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Rebel with(out) a cause? Inter-generational smoking dependence in Chinese households*

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Abstract

This paper examines inter-generational (parent-child) dependence and intra-personal smoking persistence within Chinese households. A dynamic bivariate probit specification is estimated using data from the Chinese Health and Nutrition Panel Survey (CHNS). Our results (unsurprisingly) show that, for both parents and children, individual past smoking behaviour increases the risk of smoking today; however, past smoking by the parent (child) reduces the likelihood of a child (parent) smoking today - to the best of our knowledge, a novel inter-generational effect in the literature. Importantly, these results have relevant implications in the design and implementation of policies aimed at reducing smoking prevalence.

JEL Classification: D1, I12, I18

Keywords: smoking, inter-generational effects, dynamic probit, anti-smoking policies.

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1 Introduction

In many areas of analysis it has been found that current individual behaviour is strongly influenced by past behaviour and past experiences. Smoking decisions are a prime example of this. Smoking is physically addictive: individuals who experience high intakes of nicotine tend to show a strong lifelong dependency on tobacco.[1,2] More generally, smoking is state dependent: individuals who smoked in the past are more likely to smoke today and past smoking intensity also explains current smoking intensity (Gilleskie and Strumpf, 2005).

Clearly, smoking decisions go well beyond a physical dependence on nicotine. In addition, there is significant evidence that social experiences influence smoking behaviour. Various types of peer and family effects have been identified as very relevant for smoking decisions: spousal behaviour (Clark and Etilè, 2006), adolescents friends' behaviour (Card and Giuliano, 2013) and inter-generational (parent-child) transmission of smoking behaviour (Göhlmann et al., 2010; Simon, 2016).[3] Vandewater et al. (2014) argue that the latter extends all the way to grandparents (across three generations). In addition, this inter-generational effect appears to be gender-based: sons primarily imitate their fathers whilst daughters follow their mother's smoking decisions (Loureiro et al., 2010).[4]

We look at these inter-generational effects, but, differently from the earlier literature, we consider the two-way interdependence between smoking decisions of different family members. Parent-child relationships were for a long time viewed through the lens of unidirectional models of influence, where parents are assumed to impact their children's development and are, thus, the key element in the parent-child relationship (Paschall and Mastergeorge, 2016; see also Bell, 1979). Göhlmann et al. (2010) implicitly assume such a unidirectional model

[1] Smoking appears to be associated with other explanatory factors as well. Evidence suggests that observed individual characteristics – socio-demographic status (Gilman et al., 2003), gender, age, marital status, education and employment status (Anger et al., 2011) – as well as unobserved individual heterogeneity (e.g., degree of risk aversion, rate of time preference or health attitudes – see Christelis et al., 2011) are typically associated with smoking decisions.

[2] For instance, Benowitz and Henningfiled (2013) propose a public health strategy anchored on a mandated reduction of the nicotine content of cigarettes.

[3] In a recent paper, Carrieri and Jones (2017) also look at inter-generational within a household, but their focus is on children's (below 15 years old) exposure to nicotine (passive smoking) due to parents' smoking behaviour.

[4] Göhlman et al. (2010), looking at Germany, do not find evidence of this gender-based effect uncovered by Loureiro et al. (2010) in the UK.

to understand inter-generational smoking effects between parents and their children.[5] However, evidence suggests that both parents and children play a crucial role in their relationship and the model has, therefore, evolved into one of bilateral effects, where parents and children mutually influence each other and are co-responsible for dyadic outcomes. The seminal contributions in the psychology literature supporting this model were those of Bell (1968), Thomas et al. (1963) and Thomas and Chess (1977), which, to the best of our knowledge, have not yet resonated in the economics literature.[6] In the context of smoking, for example, the child may look at parents as role models and behave as they do or they could rebel against their parents and behave in an opposite way (the typical unidirectional effects). But parents may also respond to observed child behaviour: upon observing that their child mimicked their smoking behaviour, they may feel guilty and stop smoking as a psychological compensation mechanism; or by observing that, unlike them, their child chose not to smoke, they may feel inspired to behave in a similar way.

We choose to focus on China for two main reasons. Firstly, because of its size and the observed prevalence of smoking: there are an estimated 301 million smokers (28% of the population) in China (WHO, 2010), nearly one-third of the world's total.[7,8] This makes China the largest consumer of tobacco in the world (Li et al., 2011). Secondly, the China Health & Nutrition Panel Survey (CHNS), an unbalanced panel covering up to 20 non-consecutive years of lifetime smoking behaviour, allows for the study of dynamic interactions between past and current decisions over a significant part of a parent-child's life.[9]

[5] Göhlmann et al. (2010) also look at inter-generational effects on starting smoking and find that when parents smoke, the offspring's probability of starting to smoke increases (unidirectional inter-generational effects, from parent to child). Their paper differs from ours in two additional dimensions: firstly, only children aged 12 to 22 years old are considered, whereas we place no upper limit on child age and look at effects throughout a child's lifetime. Secondly, Göhlmann et al. (2010) look at contemporaneous inter-generational effects, that is, the influence of parents' current smoking behaviour on their childs' smoking initiation decision; by contrast, our data allows for an analysis of inter-generational effects over time, identifying the impact of past decisions on current smoking status.

[6] Bell (1968) views socialization as a mutually interactive process where children (or 'child effects') are also drivers of parent-child relationships. Thomas et al. (1963) and Thomas and Chess (1977) identify child temperament as an influential factor in the parent-child relationship.

[7] It is estimated that every year one million smokers die because of tobacco-related illness, with an estimated 2.2 million deaths by 2020 (Jiang et al., 2009).

[8] Among these smokers, only 2.4% are women, more than half started to smoke before the age of 20 (WHO, 2010), and 75.6% have no plan to quit smoking (International Tobacco Control Policy Evaluation Chinese Survey, available from http://www.itcproject.org/countries/china).

[9] Kenkel et al. (2009), Xiao et al. (2014) and Guo and Sa (2015) also use the CHNS to look into smoking

Methodologically, in order to identify and quantify these effects of past smoking behaviour on current smoking status, we estimate a bivariate dynamic probit model by simulated maximum likelihood allowing for correlated random effects between parent and child.[10] Unsurprisingly, we find strong evidence of dynamic effects associated with habits and/or addiction which decay over time. However, we also find evidence of inter-generational bilateral effects within each parent-child pair, whereby the (smoking) behaviour of one member of the pair exerts an influence on the other, but we find this influence to be *negative*, that is, the probability of a child smoking is negatively affected by the parent's past behaviour (and vice-versa). This is, to the best of our knowledge, a novel result in the literature and we define it as a *negative model* effect, whereby a member of the pair behaves in way that is opposite to the past behaviour of the other member of the pair.

