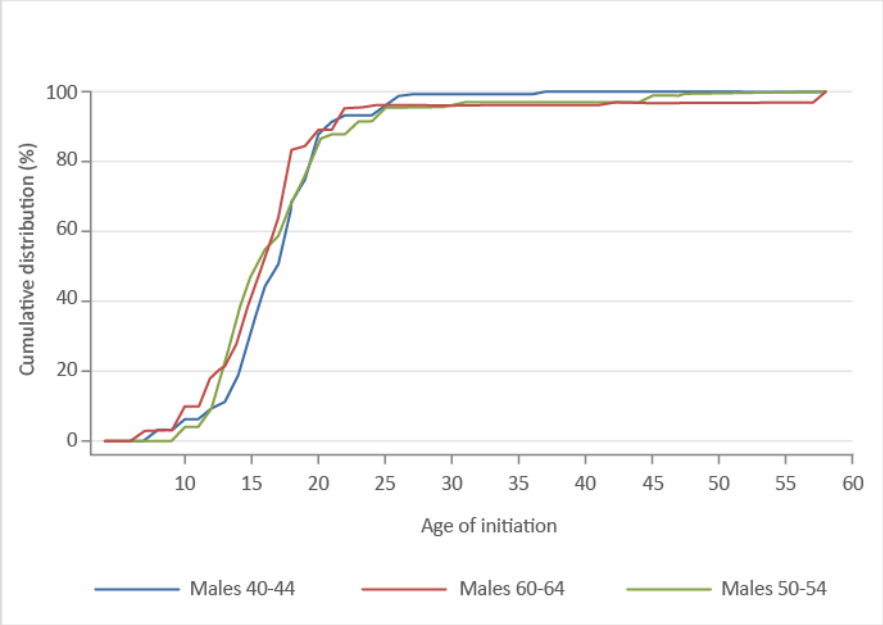
We know from previous research that adult smoking behaviour is decided early in life, that a smoking debut during early adolescence increases smoking intensity, plays a role in decision-making processes about quitting, modulates successful desistance and may even be implicated more directly in the nature of damage induced by smoking (WHO 2008; Galajakshmi et al. 2000). Figures 1a and 1b display the cumulative distribution of age of smoking initiation among current male and female smokers aged 40–44, 50–54 and 60–64 in Brazil in 2008.

The most striking feature of these figures is the steepness with which these curves rise. Among males, virtually all those who were smoking at the time of the survey began before age 20, regardless of birth cohort. The shift toward later ages of initiation in the more recent birth cohorts is almost imperceptible. Among females, there is more diversity across cohorts but with an ominous pattern: younger female cohorts clearly start smoking at earlier ages. Even among older female cohorts, fully 80 per cent of those smoking at the time of the survey did so before age 25. These patterns are not exclusive to Brazil but fairly common elsewhere, particularly in countries where the smoking epidemic is in its early stages (Lopez et al. 1994; WHO 2008).

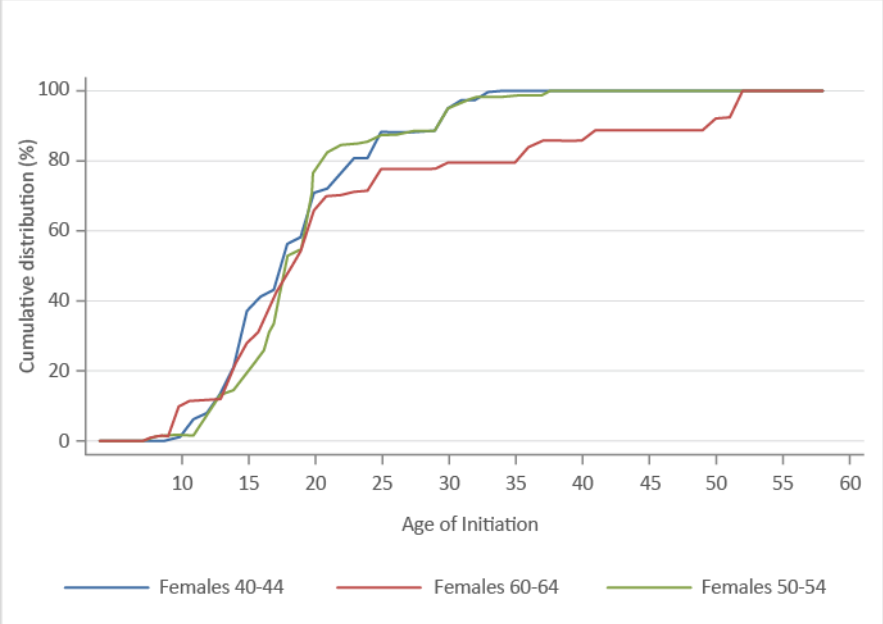
FIGURE 1

Age of smoking initiation by sex and cohort

1A: Male cumulative distribution of smoking age of initiation by birth cohort - Brazil, 2008



1B: Female cumulative distribution of smoking age of initiation by birth cohort - Brazil, 2008



Source: PNAD data, IBGE (2008).

The period of high vulnerability to tobacco addiction is narrow—certainly less than 10 years. It overlaps with a life stage (Swan et al. 1990) when most adolescents live in the parental home and are maximally exposed to material, social and emotional conditions shaped by the influences of parents, siblings and extended kin. If these conditions increase the propensity of early

uptake, their effects will linger on during the life course of young members of the household: those who start at all, but particularly those who start earlier, are less likely to successfully quit, and, if they do so at all, do it much later—thus enhancing exposure to cigarette-induced physiological damage (Lariscy 2013). Admittedly, the onset of smoking is influenced by many conditions experienced in early childhood and adolescence, including socio-economic conditions, household characteristics, family stability, poverty and insecurity, individuals' experiences with externalising and internalising behaviours, social acceptance and maladjustment. We focus here on a small subset of these, which have a long reach and disproportionate influence—conditions associated with parental and sibling smoking and the environments they reflect, such as school peers, neighbours and kin.

The possibility that parental smoking may by itself increase the propensity of uptake among young adolescents is most damaging, as it constitutes a unique channel through which the behaviours of one generation, with its singular effects on its health and mortality, influence the adult health and mortality of the next one. The precise mechanism that drives this relation remains obscure, since in most cases the empirical literature fails to separate the direct effects of parents and siblings from the effects associated with conditions *shared within a household by parents and offspring*. It is necessary to identify more precisely the nature of these mechanisms, as the public health implications of each are starkly different. Indeed, if direct effects swamp the influence of shared environments, public health campaigns to discourage smoking could have additional benefits for younger cohorts. If the campaigns efficiently increase desistance among adults who already smoke, they will simultaneously erode the conditions that encourage early uptake among their offspring. At the very least, this will reduce the interval of time during which the health and mortality consequences of smoking are felt in a population by, approximately, the length of a generation.

