THE SOCIAL AND PHYSIOLOGICAL EFFECTS OF PARENTAL SMOKING ON YOUTH ADDICTION: THE CASE OF BRAZIL

Alberto Palloni, Laeticia Souza, and Leticia Marteleto

PROBLEM

Elsewhere (Palloni et al, 2012) it has been shown that Brazil is a singularity in the smoking epidemic spreading across low and medium income countries. Indeed, smoking prevalence rates by age are concave downward suggesting that anti-smoking campaigns launched in the country beginning in 1989 are making a dent among the youth, the most vulnerable sector of the population. Since 1989, the National Institute of Cancer (INCA), an agency of the Ministry of Health responsible for the national cancer control, coordinated actions developed in partnership with the state and municipal Health departments and various sectors of civil society organizations, especially, scientific societies and professional councils in the healthcare field (Cavalcante et al., 2005).

Can this feature of Brazilian smoking behavior be sustained? Can one confidently expect that adolescent smoking will continue to lag behind adult smoking until the epidemic extinguishes itself or reaches a steady state where it is contained within the confined of fringe subpopulations?

The answer to this question depends on a number of factors, including the scope and continued efficiency of the anti-smoking campaigns and education improvements. An equally important but completely unexplored factor is the degree to which youth smoking is propped by network of peers, sibling imitation and parental influence. Antismoking campaigns are as good as is their ability to erode the influence exerted on individuals at risks by those who are closest to them: parents, siblings and friends.

In this paper we examine the association between parental and sibling's *(alter)* smoking on the one hand, and the smoking of youngsters aged 15 to 25 *(ego)* living in the same households. Our preliminary findings indicate that there is a strong association between alter's and ego's smoking of the order of .45 implying relative risks of smoking among ego's (whose alters

smoke) of the order of 2.4, a figure of comparable magnitude and sometimes larger than the relative risk associated with lower education. The magnitude of this association (and corresponding relative risk) is surprisingly large and the paper's main objective is to identify the mechanisms producing it.

We investigate three mechanisms. The first is shared environments of egos and alters. Individuals who share the same household also share behaviors, material conditions, and exposure to exogenous influence (Davis, Nonnemaker and Farrelly, 2007). And these may all conspire to elevate (decrease) the risk of taking up smoking and persevering in the behavior. The second mechanism is imitation or the influence of one person's smoking behavior on another. Imitation is more likely when the person practicing the behavior is older, has more authority or a privileged status, all conditions satisfied by parents and older siblings vis a vis a younger siblings. Thus, parental or sibling smoking provides an opportunity and sometimes an incentive for youngster imitation of the smoking behavior (Olvera, Poston and Rodriguez, 2006; Leonardi-Bee, Jere and Britton , 2011). Further, onset of smoking among younger children is facilitated since obstacles and restrictions are of lesser importance and sharing of the tobacco product lowers individual's economic costs.

The third and perhaps most intriguing mechanism is exposure to smoking *in utero* (Roberts, Munafo and Rodriguez, 2005). Extant, albeit inconclusive research, suggests that mothers who smoke during pregnancy not only seed conditions that increase the odds of fetal oxygen deprivation but also blood driven nicotine sensitizes nicotine receptors that eventually make the child more likely to become addicted to cigarettes (O'Callaghan, 2009). Nicotine and other chemical compounds contained in cigarettes can also exert a host of other influences, including disregulation of neurotransmitters and hypothalamic development and function.

The models we propose below are designed to identify the mechanism(s) responsible for the strong association between youngsters' smoking and parental and sibling smoking.

DATA

We use two related data sets. First, the 2008 Brazilean PNAD (*Pesquisa Nacional por Amostra de Domicílio*) which is a nationally representative survey collected annually by the Brazilian Census Bureau (*Instituto Brasileiro de Geografia e Estatística, IBGE*). PNAD is analogous to the US CPS and consists of a probability-based, stratified, multistage household survey. The sampling design follows a three-step probabilistic procedure based first on municipalities, then census tracts within municipalities, and finally households within sectors. The PNAD contains information on every household and family member, and is therefore a rich source of social, economic and demographic data, including information on schooling and income for all family members older than 10. We make use of a special battery of questions on smoke that were introduced in the Supplemental Health Survey of 2008. This supplement comprised several topics among which smoking was included and was applied to all members of the household who were older than 15. The items contained in the smoking module are a subset of the items applied in the Special Survey on Smoking Use (see below).

Second, we also use the Special Survey on Tobacco Use carried out using a one-third subsample of the households selected for the sample defined by the PNAD basic Sampling Plan. In each of the chosen households one resident aged 15 or older was randomly selected and interviewed for the Special Survey on Tobacco Use through which one can reconstruct the smoking history of the respondent. Because linkage of individuals in the Special Survey on Tobacco Use and the household to which he belongs is feasible, we can append all household characteristics (including parental smoking) to those younger than 25 who were interviewed in the Special Survey on Tobacco Use.