Our results have interesting policy implications. Whether and how smoking behaviour is transmitted across generations is relevant when designing or implementing policies aimed at reducing smoking prevalence, either of parents or of children (or both). Examples of such policies are anti-smoking campaigns (including health warnings on packages), advertising restrictions, indoor (or outdoor) smoking bans, tobacco taxes or the promotion of nicotine addiction treatment.[11] For instance, the results of Göhlmann et al. (2010) suggest that targeting parents in anti-smoking campaigns is likely to be beneficial, as it reduces the likelihood of smoking initiation by their children. We find this to be true (i) only in the short run, where targeting parents with such policies would reduce the overall (parents and children) smoking prevalence, and (ii) only if such policies are permanent in nature. By contrast, using our long run estimates, we find that policies aimed at children are preferable. Again, the role played by the inter-generational effect - thus far unexplored in the literature - hinders the success of standard anti-smoking policies and introduces a trade-off for policymakers, when

behaviour in China.

[10] This is consistent with the Simulated Annealing Expectation-Maximization algorithm used by Clark and Etilé (2006). We therefore extend Plum's (2013, 2014) univariate simulated maximum likelihood programme, in a way that is similar in nature to Miranda (2010).

[11] There is a large body of literature analysing and evaluating these policies. For instance, Callinan et al. (2010), in a review of over 50 studies, finds limited evidence on the effectiveness of smoking bans in reducing active smoking. Jones et al. (2015) reach similar conclusions when looking at the introduction of smoking bans in Scotland (2005) and England (2007). For a broad overview, especially on taxes, see Chaloupka and Warner (2000).

choosing the policies' target populations, that needs to be carefully considered.

The paper is organized as follows: in section 2 we look at related literature, section 3 describes the data, section 4 contains the empirical model, section 5 presents our results, section 6 discusses policy implications and section 7 concludes. Several appendices contain additional material.

2 Related literature

A significant body of literature (both theoretical and empirical) has emerged within the context of smoking decisions. Therefore, we refer mainly to prior literature on intra-personal persistence (experience-based formation, habit formation and individual addiction literature) and inter-generational transmission in smoking.

At the individual level, to make many decisions, people recall their experience. In essence, the experience-based formation literature (e.g., Layton, 1978; Botsch and Malmendier, 2015; Alesina and Fuchs-Schundeln, 2007) assumes that individuals who have (not) experienced an event in the past are more likely (less likely) to experience it again. Additionally, past experience can be viewed as habits (e.g., Pollak, 1970). The consumption of tobacco can be strongly correlated to habits, since the development of smoking habits involves a transition from occasional smoking to habitual behaviour. For instance, Clemens et al. (2014) claim that smoking is started by poor self-control and it becomes a habit via repetition.

From an inter-generational perspective, a large body of literature suggests strong linkages between parents and children's smoking behaviour. In a Chinese cross-sectional survey, out of 6674 respondents, 75% mentioned the source of cigarettes is home and 10% obtained cigarettes from self-purchase (Hesketh et al., 2001). In the US Panel Study of Income Dynamics (PSID), regardless of the generation, parent smoking behaviour has a direct impact on offspring smoking behaviour (Vandewater et al., 2014). Göhlmann et al. (2010) reach similar conclusions regarding the parents' smoking status on their children's likelihood of smoking initiation.

In these contexts, smoking behaviour is clearly family-influenced but implicitly assumes a unidirectional model of influence, whereby parents' behaviour are key drivers of children's behaviour (Bell, 1979). However, following on from Bell (1979), significant evidence has

emerged in the psychology field in favour of a bilateral effects model in which parents and children mutually influence one another. These bidirectional effects (especially the role played by children in influencing the parent-child relationship) were analysed in a wide variety of circumstances: the use of corporal punishment (e.g., Holden et al., 1997); maternal sensitivity and children's development (e.g., Spinrad and Stifter, 2002; Martin et al., 2012) or child disruptive behaviour (e.g., Burke et al., 2008; Del Vecchio et al., 2010; Shaffer et al., 2013). Closer to the aim of our paper, Otten et al. (2008) look at bilateral parent-child effects in alcohol use, generally finding a positive relationship between young children's alcohol use and that of both parents and a small positive effect on parents' behaviour driven by older children's alcohol use.

From a methodological viewpoint, analysing bilateral effects requires data from both members of the pair (parent-child) and techniques that take into account the non-independence of the data. In the psychology field, Del Vecchio et al. (2010, p. 518) note that "behaviours by both members of the dyad are influenced by their own behaviour (actor effects) and by the behaviour of their partner (partner effects)"; in particular, "the behaviour of the actor at time t could be influenced by the behaviour of the partner at time t-1, but could also be influenced by his/her own behaviour at time t-1".[12]

3 Data

The panel data are from the CHNS (Chinese Health and Nutrition Panel Survey), an ongoing open cohort, nationally representative, health and nutrition survey of households randomly selected in mainland China.[13] The CHNS is an unbalanced panel dataset covering approximately 7,200 households (30,000 individuals) in 15 (out of 34) provinces and municipal cities.[14] This study uses 8 surveys spanning 20 years (1991 - 2011) of lifetime smoking be-

[12] In psychology, a largely used model to address the non-independence of the data is Kenny's (1996) Actor-Partner Interdependence Model (APIM) (see also Kashy and Kenny, 1999; Cook and Kenny, 2005; Kenny et al., 2006), which considers the pair as the main unit for analysis and is able to identify actor and partner effects. From a technical or statistical viewpoint, APIM is typically implemented through structural equation modelling (SEM) or multilevel modelling.

[13] The China Health and Nutrition Survey is publicly available on http://www.cpc.unc.edu/projects/china.

[14] China has 34 provincial-level administrative units: 23 provinces, 4 municipalities (Beijing, Tianjin, Shanghai, Chongqing), 5 autonomous regions (Guangxi, Inner Mongolia, Tibet, Ningxia, Xinjiang) and 2 special administrative regions (Hong Kong, Macau).

haviour. The survey was not carried out at regular time intervals: these 8 surveys were carried out in 1991, 1993, 1997, 2000, 2004, 2006, 2009 and 2011. Less than 3% of the smoking individuals are females, and they are not considered in our study.

We consider each father (who we refer to as 'parent') and son (who we refer to as 'child') sharing the same household identity number as one pair. In order to develop insights into how parent and child interact, we consider only pairs observed in at least 2 surveys. Note, however, that a parent-child pair is not necessarily observed in (at least two) consecutive surveys: for example, a parent-child pair may be observed in 1993 and then again only in 2000. For this reason, we use the term 'wave' to define the surveys in which a parent-child pair is observed, where wave 1 corresponds to the first survey in which the pair is observed, wave 2 corresponds to the second survey in which the pair is observed, and so on. Naturally, a parent-child pair is observed in at most 8 waves. Note also that this means the calendar time gap between observed behaviour for a pair can vary across pairs. Our econometric approach requires all the regressors to be non-missing, leaving us with 2,185 observations over 8 waves (see Table 1) and 9 provincial-level administrative units (see Figure 1).

Parent-Child Pair	**Waves**								
	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6	Wave 7	Wave 8	Total
Total	854	854	303	120	38	13	2	1	2185

Table 1: Parent-child pairs by waves (number of observations)

Across the 8 waves (see Table 2), parent and child share the same smoking pattern more often than not, that is, the percentage of observations in which both parent and child either smoke or do not smoke is larger than the alternative. Intertemporally, the effect of personal persistency is indicated by the diagonal elements of Table 2: for instance, 17% of all observations refer to cases where both parent and child smoke in period t and $t-1$, whilst in 5% of all observations neither smokes in both periods.