1.3 MECHANISMS OF FAMILIAL INFLUENCE: PARENTS AND SIBLINGS

A great deal of research on smoking behaviour comes from studies in high-income countries where the smoking epidemic is in its late stages (Pampel 2002; Pampel et al. 2010; Pampel and Rogers 2004; Rogers et al. 2005). With appropriate caveats, however, this knowledge is useful as aguide for the study of determinants in countries that are just now facing the early stages of the epidemic.

The determinants of early uptake of smoking behaviour are similar to, and overlap with, the determinants of adoption of other risk behaviours. Adolescence is, in general, a period in life marked by the adoption of risk behaviour, and this includes smoking initiation. Studies have identified several factors associated with an increased probability of smoking initiation during adolescence.

First, the highest smoking rates and lowest desistance are found among those with low levels of socio-economic status (Pampel 2002; Pampel et al. 2004; Pampel et al. 2010; Rogers et al. 1995). What exactly it is about low socio-economic status that increases the propensity to smoke is not at all clear, although it is unlikely that (lack of) access to information has anything to do with it. A more likely possibility has to do with group-specific outlooks and attitudes, time discounting, and propensities to defer rewards and gratification (Ert et al. 2013). Second, there is widespread agreement about the role of peers[4]. (Argys and Rees 2008; Lantz et al. 2000), parents (Flay et al. 1994) and siblings (Otten et al. 2007; Slomkowski et al. 2005). Yet, extant research fails to clearly separate which effects are associated with conditions shared by peer groups, siblings, parents and the adolescents themselves, rather than interpersonal interaction or, in the case of peers, with individual propensity to join peer groups.

Parental social influence

Children are often first exposed to smoking within the household. Commonly in a nuclear family, parents are the prevailing socialisation agents. Through imitation, role modelling, incentives and sanctions, the smoking habits of parents can influence adolescent smoking initiation (Otten et al. 2007). In fact, the role model framework suggests that younger siblings consider parents of either sex and older siblings alike as role models to be followed and imitated (Buhrmester et al. 1992; Gilman et al. 2009; Hill et al. 2005; Otten et al. 2007). The strength of parental social influence is highly variable and modifiable in a number of ways.

First, the risk of smoking initiation in adolescence is much higher when parents are active smokers than when parents are former smokers (Gilman et al. 2009). Children of parents who have ceased smoking have chances of smoking no higher than those of peers whose parents have never smoked (ibid.). This could be related to both imitation and new norm enforcement. Second, children of a given age who are exposed for a longer time to at least one parent who smokes have a higher risk of uptake than children of the same age whose parents smoked for a shorter duration. Third, the risk of smoking initiation by an adolescent is higher when both parents smoke than when only one of them does (ibid.). Fourth, although researchers suspect that paternal and maternal smoking should produce different influences (Kandel and Wu 1995), the empirical evidence is unclear at best. Some studies document that maternal and paternal smoking are equally consequential (Hu et al. 2006), while others show that maternal smoking is much more relevant (Swan et al. 1990). Others still suggest that paternal smoking overwhelms maternal smoking (Gilman et al. 2009). Furthermore, the association between parental smoking and adolescent smoking initiation may vary according to the combination of the sexes of parent and child (Rossow 1994). Some studies suggest that the effects of paternal smoking are stronger for boys than for girls, whereas those of maternal smoking are equally important for both sexes (Gilman et al. 2009). In this paper we show that maternal smoking exerts a more powerful effect among children of both sexes.

Parental smoking and 'second-hand addiction'

Parental smoking literally contributes to a smoky environment. Not only is second-hand smoking damaging to health, but it can also generate conditions of indirect addiction to nicotine (Brody et al. 2011). This mechanism is particularly relevant for children in early stages of their physiological growth and development, especially among children with a genetic propensity to become addicted. If the creation of an environment with second-hand smoke were the only direct channel through which parental smoking influences adolescent uptake, then estimated effects should not vary by parental sex: keeping intensity constant, paternal and maternal smoking should be equally damaging.

In utero exposure

Finally, there is a third mechanism through which parental (in this case maternal) smoking can influence early uptake by a child or adolescent: *in utero* exposure to maternal smoking during pregnancy. Since nicotine is absorbed and circulated in the blood crossing the placental barrier, there is the distinct possibility that nicotine addiction is enhanced when a child is in the womb (Niaura et al. 2001; Roberts et al. 2004; Rydell et al. 2012). Were this mechanism important at all, maternal smoking alone should influence smoking uptake among adolescent children, irrespective of paternal smoking behaviour and the offspring's sex.

Sibling influence

The social influence of siblings can be as powerful as that of parents (Shanahan et al. 2001). In particular, older (and same-sex) siblings tend to be role models for the younger ones, expose them to similar peer groups and exert socialisation effects comparable to or even stronger than those of parents (Swan et al. 1990) . Consistent with these ideas, empirical research confirms a high level of concordance between the smoking behaviours of children (Slomkowski et al. 2005). Some research also suggests that exposure to smoking through older siblings is a more effective trigger for early initiation than exposure to smoking by younger ones (Hiemstra et al. 2012; Ouyang 2004). By the same token, birth order interacts with the number of siblings to determine the underlying effects of siblings on the onset of smoking. Firstborns, for example, are less likely to smoke cigarettes than their younger siblings (Argys et al. 2006), although the exact reasons for this phenomenon are unclear. Some research suggests that the influence exerted by sibling smoking is very much a function of the quality of the interpersonal relationships of siblings (Slomkowski et al. 2005). An intriguing, but as yet unexplored, possibility is that sibling influence might be contingent on parental smoking: the direct (social) influence of the smoking behaviour of older siblings may be larger when parents do not smoke at all compared to when they do.

Just as in the case of parents, observable effects of sibling smoking on adolescent uptake may also be the result of addictive behaviours generated by exposure to second-hand smoke. However, if this were the only mechanism at play, none of the nuances in the relations between sibling smoking behaviour by sex, age differentials or birth order effects would be observed.