The 2008 PNAD sample size is 391,868 households. We use an analytic sample of 15 to 25 year-old people numbering about 8,605. To accurately include family variables in the models, we restrict the sample to sons/daughters of the head of the family and excluded non biologically related members.

MODELS AND ESTIMATION

We estimate several sets of models. The first set of models are a set standard logit/probit models for the probability of being a current smoker among youngsters aged 15-25 in a

household. We use different upper age restrictions (18,20,22,24) to minimize the risk of selection. Older children who are out of the home and for whom we have no information on smoking could be selected on a number of characteristics related to smoking behavior. However, preliminary analysis suggests that if there is any selection, it does not affect the parameters of interest to us. These models control for a number of variables (parental and ego's education, region, parental and maternal labor force status, ethnicity, type of household, etc…). Our main targets are the effects of dummy variables that classify siblings and parents by smoking status. The first set of models only focuses on current smoking whereas other models take advantage of the various combination of current versus past smoking. These are useful to better understand the nature of the effect. Thus, the effects of a parent currently smoking should be stronger than the effects of past parental smoking if the mechanism is one of imitation but not if the main pathway is through shared environments or via uterine exposure. These models pay considerable attention to differences between maternal and paternal smoking as well as those between females and males siblings.

The second set of models is designed to identify the existence of *in utero* effects. To do this we use of information on dates of maternal smoking initiation and quitting behavior. We then distinguish children who share all other conditions (including current parental and sibling smoking) but who were (were not) exposed to in utero maternal smoking. The effects in preliminary results are large and in the expected direction. They are larger than those that could be imputed to pure imitation or shared environments. This evidence supports the conjecture that addiction to nicotine is reinforced if first exposure to the substance precedes birth.

The third set of models consists of biprobit model where we use pairs of randomly selected siblings and include the same covariates that were used in the standard probit models. The bivariate probit model enables us to estimate effects that are purged of biases induced by unmeasured shared conditions and, as a consequence, must be smaller than those from the standard probit model. One of the parameters estimated by bivariate probit models is the residual correlation of smoking behavior between the pair. This correlation must be

attributable to imitation effects (the influence of smoking of one member of the pair on the other) or, alternatively, on unmeasured non-shared conditions that are correlated across members of the pair. Preliminary estimates suggest that the residual correlation of the biprobit model is quite large and is not reduced when we introduce controls for aggregate exposure to community anti-smoking campaigns.

The fourth and last class of models are survival models designed to determine the influence of parental and sibling smoking on younger siblings timing of onset of smoking initiation and the probability and timing of quitting behavior. These analysis use semi-parametric proportional and non-proportional hazard models with unmeasured heterogeneity and are designed to pinpoint with precision the different effects of parental and sibling behavioral combinations: are parent's who quit smoking more likely to influence their children not to smoke at all or to quit smoking than parents who are current smokers? Are these influences the same regardless of the gender of the parent?

IMPLICATIONS

The implications of our findings are twofold. First and most devastating is that smoking behavior has a high degree of intra-family "stickiness", almost guaranteeing a sort of socially driven intergenerational transmission of smoking. The second is that there is also a physiological route whereby mothers can pass on the addiction to their daughters (more so than to sons).These two implications suggest that beneficial behavior modification of parents may be an efficient route to lower smoking addiction in the next generation. By the same token, a campaign that effectively reduces children smoking almost guarantees very low rates of smoking uptake among their offspring.

REFERENCES

Cavalcante, T. M., 2005. O controle do tabagismo no Brasil: avanços e desafios. Rev. psiquiatr. clín., vol.32, no.5, p.283-300. ISSN 0101-6083.

Palloni, A., B. Novak and G. Pinto, 2012. The enduring effects of smoking in Latin America" Paper presented at the Population Association of America Meetings, San Francisco, April 2012.

Roberts, K. M. R. Munafo, D. Rodriguez et al., 2005. Longitudinal analysis of the effects of prenatal nicotine exposure on subsequent smoking behavior of offspring. Nicotine & Tobacco Research Vol 7(5) 1-8.

O'Callaghan, F. V., Al Mamun, A., O'Callaghan, M., Alati, R., Najman, J. M., Williams, G. M. and Bor, W., 2009. Maternal smoking during pregnancy predicts nicotine disorder (dependence or withdrawal) in young adults – a birth cohort study. Australian and New Zealand Journal of Public Health, 33: 371–377. doi: 10.1111/j.1753-6405.2009.00410.x.

Olvera N., Poston W. S. C., Rodriguez A., 2006. Parental socialization of smoking initiation in Latino youth. *Journal of Adolescent Health*; 39:758-760.

Davis K.C., Nonnemaker J., Farrelly M.C., 2007. Association between national smoking prevention campaigns and perceived smoking prevalence among youth in the United States. *J Adolesc Health*. 241:430–436.

Leonardi-Bee J., Jere, M. L., Britton J., 2011. Exposure to parental and sibling smoking and the risk of smoking uptake in childhood and adolescence: a systematic review and meta-analysis. *Thorax - Smoking cessation update*; 66:847-855 doi:10.1136/thx.2010.153379