The effect of inter-generational dependence is shown by the off-diagonal elements of Table 2. Take for example columns '10' and '01' in period $t-1$. In column '10', 7% of the children whose parents were smokers at $t-1$ started or re-started smoking in period t, while 3% of parents whose children were nonsmokers at $t-1$ stopped smoking; in column '01', only 1%

of the parents whose children were smokers at $t-1$ switched from non-smokers to smokers.

Current time=t	**First wave**	**Other waves**				
		Previous time=t-1				
	.	**11**	**10**	**01**	**00**	Total
11	14%(311)	17%(374)	7%(147)	1%(30)	1%(31)	893
10	14%(310)	4%(79)	10%(225)	0%(9)	2%(42)	665
01	4%(82)	2%(35)	1%(22)	4%(79)	2%(36)	254
00	7%(151)	1%(26)	3%(62)	1%(18)	5%(116)	373
Total	854	514	456	136	225	2185

1=Smoke; 0=Don't Smoke; e.g. 11=parent and child both smoke; 10=parent smokes, child does not smoke

Table 2: Inter-generational inter-temporal smoking dependence: percentage (number of observations)

On average, the age gap between parents and childs is around 20 to 30 years (see Figure 2 in Appendix A).[15] For both parents and children, the age distributions for smokers (noted as '1') and non-smokers (noted as '0') are very similar, although a higher percentage of parents are smokers compared to children. Within each parent-child pair, parents are more likely to be married than their child: 92% of parents are married compared to only 42% of childs (see Table 7 in Appendix A).[16] Finally, Figure 3 (Appendix A) shows that children are better educated than parents and a large proportion of smoking parents exhibit a low level of education. For more details on other variables, please see Table 8 in Appendix A.

4 Econometric Model and Estimation

4.1 Model

We consider a dynamic bivariate probit model to represent the smoking decisions of parent (p) and child (c). For person $j \in \{p, c\}$ in household $i = 1, ..., n$ (n households in total) and in period $t = 2, .., T_i$ (T_i is the number waves in which a parent-child pair is observed), observed smoking (Y) and latent smoking (y^*) are defined as:

[15] At the 1st age quartile, the age gap is about 20 years. At the median and at the 3rd age quartile, the age gap increases to 30 years.

[16] This is a relevant variable for our analysis, because inter-generational smoking dependence may be influenced by the child's marriage status, as the spouse may exert a stronger influence over the child than the parent.

$$Y_{j,i.t} = \begin{cases} 1, \text{ if } y^*_{j,i,t} = \beta_j X'_{j,i,t} + \alpha_j Y_{j,i,t-1} + \gamma_j Y_{-j,i,t-1} + v_{j,i,t} \geq 0 \\ 0, \text{ otherwise} \end{cases} \quad (1)$$

where $y^*_{j,i,t}$ is a function of the observed smoking behaviour ($Y_{j,i,t-1}$) of person j in household i in the previous period $t-1$ and of his counter-party $-j$ ($Y_{-j,i,t-1}$) in the same period; α captures the effect of smoking persistency (or the degree of addiction/habit) whilst parameter γ shows the degree of inter-generational dependence in smoking; X is a vector of exogenous variables, such as age or educational level.[17]

The composite error is

$$v_{j,i,t} = a_{j,i} + u_{j,i,t} \quad (2)$$

where a is the individual random effect and u is the idiosyncratic shock. The idiosyncratic shocks are normally distributed with zero mean, unit variance and are uncorrelated over waves, with each other and with the random effect. For each household i at time t ($t > 1$), $v_{p,i,t}$ (composite error for parent) and $v_{c,i,t}$ (composite error for child) follow a bivariate Normal distribution: $N\left(0, \begin{bmatrix} \sigma_p^2 + 1 & \rho\sigma_p\sigma_c \\ \rho\sigma_p\sigma_c & \sigma_c^2 + 1 \end{bmatrix}\right)$, where ρ is the correlation, and σ_p^2 and σ_c^2 are the variance of the random effects for parent and child respectively.

The complete model includes an initial condition equation (in order to address the latent variable specification for period 1) and additional equations (for subsequent periods), which forms a system. Appendix B contains a detailed description of the underlying econometric approach to estimate this system.

4.2 Estimation program

Because of the cross wave correlation of the random effects, we estimate the system by maximising the likelihood with respect to the parameters across all waves and all pairs simultaneously, including the initial wave estimation. This is similar to Plum (2013, 2014) and Miranda (2010).[18] Since calculation of the likelihood function requires multivariate

[17] It is worth noticing that the inclusion of the actual smoking behaviour instead of the propensity to smoke (as in Labeaga, 1999) allows us to estimate the true state dependence of smoking behaviour working through both persistence and dynamic peer effects.

[18] Puhani (2000) shows, through Monte Carlo simulations, that these full information estimators perform better (in terms of efficiency) even in small samples.

probabilities at different points in the multivariate distribution, we use simulated maximum likelihood to calculate the objective at each iteration.

Unfortunately, there is no publicly available program to estimate a dynamic bivariate random effect probit model by simulated maximum likelihood (see also Miranda, 2010). Plum (2014) has written and distributed a multivariate dynamic SML probit program for an unbalanced panel but in which there are no lagged peer effects in the latent variable equations (although the random effects can be correlated). His model only allows for limited peer interactions via the random effects of different generations. In this case, we cannot capture the full inter-generational dependence and personal persistence in smoking. We have therefore written a new bivariate dynamic program, extending Plum's (2013, 2014) dynamic probit programs. To do this we also use multivariate normal probabilities, calculated using the Geweke-Hajivassiliou-Keane (GHK) simulator. In particular we allow for correlation between equations of the random effects as in Miranda (2010) and between the initial and later waves for each parent-child pair as in Plum (2013, 2014).

5 Results

Table 3 shows the estimated results for the initial wave of each parent-child pair and the results for each later wave of that pair in which the dynamic lagged effects are included. Apart from past smoking behaviour, the regressors include individual characteristics of each member of a pair: continuous regressors age, individual income, as well as categorical or dummy variables for marital status, education level, employment/school enrollment status (for parent and child respectively), being urban (and living in a city or suburbia) or rural and the province of residence. The dependent variable is coded 1 for smoking and 0 otherwise.

The most interesting aspect we wish to explore is the interplay of past experiences and past behaviour in each equation for parent and child. Recall that this is an unbalanced panel of parent-child pairs where each pair is observed in waves with variable time gaps from the preceding waves. Lagged smoking behaviour is defined as smoking in the preceding wave, that is, the basic unit of timing of past experiences is the preceding wave in which a parent-child pair is observed, which may not necessarily coincide with the preceding survey. Thus, for some pairs, the time gap between successive observations (waves) can vary between 2

and 8 years (either because the pair was not observed in some surveys and/or because the survey was not carried out at regular time intervals). But experiences or habits from longer ago are likely to have different effects compared to more recent experiences/habits. To allow for the differential time effect between observations, we define dummy variables associated with the reported age of a pair's member in two successive observations (waves): an age gap of 2-4 years and an age gap larger than 4 years.