Genetic inheritance of smoking propensity

More recently, there has been a body of research using genetically informed samples to attempt to disentangle the extent to which genetic and/or environmental factors contribute to similarities between sibling (Slomkowski et al. 2005) and parent–child smoking behaviours. Although some of this research suggests a genetic component regulating nicotine craving, nicotine metabolism or both, it is unlikely that genetics alone can explain a substantial fraction of parent–offspring or sibling concordance. Recent empirical evidence (Boardman et al. 2010; 2011; Daw et al. 2013) convincingly shows that genetic influences are at play, but their expression (directly or through interactions between genes and environment) can vary over time as a function of how social constraints or social acceptance of smoking grows or diminishes.

1.4 HYPOTHESES

There are a handful of conjectures we can infer from existing knowledge about familial influences on adolescent smoking behaviours. Not all of them can be falsified equally well with the data available to us. Thus, those regarding the relative importance of shared conditions *vis-à-vis* direct behavioural influences are more amenable to testing than those invoking *in utero* influences, which we can only superficially examine. Similarly, because we do not have a combined twin/sibling sample, we cannot test hypotheses implicating either direct effects of genes or interactions between genes and environment. The following are succinct statements describing the main hypotheses we attempt to verify:

- Hypothesis 1: Familial effects operating via direct influence of parent/sibling smoking are strong even after accounting for the effects of measured shared environments.
- Hypothesis 2: Parental smoking has a direct influence on adolescent smoking uptake and is distinct and separable from the effect of sibling smoking behaviour.

- Hypothesis 3: Parental and offspring sex modulates relations only if parental smoking influences offspring smoking via mechanisms other than the contribution to second-hand smoking. We expect paternal smoking to have a stronger influence on male offspring than on female offspring, and maternal smoking to have a stronger effect on female offspring smoking than on male offspring smoking.
- Hypothesis 4: If only or mostly due to role-model dynamics, the influence of the smoking behaviour of older siblings should be stronger than the influence of younger sibling smoking and amplified in the absence of parental smoking. By the same token, we would expect same-gender effects to be stronger than opposite-gender effects.
- Hypothesis 5: If in utero exposures are relevant, we should verify that the effects of maternal smoking are stronger that paternal smoking regardless of the offspring's gender and current maternal and paternal smoking status.

2 A STRATEGIC EXAMPLE: THE CASE OF BRAZIL

While some countries of the Latin America and Caribbean (LAC) region have led in the adoption of tobacco consumption (Argentina, Chile, Cuba and Uruguay) others have remained behind and may never become fully exposed to it (Andean countries and Central America). The case of Brazil is unique, as it is currently going through the early stages of the smoking epidemic; it could halt its fully fledged development with efficient public health campaigns. If these should fail, Brazil will follow the same course followed by other countries in the region. As we discuss further, a key feature that these policies should contemplate is the extraordinary impact exerted by family environments and behaviours.

2.1 SMOKING IN BRAZIL

According to a useful typology (Lopez et al. 1994), Brazilian males are currently experiencing the late phase of the third stage in the smoking epidemic, whereas females lag behind in the second stage (Palloni et al. 2012). Overall smoking prevalence among those aged 15 and over is around 24 per cent among males and 15 per cent among females. The damage caused by smoking in Brazil's adult population amounts to losses in life expectancy at age 50 in 2008 of 5 per cent among males and 1 per cent among females; those losses are expected to mount rapidly and reach about 8 per cent among males and 5 per cent among females by 2020 (Palloni et al. 2013). In terms of mortality impact, Brazil is placed in an intermediate situation, between the forerunners of the smoking epidemic in the region (Argentina, Chile, Cuba and Uruguay) and the laggards (Andean and Central American countries). A distinct trait of Brazil is an important shift toward lower smoking rates among the youngest age groups, well before such a decline is expected according to standard typology.

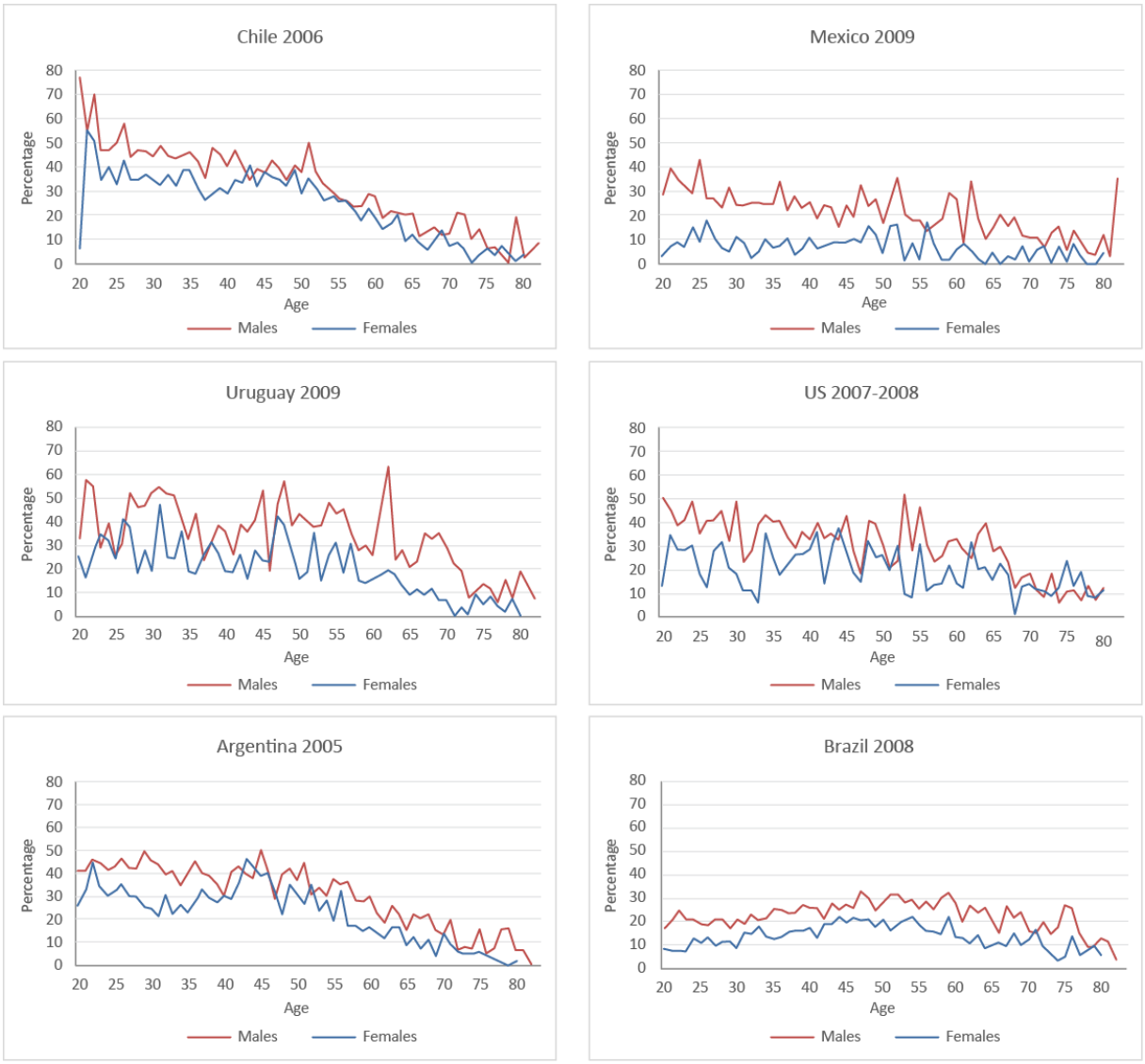
Figure 2 compares Brazil with the USA and selected other countries of the LAC region. It is likely that Brazil's rather unique age pattern of smoking prevalence is the result of recently adopted anti-smoking campaigns (Cavalcante 2005; Monteiro et al. 2007).