5.1 The Initial Wave

For the child, the initial smoking state is significantly (at the 0.1% significance level) affected by age: the probability of being smokers follows an inverted U-shaped pattern in age with a maximum in the mid 20s. It is also affected by the education level: a higher education level reduces the chance of a child smoking. Interestingly the marital status of the child is unimportant both in the initial wave equation and in the equation for subsequent waves. In the initial wave, parents' age matters, as older parents are less likely to smoke; by contrast, married parents are more likely to smoke. The education level is significant (at the 5% level) for parents and appears with a negative coefficient, suggesting that more educated parents are less likely to smoke. Children who are still in school are also less likely to smoke.

5.2 The Later Waves

For each equation (parent and child), we then include as regressors the previous wave smoking status of that pair member (which we define as lagged selfsmoking) as well as the other pair member's smoking status in the previous wave (lagged peersmoking). Additionally, we interact these variables with the age gap dummies, in order to evaluate the differential time effects. In particular, we refer to the interaction with an age gap of 2 to 4 years as a 'medium run' effect and with an age gap larger than 4 years as a 'long run' effect. Results are presented in Table 3.[19]

The child's educational level impacts negatively on the child's propensity to smoke, but

[19]The variances and correlations of the random effects are quite precisely determined: the variance for the parent's equation is larger than in the child's equation. Thus, the variability of random effects between parents is greater than between children. The correlation is positive (0.65) and statistically significant. This positive correlation may indicate common genetic effects within a pair that distinguish that pair from other pairs.

	Parent	**Child**
Initial wave		
Age	-0.02^{**}(-2.36)	0.267^{***}(3.72)
Age^2		-0.004^{***}(-2.91)
Married	0.91^{***}(3.42)	-0.01(-0.07)
Education level	-0.13^{**}(-2.24)	-0.09(-1.63)
In school		-0.83^{**}(-2.27)
Constant	1.22^{***}(2.64)	2.62(0.31)
Remaining waves		
laggedSS(lagged selfsmoking)	**0.81^{***}(2.86)**	**1.02^{***}(5.31)**
laggedPS(lagged peersmoking)	**-0.53^{***}(-2.69)**	**-0.56^{***}(-3.11)**
Agegap(2to4yr)	0.24(0.97)	0.13(0.67)
Agegap(>4yr)	0.23(0.86)	0.26(1.17)
LaggedSS . Agegap(2to4yr)	-0.54^{*}(-1.89)	-0.47^{**}(-2.16)
LaggedSS . Agegap(>4yr)	-0.56^{*}(-1.83)	-0.63^{***}(-2.65)
LaggedPS . Agegap(2to4yr)	0.23(0.96)	0.28(1.47)
LaggedPS . Agegap(>4yr)	0.52^{*}(1.82)	0.16(0.65)
Age	-0.007(-0.90)	0.012(1.14)
Income (RMB)	$-1.48e-06$(-0.18)	$4.77e-06$(0.76)
Married (yes/no)	0.34(1.52)	0.13(1.28)
Education level	-0.03(-0.51)	-0.18^{***}(-3.33)
Working now (yes/no)	0.40(1.47)	
In school (yes/no)		-0.29(-1.27)
Farmer (yes/no)	0.23(1.39)	-0.08(-0.67)
Live in city (yes/no)	-0.08(-0.27)	-0.15(-0.64)
Live in suburban area (yes/no)	0.11(0.56)	0.20(1.42)
County/town capital	$-.01$(-0.05)	0.29(1.44)
Liaoning Province	-0.14(-0.42)	0.17(0.69)
Heilongjiang Province	-0.44(-0.89)	-0.43(-1.17)
Jisangsu province	-0.65^{**}(-2.54)	-0.30^{*}(-1.65)
Shandong Province	-0.60^{**}(-2.15)	-0.41^{**} (-2.12)
Henan Province	-0.59^{**}(-2.23)	-0.22(-1.19)
Hubei Province	-1.11^{***}(-3.70)	-0.37^{*} (-1.69)
Hunan Province	-0.78^{**}(-2.53)	0.133(0.58)
Guangxi Province	-0.46^{*}(-1.96)	-0.11 (-0.67)
Constant	0.71(0.97)	0.18(0.57)
Sigma for parent	1.35^{***}(12.93)	
Sigma for child	0.81^{***}(16.88)	
Theta	0.83^{***}(13.44)	
Rho corr	0.65^{***}(4.45)	
Log likelihood	-2356.7074	
Number of observations	2185	
t statistics in parentheses	* p<0.10, ** p<0.05, *** p<0.01	

Table 3: Parent-child inter-generational smoking behaviour

the parent's educational level appears to play no role. In the parent equation, the province matters for the majority of cases, reflecting the spatial heterogeneity in China. The base case is Guizhou and all other provinces have lower (and in most cases statistically significant) smoking effects than these. Marital status is not statistically significant in either equation. Similarly, individual income is unimportant, perhaps because the real cost of tobacco products in China is relatively low.

Lagged selfsmoking and lagged peersmoking measures the impact of the most recent past on current smoking. We must then calculate the full lagged selfsmoking and peersmoking effect by taking into account the interacted age gap coefficients: we sum the selfsmoking (peersmoking) coefficient with that of selfsmoking (peersmoking) interacted with the age gap dummies.[20] This will measure the full effect of the latest observed past event on current behaviour. Results are presented in Table 4. We find strong evidence of dynamic effects both through habits/addiction as well as through inter-generational peer effects. With respect to habits, not surprisingly, the larger the age gap between observations, the lower is the selfsmoking estimated coefficient. Habits exhibit a much stronger effect when observations are closer in time. Also, the habit effect of the child is much stronger than that of the parent.

	short run	medium run	long run
	Lagged effect	Interaction with age gap 2 to 4 years	Interaction with age gap >4 years
Habit (selfsmoking)			
Parent equation	0.81^{***}	$0.81^{***}-0.54^{*}=0.27$	$0.81^{***}-0.56^{*}=0.25$
Child equation	1.02^{***}	$1.02^{***}-0.47^{**}=0.55$	$1.02^{***}-0.63^{***}=0.39$
Peer effect (peersmoking)			
Parent equation	-0.53^{***}	$-0.53^{***}+0.23=-0.3$	$-0.53^{***}+0.52^{*}=-0.01$
Child equation	-0.56^{***}	$-0.56^{***}+0.28=-0.28$	$-0.56^{***}+0.16=-0.4$

Table 4: Estimates: habit and peer effects

The inter-generational peer effects are all negative. This suggests that the parent's (child's) currently observed smoking behaviour is negatively affected by the child's (parent's) previously observed smoking behaviour.[21] It is as if a parent (child) is more likely to

[20] Both for parent and child, the pure time effect (measured by the age gap dummies) is insignificant.