Despite public health campaigns, studies in Brazilian cities continue to show very young ages of smoking initiation, ranging from 10 to 17 years old (Almeida et al. 2008; CEBRID, UFSP and EPM 1997; Menezes et al. 2006; Zanini et al. 2006). The more than 20 million adolescents between 15 and 19 years old and more than 30 million young people aged between

20 and 29 are a prime target of an intense publicity campaign by the tobacco industry (Barreto et al. 2009). However, very little is known about the factors that precipitate early smoking uptake, as most of the studies tend to focus on tobacco consumption and their health consequences during adulthood.

FIGURE 2

Smoking prevalence by age and sex, selected countries



Source: PNAD data, IBGE (2008).

2.2 THE DATA

Nature of the data set

The data set we use in this paper has partially filled what was until recently a vast landscape of non-existent, inaccurate or incomplete information. We use the 2008 Brazilian Household Sample Survey (*Pesquisa Nacional por Amostra de Domicílio*—PNAD), a nationally representative survey with data collected by the Brazilian Institute for Geography and Statistics (*Instituto Brasileiro de*

Geografia e Estatística—IBGE). Annual data collection has been taking place since 1971, except in 1994 and during the census years: 1980, 1991, 2000 and 2010. The PNAD is analogous to the US Current Population Survey and consists of a probability-based, stratified, multi-stage household sample. The sampling design follows a three-step probabilistic procedure based on municipalities, census tracts within municipalities, and households within sectors.

The PNAD contains information on every household and family member, and is a rich source of social, economic and demographic data. In all years, PNAD includes information on several characteristics (education, work, income and household infrastructure). Other information on migration, fertility, marital status, health, nutrition and other topics is collected episodically.

The 2008 PNAD included supplementary health items and a thorough questionnaire on smoking. In this paper we use a number of questions on smoking that were part of the Supplemental Health Survey of the 2008 PNAD. The smoking module was administered to all household members who were aged 15 or older. Questions were asked about current and previous smoking status, frequency of smoking, access to anti-smoking messages etc.[5]

The 2008 PNAD sample size is about 391,868 people. We use a sub-sample of 8,604 individuals aged 15 to 25 years old, restrict our sample to offspring of the head of household and exclude other kin. In the next section we describe the sub-samples used in our empirical strategy. As mentioned earlier, the PNAD sampling design follows a three-phase stratified procedure. Therefore, we take into account its complex sample design while estimating any statistics either concerning the descriptive or the multivariate analysis presented in this paper.

Variables and measures

Table 1 displays summary statistics for our analytical sample of adolescents aged 15–25 who live in households with at least one sibling (see below). In households with two siblings satisfying the age constraint, we selected both individuals as members of the sample with probability one. In households with more than two siblings satisfying the age constraint, we randomly selected one possible sibling pair.[6] For convenience, we chose one sibling as the 'target' and the other as the target's sibling. The statistics in Table 1 pertain to a target individual and his/her chosen sibling.

The table shows that 7 per cent of the target adolescents are smokers, 43 per cent are females, and almost 70 per cent are delayed in school (i.e. they are in a school grade that is at least one year less than expected given their age). The table also shows parents' and siblings' characteristics. As expected, mothers are younger than fathers (45 and 49 years old, respectively) and with a similar level of education, as the mean years of completed schooling is 6.3 for fathers and 6.8 for mothers. Less than 17 per cent of mothers are smokers, while 27 per cent of fathers smoke. In about 2 per cent of the pairs, both siblings smoke, and in about 88 per cent of the pairs neither sibling smokes. The young people (aged 15–25 years) in our sample have an average of slightly less than two siblings, and they mostly live in urban areas (80.7 per cent), and in the Southeast (42.3 per cent) and the Northeast regions (30.1 per cent).

TABLE 1

Descriptive statistics for adolescents (one pair per household) aged 15–25 years old, Brazil, 2008

Variables	Mean	Percentage
Target adolescent		
Smokers	6.97	(0.3241)
Female	42.93	(0.5951)
Age	19.16	(0.0373)
Delayed at school (omitted category: not delayed)		
Delayed one year	31.86	(0.6007)
Delayed two years	37.46	(0.6685)
Skin colour/race (omitted category: White/Asian)		
Black	6.03	(0.3010)
Pardo (mixed race)	45.15	(0.6451)
Parents		
Mother's age	45.23	(0.0775)
Mother's education	6.80	(0.0662)
Mother works for pay	59.94	(0.6479)
Mother smokes	16.86	(0.4678)
Mother smoked while pregnant	4.77	(0.2751)
Father's age	48.98	(0.0949)
Father's education	6.32	(0.0679)
Father works for pay	87.54	(0.3969)
Father smokes	27.04	(0.5779)
Smoking status (omitted category: neither smoke)		
Both parents smoke	8.54	(0.3467)
Only mother smokes	8.37	(0.3382)
Only father smokes	18.52	(0.4764)
Target's siblings		
Smokers	7.32	(0.3284)
Both smoke (concordant smoker pairs)	1.79	(0.1614)
Neither smokes (concordant non-smoker)	87.55	(0.4191)
Only one smokes (discordant)	10.66	(0.3808)
Female	43.10	(0.6141)
Same sex	51.87	(0.6011)
Age	19.15	(0.0347)
Delayed at school (omitted category: not delayed)		
Delayed one year	31.88	(0.5930)
Delayed two years	37.66	(0.6422)
Skin colour/race (omitted category: White/Asian)		
Black	5.66	(0.2711)
Pardo (mixed race)	46.72	(0.6501)
Family and household characteristics		
Number of siblings	1.99	(0.0167)
Area of residence		
Urban	80.66	(0.7247)
Region of residence (omitted category: Southeast)		
North	8.76	(0.2846)
Northeast	30.07	(0.5325)
South	11.77	(0.3452)
Centre-West	7.05	(0.2317)
Number of observations	8,604	

Source: PNAD data, IBGE (2008).

Note: We only show information on the target youngster to confirm that both samples are composed of similar individuals on average. The remaining characteristics are also very similar among the samples, as the probit model has individuals pertaining to the same family, therefore sharing similar conditions as the ones in the biprobit sample. Standard deviation in parentheses.

3 METHODS AND RESULTS

3.1 METHODS

We proceed in two stages. We first estimate simple probit models to ascertain the probability that *any* adolescent in a household is a current smoker. Since we are interested in comparing results from the conventional probit model (with no control for unmeasured shared conditions) to those from a bivariate probit model (controlling for unmeasured shared conditions), we compute estimates using the same sample of sib pairs we use for biprobit models.[7] We estimate effects of parental and sibling smoking while controlling for a number of individual and observed household shared conditions known to be associated with early smoking. These include adolescent's gender, parental and adolescent educational attainment, region of residence and other controls

These simple probit models are useful but could be potentially misleading; if unmeasured characteristics shared by household members affect whether an adolescent is a current smoker, the models may overstate the effects of both parental and sibling smoking. To determine the existence and magnitude of the bias, we proceed to estimate biprobit models for sibling pairs. We estimate the probability that one member of the sibling pair is a current smoker using biprobit models that include traits of household, parents and siblings.

A biprobit model includes parameters for two equations, one for each sibling's smoking status, and an extra parameter for the correlation between the errors of those equations. The equations are estimated simultaneously and yield estimates of parameters associated with covariate effects (in probit metric), as well as an estimate of the residual correlation between error terms (conditional on observed covariates) (Greene 2012). The key feature of biprobit models is that, as other methods for multiple dependent variables of the seemingly related genre, they allow a non-zero correlation between the residuals associated with each equation in the model.

A non-zero value for the residual correlation between pairs of siblings can originate from three sources. First, the model omits important conditions shared by both siblings that affect siblings' propensity to smoke. Second, there could be errors in reporting of smoking that are correlated across siblings. Third, and most important, the residual correlation could reflect the existence of a direct influence of one sibling's smoking on the other sibling's smoking status *conditional on measured shared and individual characteristics.*

The exact specification of the model is shown in Equations (1a) and (1b):

$$Y_{1j}=X_j\beta+\varepsilon_{1j} \quad (1a)$$

$$Y_{2j}=Z_j\varphi+\varepsilon_{2j} \quad (1b)$$

where Ys are latent variables corresponding to sibling 1 and 2 of household j, $E(\varepsilon_{1j})==E(\varepsilon_{2j})=0$, $Var(\varepsilon_{1j})=Var(\varepsilon_{2j})=1$ and $Cov(\varepsilon_{1j}\varepsilon_{2j})=\rho$. The observed variables are $y_{1j}=1$ or $y_{2j}=1$ if $Y_{1j}>0$ and $Y_{2j}>0$ respectively. Our task is to assess the magnitude and sign of the coefficients, to assess the value, sign and changes in the estimates of ρ as we alter the model specification, and, finally, to compare inferences drawn from the biprobit model with those drawn from simple univariate probit models.

The biprobit model's estimates are uncontaminated by unmeasured shared conditions (these effects are all absorbed in the parameter ρ). It follows that a comparison between simple-probit and biprobits estimates can tell us a great deal about the relative importance of shared unmeasured conditions for the observed gross association between adolescent and parental and sibling smoking.

3.2 RESULTS

Simple probit models

Table 2 displays estimates from four variants of simple probit models calculating the probability of current smoking among individuals (both the target and their sibling) aged 15–25 in the sample of all households with at least two siblings.[8] Model 1 includes baseline covariates reflecting individual traits (gender, race, age and the indicator for education) and household characteristics. They include parental education and occupation, household income, region of residence, rural/urban residence and number of siblings in the family.[9] Probabilities of smoking are smaller for females than for males, increase sharply with age and are augmented by adolescent education deficiencies/delays.

Model 2 adds variables to capture the smoking status of parents, and Model 3 includes an indicator for sibling's smoking status. Models 2 and 3 address Hypothesis 1; estimates show that both parental smoking and sibling smoking exert powerful effects above and beyond those implied by shared *measured* conditions. As expected from Hypothesis 2, Model 2 is consistent with the conjecture about influences of parental smoking. According to Hypothesis 3, the effects of mothers' and fathers' smoking should differ. Furthermore, the '*in utero* exposure' conjecture predicts that maternal smoking should be more influential than paternal smoking. This is indeed the case, but the differences between the corresponding effects are not statistically significant. More consequential is that two parents smoking make more of a difference than one parent smoking.

However, much more powerful than parental effects are those associated with sibling smoking. Their effects are significantly higher (β=1.99 for a sibling currently smoking, against β=0.544 in case of both parents smoking), showing that sibling effects are not just distinct from but much more consequential than parental effects. Model 4 probes more deeply into the nature of sibling effects and addresses Hypothesis 4. It includes a categorical variable to measure sibling smoking status, jointly allowing for differences in both siblings' gender and birth order. The residual category is composed of observations with older and same-gender siblings who do not smoke.[10]

According to Model 4, neither birth order nor sibling gender are influential, only the smoking status of the sibling. Somehow different from what should be expected by role theory, among same-gender siblings the effects of an older sibling who smokes (β=0.68) is virtually identical to the effect of a younger sibling who smokes (β=0.67). Thus, while the predicted probability of smoking among those whose older sibling smokes (0.41) is about 1.7 times larger than among those whose younger sibling smokes (0.24), by contrast, the predicted probability of smoking among those who have a sibling who smokes (regardless of birth order) is more than four times larger than among those who do not. A similar pattern

prevails among siblings of opposite sex (β=0.66 vs β=0.80). Furthermore, the difference of effects associated with siblings of similar birth order (but opposite sex) who smoke (β=0.67 vs 0.80 for younger siblings and 0.68 vs 0.66 for older siblings) is quite small. These results do not support the conjecture from role-model theory (Hypothesis 4).