[21] A negative peer effect has been found in other circumstances. Angrist (2014) reports several instances in which positive peer effects are observed (positive association between a variable of interest for the individual and for the peers), but also identifies circumstances in which they were found to be negative: for example,

do today the opposite of what their child (parent) did in the previous period. Interestingly, in this case, the age gap points to different effects for parent and child: in the long run, for the parent, the child's past behaviour has less of a (negative) impact (coefficient is lower in absolute value), whilst for the child, the (negative) peersmoking (parent) coefficient remains relatively high in absolute value (note, however, that the interacted long run age gap coefficient is not statistically significant).

5.3 Post-estimation

5.3.1 Goodness of fit

A goodness of fit measure that could be used for our results is the percentage of correctly predicted observations for each of the four possible outcomes: 11, 10, 01 and 00. Such a measure is preferable to an overall percentage (across outcomes) of correctly predicted observations (Wooldridge, 2002, p. 465). Making use of the bivariate Normal distribution that we have assumed for the composite error term and the latent variable specification (equation (1)), we obtain the results presented in Table 9 in Appendix A.[22]

Overall, these percentages are consistently high, both across outcomes as well as across waves: for example, looking only at waves 2, 3 and 4 (with a larger number of observations), the percentage of correctly predicted observations varies between 62% and 88%.

5.3.2 Specification test

A link test is used to check whether the model specification is correct: the model is augmented by introducing in the regression powers of the predicted latent variable and then its statistical significance is tested.[23] Results are presented in Table 10 in Appendix A. We find that the square of the predictions are not statistically significant, suggesting a correct model specification.

for Dartmouth college freshmen and roommates, their GPA depends positively on own SAT scores, but negatively on the average SAT scores within a room dorm.

[22] In our setup, this calculation is not as straightforward as in standard probit or bivariate probit models. Indeed, our latent variable specification contains lagged values of our dependent variable. In order to proceed, we make use of the bivariate Normal composite error distribution, as well as the observed lagged values. For this reason, results can only be presented from wave 2 onwards.

[23] More concretely, the estimated coefficients are used to obtain the predicted values for the latent variable. The model is then re-estimated using as explanatory variables only the predicted values for the latent variable, as well as its square.

5.3.3 Model stability

We test model stability and robustness by randomly dividing the full sample into two halves and estimating the model separately on each subsample. We can then compare the coefficients estimated in the two subsamples with each other and with those estimated on the full sample. We performed a likelihood ratio test of the hypothesis that there is a single set of parameters over the full sample.[24] In essence, each observation (parent-child pair) has an equal chance of being chosen for a particular sample. The 854 parent-child pairs are therefore randomly allocated to one of the two samples. Results are presented in Tables 11 and 12 in Appendix A. The likelihood ratio test indicates that the full sample (less restricted) model performs no worse than the restricted models (random sample 1 and 2).[25]

First, it is reassuring to find that the most important results hold. In particular, habit and inter-generational peer effects appear with the same sign and similar magnitudes as in Table 3, although not all inter-generational peer effects are statistically significant. By applying a likelihood-ratio test we find that the restriction of a common set of parameters (including the covariance matrix parameters) cannot be rejected. In addition, a similar test was carried out to compare our model with a constant-only model. The latter is rejected at conventional significance levels.

6 Policy implications

Our model implies a dynamic process in the joint smoking behaviour of parents and their children. In particular, a careful understanding of this dynamic process provides insightful policy implications for our results. The estimated coefficients for each parent-child pair evaluated at the sample means are essentially constant over time. This implies that our estimated equations (based on latent variables) can be written as:

[24] The full sample model performs better than the intercept-only model. We regress both models and obtain the log-likelihoods (LL). The likelihood ratio test statistic is given by $LR = 2(LL^{full\ sample} - LL^{intercept\ only}) = 191.52$ and provides evidence against the intercept-only model.

[25] The likelihood ratio test statistic is given by $LR = 2(LL^{\text{random sample 1}} + LL^{\text{random sample 2}} - LL^{\text{full model}}) = 69.05$. The critical value is $\chi\,(p = 0.2, df = 63) = 72.2$ and the null hypothesis cannot be rejected.

$$\begin{cases} y_{p,t} = \widehat{a}_p + \widehat{ss}_p y_{p,t-1} + \widehat{ps}_p y_{c,t-1} \\ y_{c,t} = \widehat{a}_c + \widehat{ps}_c y_{p,t-1} + \widehat{ss}_c y_{c,t-1} \end{cases} \tag{3}$$

where t represents the time dimension, $\widehat{ss}_i$ ($\widehat{ps}_i$), $i \in \{p, c\}$, is the selfsmoking (peersmoking) coefficient and $\widehat{a}_i$ subsumes the remaining coefficients in equation (1) (namely the constant and the remaining regressors).

Differences between two periods are thus given by:

$$\Delta y_p = y_{p,t} - y_{p,t-1} = \widehat{a}_p + (\widehat{ss}_p - 1)\, y_{p,t-1} + \widehat{ps}_p y_{c,t-1} \tag{4}$$

$$\Delta y_c = y_{c,t} - y_{c,t-1} = \widehat{a}_c + \widehat{ps}_c y_{p,t-1} + (\widehat{ss}_c - 1)\, y_{c,t-1} \tag{5}$$

At a stationary state, $\Delta y_p = 0$ and $\Delta y_c = 0$, which implies that $y_{p,t} = y_{p,t-1} = y_p^*$ and $y_{c,t} = y_{c,t-1} = y_c^*$. Therefore, at a stationary state, $y_p^* = \frac{\widehat{a}_p(1-\widehat{ss}_c)+\widehat{ps}_p\widehat{a}_c}{(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c}$ and $y_c^* = \frac{\widehat{a}_c(1-\widehat{ss}_p)+\widehat{ps}_c\widehat{a}_p}{(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c}$.

From a policy perspective, let us presume that the ultimate goal is to reduce smoking prevalence, that is, to induce a change to $\left(y_p^{'}, y_c^{'}\right)$ where $y_p^{'} < y_p^*$ and $y_c^{'} < y_c^*$. We can consider two types of policy: permanent policies, where $\left(y_p^{'}, y_c^{'}\right)$ becomes the new stationary state, or one-off policies, that induce an instantaneous shift in the variables, which then evolve over time according to the underlying dynamics. From an economic standpoint, permanent policies are likely to be costlier than one-off policies, that is, if both achieve the same outcome, policymakers should clearly prefer the latter.

There are two mechanisms through which policies can operate: first, policies could affect the exogenous variables in equation (1), given by vector X. In doing so, this would then impact on the values of $\widehat{a}_i$, $i \in \{p, c\}$.[26] Second, policies could affect directly the selfsmoking coefficients. Anti-smoking campaigns, indoor smoking bans, tobacco taxes or promotion of nicotine addiction treatment are all likely to reduce cigarette addiction (i.e., the selfsmoking coefficient). In addition, policies can be targeted at parents, at children or both. For example, an anti-smoking advertising campaign can appeal directly to parents or to children, depending on the campaign characteristics (message conveyed, form through which it is

[26] Note that we leave aside policies that could impact on the peersmoking coefficients. In doing so, we implicitly presume that the 'within-household' behaviour between parents and children is unaffected by any of these policies.

conveyed, choice of advertising platforms, etc.). We explicitly assume that a policy targeted at parents (children) would only affect the parents' (children's) selfsmoking coefficient.

We can use the estimated coefficients in order to gain further insight into the smoking dynamics and its policy implications. Table 5 displays the partial derivatives of the stationary state $\left(y_p^*, y_c^*\right)$ with respect to $\widehat{a}_i$ and $\widehat{ss}_i$, $i \in \{p, c\}$, evaluated at the estimated long run coefficient values: at the sample means, our results point to $\widehat{a}_p \simeq 0.53$ and $\widehat{a}_c \simeq 0.17$; in addition, the long run coefficient values for selfsmoking and peersmoking (see Table 4) are $\widehat{ss}_p = 0.25$, $\widehat{ps}_p = -0.01$, $\widehat{ps}_c = -0.4$ and $\widehat{ss}_c = 0.39$. These coefficients point to a stationary state $\left(y_p^* = 0.715, y_c^* = -0.189\right)$ located in quadrant I of the phase diagram (see Figure 4 in Appendix A).

First, one-off policies will ultimately be unsuccessful in inducing lasting changes in smoking prevalence. Note that the system converges to the stationary state, that is, a deviation, in one period, from $\left(y_p^*, y_c^*\right)$, for any of the two variables is ensured to be eliminated over time as the system dynamics brings (y_p, y_c) back to $\left(y_p^*, y_c^*\right) = (0.715, -0.189)$.[27] Therefore, only permanent policies can induce lasting changes in smoking prevalence.

partial derivative of → with respect to ↓	y_p^*	y_c^*
$\widehat{a}_p$	$\frac{1-\widehat{ss}_c}{(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c} = 1.345$	$\frac{\widehat{ps}_c}{(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c} = -0.882$
$\widehat{a}_c$	$\frac{\widehat{ps}_p}{(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c} = -0.022$	$\frac{1-\widehat{ss}_p}{(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c} = 1.654$
$\widehat{ss}_p$	$\frac{(1-\widehat{ss}_c)\left(\widehat{a}_p+\widehat{a}_c\widehat{ps}_p-\widehat{a}_p\widehat{ss}_c\right)}{\left[(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c\right]^2} = 0.961$	$\frac{\widehat{ps}_c\left(\widehat{a}_p+\widehat{a}_c\widehat{ps}_p-\widehat{a}_p\widehat{ss}_c\right)}{\left[(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c\right]^2} = -0.63$
$\widehat{ss}_c$	$\frac{\widehat{ps}_p(\widehat{a}_c+\widehat{a}_p\widehat{ps}_c-\widehat{a}_c\widehat{ss}_p)}{\left[(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c\right]^2} = 0.004$	$\frac{(1-\widehat{ss}_p)(\widehat{a}_c+\widehat{a}_p\widehat{ps}_c-\widehat{a}_c\widehat{ss}_p)}{\left[(1-\widehat{ss}_p)(1-\widehat{ss}_c)-\widehat{ps}_p\widehat{ps}_c\right]^2} = -0.311$

Table 5: Partial derivatives of stationary state values evaluated with long run coefficients

Second, consider permanent policies that are likely to affect the selfsmoking coefficients. Consider, for instance, a workplace smoking ban that is permanent in nature (e.g., new legislation). This ban could be portrayed as a permanent policy targeting parents, which

[27] A good example of this is a temporary anti-smoking campaign, which could potentially reduce smoking prevalence, by inducing a change in the variables down and to the left of the stationary state. However, as Figure 4 in Appendix A shows, smoking prevalence would return to its original stationary state as time evolves.

could contribute towards a reduction in the parent's selfsmoking coefficient, $\widehat{ss}_p$.[28,29] Such a reduction in $\widehat{ss}_p$ would succeed in reducing the stationary state value for parents ($y_p^{'} < y_p^*$), but would increase that of children ($y_c^{'} > y_c^*$). The intuition is simple: a reduction in y_p has an opposite impact on children, through the (negative) peersmoking effect.[30]

By contrast, consider permanent policies targeted at children, such as smoking bans in schools or increases in the minimum legal age to purchase tobacco. Such policies could contribute towards reducing $\widehat{ss}_c$ but, because $\partial y_c^*/\widehat{ss}_c < 0$, would actually lead to an increase in the stationary state value for children ($y_c^{'} > y_c^*$), whilst reducing that of parents ($y_p^{'} < y_p^*$). However, the stationary state would still be located in quadrant I, with $y_c^{'} < 0$.[31] Therefore, from this simple exercise, and based on our results, we can conclude that permanent policies aimed at reducing individuals' nicotine addiction are more effective, in the long run, if targeted at children.

Consider now permanent policies that impact on the exogenous variables. For instance, policies that increase education levels or promote living in cities, of parents or of children, will reduce the value of $\widehat{a}_i$, $i \in \{p, c\}$ (negative estimated coefficients in Table 3). From the derivatives calculated in Table 5 we can see that targeting only parents or only children will not induce the desired change: reducing $\widehat{a}_i$ will result in a new stationary state $\left(y_p^{'}, y_c^{'}\right)$ where either $y_p^{'} < y_p^{*}$ and $y_c^{'} > y_c^*$ or $y_p^{'} > y_p^{*}$ and $y_c^{'} < y_c^*$. By contrast, a 'symmetric-in-impact' policy that targets both parents and children could induce an overall reduction in smoking prevalence: the total derivatives are $dy_p^* = 1.345d\widehat{a}_p - 0.022d\widehat{a}_c$ and $dy_c^* = -0.882d\widehat{a}_p + 1.654d\widehat{a}_c$ which implies that when $d\widehat{a}_p \simeq d\widehat{a}_c < 0$, both dy_p^* and dy_c^* are negative.[32]

[28] As outlined above, we explicitly assume that such a policy would not affect children's selfsmoking coefficient, $\widehat{ss}_c$, which we assume would remain constant.

[29] As mentioned earlier, there is contradictory empirical evidence in the literature regarding the effective impact of bans on smoking behaviour. We assume here that bans do have some impact (even if small).

[30] Although, for small reductions in $\widehat{ss}_p$, the stationary state would still be located in quadrant I (with $y_p^{'} > 0$ and $y_c^{'} < 0$), a larger reduction in $\widehat{ss}_p$ would actually shift the stationary state to quadrant II (with $y_p^{'} > 0$ and $y_c^{'} > 0$).

[31] A policy that targets both parents and children simultaneously (for example, a general indoor smoking ban), that is, a policy that simultaneously reduces $\widehat{ss}_p$ and $\widehat{ss}_c$, would yield a similar outcome: the stationary state value for parents would decrease ($y_p^{'} < y_p^*$), but that of children would increase ($y_c^{'} > y_c^*$).Using the partial derivatives, we can calculate the total derivatives: $dy_p^* = 0.961d\widehat{ss}_p + 0.004d\widehat{ss}_c$ and $dy_c^* = -0.63d\widehat{ss}_p - 0.311d\widehat{ss}_c$. From these expressions, when $d\widehat{ss}_p < 0$ and $d\widehat{ss}_c < 0$, dy_p^* is negative whilst dy_c^* is positive.