TABLE 2

Alternative univariate probit models

Variables	Model 1	Model 2	Model 3	Model 4
Female	-0.466***	-0.486***	-0.454***	-0.515***
	(0.04)	(0.05)	(0.06)	(0.05)
Age (logarithm)	2.188***	2.246***	2.461***	2.176***
	(0.13)	(0.13)	(0.19)	(0.14)
Delayed one year in school	0.195***	0.189***	0.187**	0.203***
	(0.06)	(0.06)	(0.07)	(0.06)
Delayed two years in school	0.872***	0.845***	0.700***	0.837***
	(0.07)	(0.07)	(0.09)	(0.07)
Mother's education	-0.00565	-0.00288	0.00529	-0.00172
	(0.01)	(0.01)	(0.01)	(0.01)
Father's education	0.0085	0.0110*	0.0119	0.0115*
	(0.01)	(0.01)	(0.01)	(0.01)
Both parents smoke		0.627***	0.544***	0.584***
		(0.06)	(0.08)	(0.06)
Only mother smokes		0.517***	0.475***	0.479***
		(0.07)	(0.10)	(0.06)
Only father smokes		0.328***	0.325***	0.303***
		(0.05)	(0.07)	(0.05)
Sibling smokes			1.987***	
			(0.05)	
Sibling sex				
Same sex				
Younger and does not smoke				-0.261***
				(0.08)
Younger and smokes				0.670***
				(0.12)
Older and smokes				0.684***
				(0.15)
Opposite sex				
Younger and does not smoke				-0.125*
				(0.08)
Younger and smokes				0.798***
				(0.16)
Older and smokes				0.658***
				(0.21)
Older and does not smoke				-0.0198
				(0.06)
Constant	-8.508***	-8.980***	-9.933***	-8.779***
	(0.45)	(0.45)	(0.64)	(0.45)
Number of observations	**16,264**	**16,171**	**16,124**	**16,171**

Source: PNAD data, IBGE (2008).

Notes: Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Finally, according to Hypothesis 3, sibling effects should be stronger *ceteris paribus* in the absence of parental smoking. To test this conjecture, we estimated a model with interactions between the variables for sibling smoking and those for parental smoking. However, this model variant does not fit the data better. We conclude that the influence of sibling smoking is equally strong, irrespective of parental smoking.

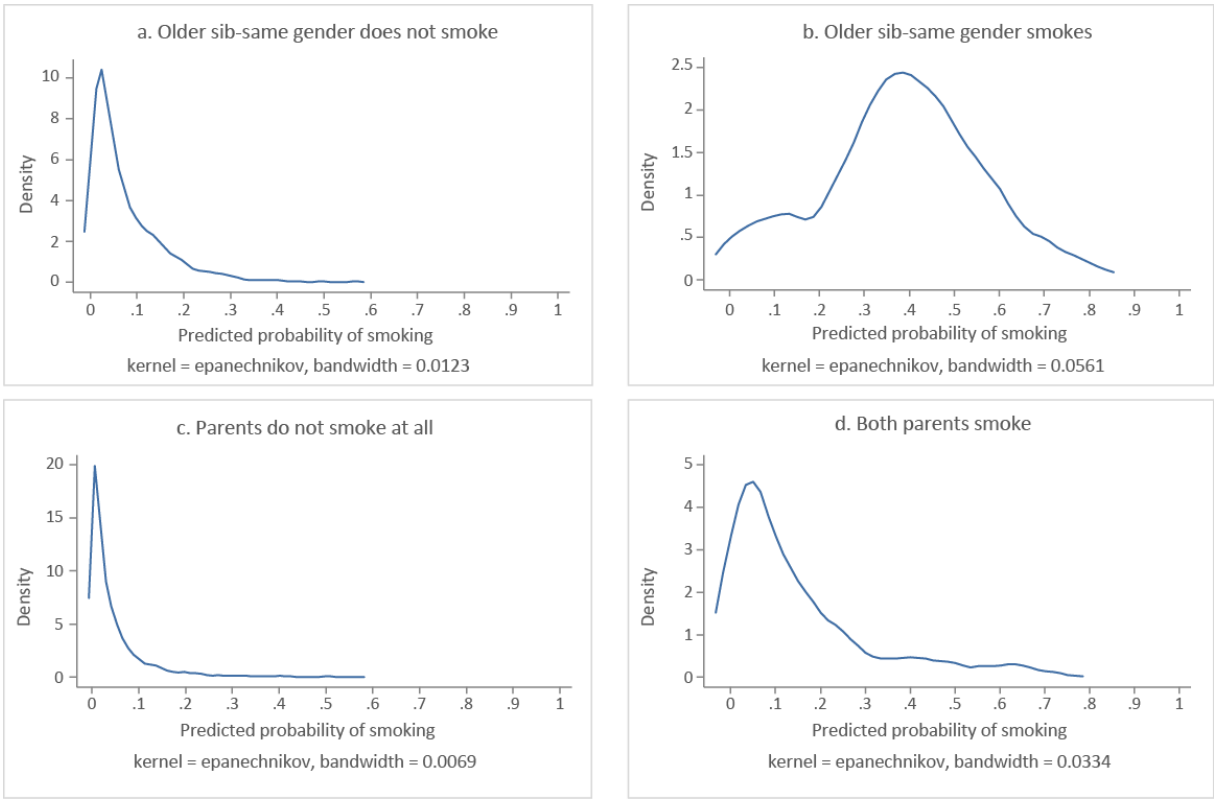
The above discussion has relied exclusively on the magnitude, sign and z-values of estimated coefficients. A much more compelling case can be made if we examine predicted smoking status. The first two panels of Figure 3 illustrate the effects of sibling smoking: the first panel displays the distribution of predicted probabilities of early smoking among individuals with average characteristics and whose sibling is (a) the same gender, older and does not smoke; and (b) the same gender, older and smokes. The last two panels of Figure 3 display the predicted probabilities of early smoking among individuals with average characteristics and (c) whose parents do not smoke at all and (d) both parents smoke. The most telling contrast is between the magnitude of sibling and parental effect: the former is equivalent to multiplying the probabilities of smoking by a factor of 1.4 to 8.7, whereas the latter increases them by a factor varying from two to at most three.

One caveat: it is risky at this stage to use the empirical evidence from Table 2, no matter how compelling it may be, to either support the conjecture that siblings' social influence is much stronger than that of parents or to promote the idea that parental effects behave as expected by the theory of second-hand smoking (the child's gender is irrelevant). At this point, we cannot dismiss the possibility that, instead of capturing interpretable direct effects of sibling or parental smoking, our estimates reflect shared unmeasured traits that equally influence the smoking behaviours of all members of the household. We address this issue more directly in the next section.