[32] We define a 'symmetric-in-impact' policy as one which induces similar changes, in absolute value, in $\widehat{a}_i$, $i \in \{p, c\}$.

These conclusions also hold if we were to use the 'medium run' (age gap 2 to 4 years) coefficient values for selfsmoking and peersmoking (see Table 4), as the phase diagram and stationary values are rather similar (see Figure 4 in Appendix A). By contrast, the 'short run' system dynamics differs somewhat from the long run case. When we consider the short run coefficient values for selfsmoking and peersmoking (see Table 4), we have $\widehat{ss}_p = 0.81$, $\widehat{ps}_p = -0.53$, $\widehat{ps}_c = -0.56$ and $\widehat{ss}_c = 1.02$. The stationary state is located in quadrant II (see Figure 4 in Appendix A), with $\left(y_p^*, y_c^*\right) = (0.34, 0.89)$. Table 6 displays the partial derivatives of the stationary state $\left(y_p^*, y_c^*\right)$ with respect to $\widehat{a}_i$ and $\widehat{ss}_i$, $i \in \{p, c\}$, evaluated at the estimated short run coefficient values.

partial derivative of → with respect to ↓	y_p^*	y_c^*
$\widehat{a}_p$	0.067	1.863
$\widehat{a}_c$	1.763	−0.632
$\widehat{ss}_p$	0.022	0.628
$\widehat{ss}_c$	1.564	−0.561

Table 6: Partial derivatives of stationary state values evaluated with short run coefficients

In this case, the system has a saddle point: therefore, one-off policies have the potential to shift the variables in any direction, but could only induce a lasting change (that is, a situation in which the variables do not return to their stationary values) in parents' or in children's smoking prevalence, but not both at the same time.[33] Permanent policies that induce small reductions in $\widehat{ss}_p$ would succeed in inducing an overall reduction in smoking prevalence, by reducing the stationary state value for parents ($y_p^{'} < y_p^*$) and children ($y_c^{'} < y_c^*$) (see partial derivatives in Table 6). However, the stationary state would still be located in quadrant II, with $y_p^{'} > 0$ and $y_c^{'} > 0$ (see Figure 4 in Appendix A). By contrast, a permanent policy targeted at children's would reduce $\widehat{ss}_c$, which in turn would lead to a new stationary state also still located in quadrant II, but where $y_p^{'} < y_p^{*}$ and $y_c^{'} > y_c^*$.[34] Policies aimed at

[33] This can be seen as one-off variable changes up and to the left of the stationary values, or down and to the right (in both cases, between the $\Delta y_p = 0$ and $\Delta y_c = 0$ lines - see Figure 4 in Appendix A). Other variable changes will, over time, converge back into the stationary values.

[34] A more significant reduction in $\widehat{ss}_c$ would have a drastic impact in the phase diagram: a reduction to a value below 1 would change the system dynamics entirely and render a phase diagram much closer to that in the long run (with a similar downward sloping 'deltayc=0' line). At best, a reduction in $\widehat{ss}_c$ to a value significantly below 1 could move the stationary state to quadrant I (exactly as in the long run case) or, if even higher, to quadrant IV.

permanently changing the exogenous variables for parents could also succeed in reducing overall smoking prevalence (see Table 6): a reduction in $\widehat{a}_p$ would induce a new stationary state where $y_p^{'} < y_p^*$ and $y_c^{'} < y_c^*$.

In a nutshell, based on our 'long run' estimates, (i) only permanent policies can succeed and (ii) permanent policies targeting children, through reductions in $\widehat{ss}_c$, are preferable, as they would reduce parents' smoking prevalence and increase that of children's, although in the new stationary state we would still have $y_c^{'} < 0$. By contrast, permanent policies impacting on the exogenous variables of both parents and children could reduce overall smoking prevalence (of parents and children). When we consider our 'short run' estimates, permanent policies targeting parents - either through reductions in $\widehat{ss}_p$ or reductions in $\widehat{a}_p$ - could succeed in reducing overall smoking prevalence. Alternatively, a one-off policy could succeed in reducing the smoking prevalence of parents or of children, but not both simultaneously.

7 Conclusion

This paper is an empirical analysis of smoking behaviour in China, with a particular focus on the interplay between family experience (inter-generational effects) and habit. We find that both are relevant in explaining smoking behaviour. In particular, we find a *negative model* effect that is, to the best of our knowledge, novel in the smoking literature: within a parent-child pair, the past behaviour of one member exerts a negative effect on the behaviour of the other. In addition, both habit and inter-generational effects are not constant over time. In particular, in the long run, parents are less affected by their children's behavior than the reverse. Naturally, these inter-generational effects have relevant policy implications: in line with Göhlmann et al. (2010), our results suggest that targeting parents with permanent smoking reduction policies is likely to produce better results (in terms of smoking prevalence reduction) than targeting children, but this is only true with our short run coefficient estimates and only if policies are permanent in nature. In the long run, the opposite is true: smoking reduction policies that target children are preferable.

Although certainly interesting on their own, our results raise several interesting and inter-related questions. For example, although we analyse, in a dynamic perspective, the

smoking status of each pair over time, we implicitly assume a 'symmetric' decision process, whereby starting to smoke (a change in the dependent variable from 0 to 1) is considered in the same way as stopping smoking (a change from 1 to 0). Previous literature points to these decisions as being different in nature, if for no other reason, because of the addictive nature of tobacco. Future research looking at inter-generational smoking effects could also go beyond our findings based on data from China. Clearly, the fact that women (both parent and child) rarely smoke is not a common feature in other countries and it would certainly be interesting to understand whether our results are observed elsewhere.

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A Appendix: additional figures and tables

A.1 Descriptive statistics

Figure 1: Map of China and identification of provincial-level administrative units covered in the data used

parent marriage status		
	number of obs.	percentage
not married	168	8%
married	2017	92%
total	2185	100%

child marriage status		
	number of obs.	percentage
not married	1258	58%
married	927	42%
total	2185	100%

Table 7: Parent-child marriage status

Figure 2: Age distribution and age distribution by smoking choice: parent (top) and child (bottom)

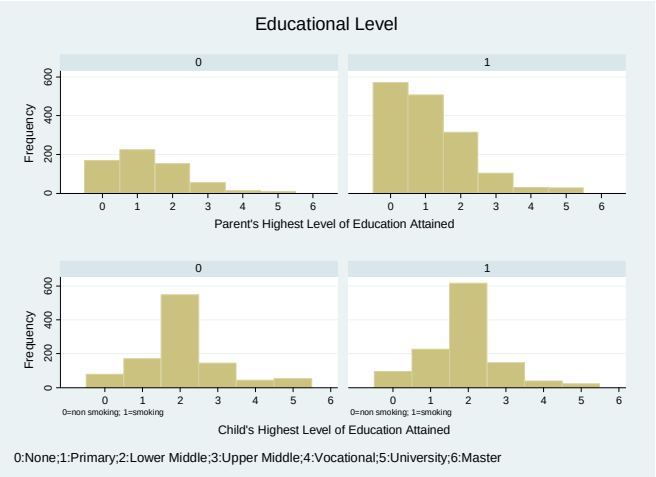

Figure 3: Educational attainment by smoking choice: parent (top) and child (bottom)