Here we limit ourselves to pointing out that it is legitimate to argue against the idea that effects of either parents or siblings are artefacts of shared omitted variables. Indeed, if estimated effects of both parents and siblings reflected only or mostly the impact of unmeasured shared conditions, why are sibling effects so much larger than parental effects? Why are maternal and paternal smoking significantly different from each other? By definition, common conditions are equally shared by parents and siblings alike. The confounding should impart similar biases to naïve estimates in each case. However, the expectation is not borne out, as, for instance, coefficients measuring sibling and parental smoking differ substantially. There is thus the distinct possibility that sibling effects at least do in fact capture the direct influence of siblings' smoking status and that there may exist a gender-based effect of parents smoking on adolescents' behaviour.

FIGURE 3

Predicted probabilities of smoking in selected subpopulations (Kernel density estimate)



Source: PNAD data, IBGE (2008).

Results from biprobit models

We now model simultaneously the smoking behaviour of any two siblings found in a household by estimating biprobit models on selected samples of sibling pairs. The main advantage of the biprobit model is that estimates of measured covariates are purged from the effects of common but unmeasured conditions affecting the smoking status of both siblings in a pair. Biprobit models retrieve an important statistic—namely, the correlation between the smoking status of the sibling pair after accounting for measured shared and individual covariates. This statistic is a measure of residual correlation that can be due to the influence of unmeasured common conditions or, alternatively, the effect of the smoking status of one sibling on the other.

Alternative samples of sibling pairs

To obtain samples of sibling pairs, we first exclude all households with no or only one offspring. In the remaining households we form all possible sibling pairs within the age range of 15–25 years. This is the *master sample* of sibling pairs and can be used for analysis as long as we estimate models with appropriate clustering corrections. An alternative strategy is to choose via a single random draw one out of multiple possible pairs that could be formed in each household.

By joining these pairs across eligible households, we arrive at a representative sample of sibling pairs. Each household in the original sample is represented by one and only one sibling pair.

Because there are multiple samples of this sort, proper estimation would require us to estimate models with each of them and then average out estimates of parameters and adjusted standard errors. To simplify procedures, we opt to work with the master sample of sibling pairs, adding *ex post* clustering adjustments.

Testing of selected hypotheses using selected samples

In an effort to generate evidence in support of Hypotheses 1 through 4, we construct three different sub-samples: one consists of female pairs, one of male pairs, and a third of siblings of the opposite sex. For each pair, we estimate three biprobit models. The baseline model includes controls for a logarithm of age of both siblings, paternal and maternal education, two dummies for each youngster's school delay, ethnicity and region of residence. Model 1 is the same as the base model but constrains the effects to be the same across equations. For example, the effects of log of age are identical for members of the pair, and so are the effects of the remaining background characteristics. Model 2 adds three dummy variables for parental smoking plus a dummy variable that flags the older member of the pair. The effects of parental smoking (as well as of the other variables except for the dummy for birth order) are constrained to be the same across the two equations.

Table 3 displays parameter estimates and statistics for the three alternative models in the three data sets identified above. The first column contains the N, the number of observations. Column 2 displays the value of the Bayesian information criterion (BIC) statistic. The remaining columns in the table are as follows: columns 3–5 show estimates associated with parental smoking, where parental smoking is captured by a suite of three dummies (both parents smoke, only father smokes, and only mother smokes), and column 6 includes the residual correlation between the smoking status of the sibling pair (ρ).

The first result that stands out is the magnitude and significance of ρ, which proves that sibling effects are at least an important component of the familial influences on adolescent smoking. Furthermore, the BIC statistics (column 2) confirm that a constrained model such as Model 1 is more parsimonious and ought to be preferred to the base model. By the same token, the BIC statistics contrasting Model 2 with Model 1 strongly suggest the superiority of models that include parental smoking, especially in the case of the same-sex samples.

Second, not all parental influence is equal. The strongest effects occur when both parents smoke, followed by maternal-only smoking. Because maternal effects remain strong irrespective of the adolescent's gender, the possibility of *in utero* influences should be considered.

Intriguingly, paternal smoking is irrelevant in the female sibling pairs, but not in the male or opposite-sex pairs. In the single probit models (Table 2), there were no interactions between parental and adolescent gender. The prevalence of smoking increased with the presence of a parent who smokes, but it was not a function of the gender disparity between parent and adolescent. However, once we control for unmeasured shared conditions, Table 3 points to an interaction between parental and adolescent gender. This contrast between fathers' and mothers' smoking status supports the conjecture that parental effects are not merely a consequence of second-hand smoke, and that there is a behavioural component that requires identification.

Third, the effects of the dummy variable created to identify siblings' birth order appear to be in play in the case of males only in the biprobit models—unlike the simpler probit models, which did not identify any effect of birth order. Again, there is some evidence for the role-model mechanism, albeit in an attenuated form.

Table 3 also includes an extra piece of information to identify mechanisms leading to familial influences. As revealed by the very large values of ρ and their insensitivity to the addition of control variables, sibling effects are the dominant part of the familial influences on adolescent smoking. The correlations within same-gender pairs are large, ranging from .44 among males to .80 among females, and are virtually invariant when we add controls, particularly parental smoking. More intriguingly, the lowest correlation within sibling pairs occurs among opposite-gender siblings, suggesting that, as role theory would have it, there is little mutual influence between male and female siblings once we account for parental smoking and other shared conditions. Moreover, note that it is precisely in the case of the oposite-sex sample where the BIC statistic that helps to decide between the biprobit models (baseline, Model 1 and Model 2) attains an intermediate value when referring to Model 2, compared to the baseline model and Model 1.

TABLE 3

Biprobit models: selected results

Samples by the combination of siblings' sex	N	BIC	Parental smoking			ρ
			Both	Father	Mother	
I. Male–Male						
Baseline model	2,747	1,462,590	-	-	-	0.4770***
Model 1	2,747	1,507,259	-	-	-	0.4734***
Model 2	2,732	1,449,603	0.66***	0.31***	0.49***	0.4383***
II. Female–Female						
Baseline model	1,612	327,057	-	-	-	0.7967***
Model 1	1,612	331,234	-	-	-	0.7863***
Model 2	1,601	319,156	0.53***	-0.11	0.59**	0.7685***
III. Female–Male						
Baseline model	4,020	1,639,560	-	-	-	0.4792***
Model 1	4,020	1,737,100	-	-	-	0.4055***
Model 2	3,994	1,658,417	0.59***	0.42***	0.55***	0.3573***

Source: PNAD data, IBGE (2008).

*** =p<.01; **= p<.05; Standard errors in parentheses.

Here we hit an interpretational roadblock. If the correlations between sibling smoking statuses are so large, particularly within same-sex sibling pairs, and if they cannot be accounted for by either measured shared conditions or parental smoking, what is the exact mechanism that produces them? There is still the possibility that ρ simply reflects commonalities among siblings that we have omitted. One of these that looms large is shared influences originating in peer groups.

Indeed, the existence of common peer group influences could also explain why the residual correlation is so much smaller in the sample with opposite-sex pairs. We argue that the direct influence of sibling smoking could be at least as high as reflected in the values of ρ from the sample of opposite-sex pairs and at most as high as the one reflected in the value of ρ for the sample with same-sex siblings.

Is there any evidence of in utero effects?

The dominance of the influence of maternal smoking on adolescents' (of both sexes) smoking seems to be clear. Not only are the effects of maternal smoking strong, and stronger than those of fathers, but the influence of paternal smoking is only relevant for sons, and even for sons these are the weakest parental effects. This scenario is consistent with the possibility of maternal effects that operate *in utero*.

As is easy to understand, a test of the *in utero* exposure conjecture is data-demanding, as one would need information on maternal smoking (ever/never, duration, quantity), timing of smoking in relation to the pregnancy, and additional information on paternal smoking and other sibling smoking. This is a tall order for the more modest data set available to us and requires the support of sometimes questionable assumptions. However, we at least succeeded in classifying adolescents into a group whose mothers *were more likely to have been smoking* during their gestational period than those who were not exposed to such risks. Although necessary to fully test the *in utero* exposure conjecture, fine-tuned distinctions, including duration and intensity of smoking during the pregnancy, could not be implemented. Not shown is our estimated simple probit model adding the new exposure variable, as it neither attenuated other covariates nor had an important effect.

Another possibility is that the case for *in utero* hypotheses may not be the most compelling. The causality mechanism behind the dominance of maternal smoking may be the greater time mothers spent with their offspring due to still common gender roles in Brazil. An interesting proxy for time spent with offspring is the time spent performing domestic tasks. It may be the case that once time spent at home is considered, parental gender will lose power in explaining adolescents' smoking behaviour.

Also, it is worth mentioning that motherhood can be established for sure in the PNAD, but fatherhood cannot. Therefore, as fathers are mixed with stepfathers, they might not act as role models. In this sense, if we have a family where there is a resident non-smoking stepfather and a non-resident smoking father, it might make the effect of paternal smoking even weaker.

4 CONCLUSION

The evidence we gather shows that familial influences on adolescent smoking are uniformly strong and ubiquitous. Parental and sibling smoking both appear to reflect genuine direct effects, rather than being the outcome of artefacts produced by shared unmeasured conditions. Box 1 summarises the evidence we are able to gather to support strongly (Yes), mildly (Weak) or not at all (None) the four hypotheses formulated at the outset.

The fact that parental effects vary in subtle ways according to the gender of parents and offspring alike points to a complex behaviuoral mechanism, not one regulated by the production of second-hand smoking. Sibling effects are stronger and overwhelm the effects of parental smoking but are mostly visible within same-sex siblings, not in opposite-sex sibling pairs. This fact is consistent (but does not prove) that the mechanisms involved must be social rather than the result of unmeasured shared conditions. It is difficult to adjudicate between social mechanisms, some of which may involve influences of common peer groups, whereas others could be related to the imitation and adoption of role models (Ouyank 2004; Slomkowski et al. 2001; Swan et al. 1990). The absence of differentials by birth order surely points in the direction of peer group influences.

BOX 1

Summary of evidence supporting the four hypotheses

	Conventional probit	Biprobit
Hypothesis 1		
Familial effects are strong	Yes	Yes
Parents	Yes	Yes
Siblings	Yes	Yes
Hypothesis 2		
Sibling and parental effects are separable	Yes	Yes
Hypothesis 3		
Gender-specific parental effects	Weak	Yes
Hypothesis 4		
Gender-specific sibling effects	Weak	Yes
Birth order-specific effects	Weak	Weak
Hypothesis 5		
In utero effects	None	None

Lastly, *in utero* exposure, crudely measured as it was in this paper, turns out to be irrelevant. Because it is unlikely that one can adequately assess the existence, let alone the intensity or duration, of *in utero* exposure to nicotine with coarse indicators (such as the ones we used here), this is an area wide open for investigation. However, *in utero* hypotheses may not be the most compelling, once the mechanism behind the dominance of maternal smoking may be the greater time mothers spent with their offspring. The PNAD offers an interesting proxy for time spent with offspring: the time spent performing domestic tasks. It may be the case that once this variable is considered, differences in smoking behaviour between fathers and mothers will lose power, while explaining the probability of adolescents' behaviour. This hypothesis is in our research agenda and will be tested in the next version of this study. Also, there is the fact that motherhood can be established for sure in the PNAD, whereas fatherhood cannot. Therefore, if we have a family with a resident non-smoking stepfather and a non-resident smoking father, it may account for making the influence of paternal smoking on adolescents' behaviour even weaker.

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NOTES

4. Argys and Rees (2008) use variation in the mandated age at which children begin school across US states as an exogenous source of variation in exposure to older classmates during teenage years.

5. In this paper we do not use information elicited in the Special Survey on Smoking that includes retrospective smoking histories of *one* selected individual within households.

6. In households with more than one pair of siblings, we randomly select one of them. This strategy reduces the number of cases in the sample (households with more than two siblings will enter into the sample represented by only one of two or more possible pairs) but prevents serious clustering problems. We pursued an alternative solution, which was to repeat estimation and create alternative sampling that resulted from different draws of pairs from each household. Because the variance of estimates is trivial, we only display results from one of the draws.

7. We also estimated parameters in a sample that includes *all* households with at least one surviving individual between the ages of 15 and 25. In this sample we are only able to identify the effects of parental smoking. These estimates are virtually identical to those obtained with the more restricted sample used here when the model excludes a control for sibling smoking status.

8. Although we estimate many models that contain variables other than those in the table (including indicators of exposure to anti-smoking advertisements and campaigns), we will only refer to the patterns of results obtained with the most elementary and parsimonious models.

9. To avoid clutter, we only display the effects and associated standard errors of a subset of covariates.

10. We also estimated models with variables for sib smoking status and birth order separately from those for sib smoking status and sex differences. Model 4, however, is more parsimonious and fits the data better.

International Policy Centre for Inclusive Growth (IPC-IG)
United Nations Development Programme

SBS, Quadra 1, Bloco J, Ed. BNDES, 13º andar
70076-900 Brasília, DF - Brazil
Telephone: +55 61 21055000

ipc@ipc-undp.org ▪ www.ipc-undp.org