Variable	Description	Obs	Mean	Std.Dev.	Min	Max
Individual characteristics for Parent						
Smoking	= 1 if smoke	2185	.71	.45	0	1
Farmer	= 1 if farmer	2185	.62	.59	0	1
Working now	= 1 if work now	2185	.94	.23	0	1
Live at home	= 1 if live at home	2185	1	0	0	1
Income	Total individual nominal income (Business, Farming, fishing gardening, livestock, retirement and non-retirement wages)	2185	5006.185	7496.354	0	37900
Individual characteristics for Child						
Smoking	= 1 if smoke	2185	.52	.50	0	1
Farmer	= 1 if farmer	2185	.62	.59	0	1
In School	= 1 if in school	2185	.02	.15	0	1
Working now	= 1 if work now	2185	.99	.12	0	1
Live at home	= 1 if live at home	2185	.99	.03	0	1
Income	Total individual nominal income (Business, Farming, fishing gardening, livestock, retirement and non-retirement wages)	2185	5520.565	8031.146	0	37900
Community characteristics						
Live in city	= 1 if live in city	2185	.06	.24	0	1
Live in suburban area	= 1 if live in suburban area	2185	.17	.37	0	1
Live in county/town capital city	= 1 if live in county/town capital city	2185	.08	.27	0	1
Live in rural village	= 1 if live in rural village	2185	.69	.46	0	1

Table 8: Summary Statistics for Other Variables

A.2 Post-estimation

wave	n	11	10	01	00
2	854	62	73	84	68
3	303	66	78	86	67
4	120	72	73	88	68
5	38	58	87	100	61
6	13	46	100	92	54
7	2	50	100	100	50
1=Smoke; 0=Does not smoke					

Table 9: Percentages of observations that are correctly predicted

	Parent	**Child**
Initial wave		
yhat	1.12(1.58)	0.89***(5.25)
$yhat^2$	−0.09(-0.018)	−0.30(-1.15)
Cons	−0.03(-0.12)	0.03(0.52)
Remaining waves		
yhat	1.08***(3.59)	0.97***(8.48)
$yhat^2$	−0.05(-0.29)	0.09(0.66)
Cons	−0.02(-0.12)	−0.02(-0.32)
Sigma for parent	1.34*** (24.71)	
Sigma for child	0.81*** (28.38)	
Theta	0.83*** (15.30)	
Rho corr	0.65*** (6.19)	
Log likelihood	-2355.7147	
Number of observations	2185	
t statistics in parentheses	* p<0.1, ** p<0.05, *** p<0.01	

Table 10: Link test

	Parent	**Child**
Initial wave		
Age	−0.025*(-1.79)	0.26**(2.37)
Age^2		−0.004*(-1.77)
Married	0.91**(2.15)	0.14(0.68)
Education level	−0.12(-1.18)	−0.14(-1.58)
In school		−0.74(-1.10)
Living home		−1.68(-0.11)
Constant	1.65**(2.10)	−2.05(-0.14)
Remaining waves		
laggedSS(lagged selfsmoking)	**0.85*(1.89)**	**0.83***(3.08)**
laggedPS(lagged peersmoking)	**-0.46(-1.61)**	**-0.63**(-2.34)**
Agegap(2to4yr)	0.16(0.43)	−0.16(-0.55)
Agegap(>4yr)	0.76*(1.73)	0.22(0.65)
LaggedSS Agegap(2to4yr)	0.01(0.03)	−0.39(-1.25)
LaggedSS Agegap(>4yr)	−1.08**(-2.22)	−0.48(-1.38)
LaggedPS Agegap(2to4yr)	−0.20(-0.56)	0.73**(2.56)
LaggedPS Agegap(>4yr)	0.70(1.59)	0.21(0.65)
Province dummies	yes	yes
Age	−0.009(-0.70)	0.018(1.25)
Income (RMB)	0.0000172(1.23)	0.0000201**(2.11)
Married (yes/no)	0.40(1.27)	0.18(1.22)
Education level	−0.06(-0.57)	−0.20**(-2.43)
Working now (yes/no)	0.20(0.50)	
In school(yes/no)		−0.46(-0.66)
Farmer(yes/no)	0.47*(1.84)	0.17(0.99)
Live in city(yes/no)	0.44(0.87)	−0.23(-0.63)
Live in Suburban(yes/no)	−0.18(-0.63)	0.03(0.12)
County/town capital	0.05(0.13)	0.33(1.07)
Constant	0.60(0.53)	−0.088(-0.18)
Sigma for parent	1.38***(7.95)	
Sigma for child	0.84***(13.27)	
Theta	0.93***(8.97)	
Rho corr	0.62***(3.78)	
Log likelihood	-1110.88	
Number of observations	1061	
t statistics in parentheses	* p<0.10, ** p<0.05, *** p<0.01	

Table 11: Random sample 1 (Parent-child inter-generational smoking behaviour)

	Parent	**Child**
Initial wave		
Age	-0.018(-1.48)	0.30^{***}(2.70)
Age^2		-0.004^{**}(-2.06)
Married	0.94^{**}(2.48)	-0.20(-0.89)
Education level	-0.14^{*}(-1.17)	-0.09(-1.17)
In school		-1.03^{**}(-2.06)
Living home		-7.11(-0.45)
Constant	1.01(1.60)	2.96(0.19)
Remaining waves		
laggedSS(lagged selfsmoking)	**0.74^{*}(1.93)**	**0.81^{***}(2.62)**
laggedPS(lagged peersmoking)	**-0.63^{**}(-2.07)**	**-0.36(-1.28)**
Agegap(2to4yr)	0.30(0.85)	0.38(1.24)
Agegap(>4yr)	-0.27(-0.70)	0.34(0.94)
LaggedSS Agegap(2to4yr)	-1.08^{**}(-2.51)	-0.51(-1.48)
LaggedSS Agegap(>4yr)	-0.16(-0.36)	-0.80^{**}(-2.09)
LaggedPS Agegap(2to4yr)	0.64^{*}(1.80)	-0.13(-0.42)
LaggedPS Agegap(>4yr)	0.72^{*}(1.67)	0.08(0.20)
Province dummies	yes	yes
Age	-0.008(-0.61)	0.015(0.85)
Income (RMB)	-0.0000151(-1.34)	$-8.32e-06$(-0.81)
Married (yes/no)	0.22(0.60)	0.14(0.83)
Education level	-0.017(-0.17)	-0.24^{**}(-2.53)
Working now (yes/no)	0.87^{*}(1.92)	
In school(yes/no)		-0.63(-1.13)
Farmer(yes/no)	-0.09(-0.38)	-0.40^{**}(-1.97)
Live in city(yes/no)	-0.41(-0.90)	0.21(0.52)
Live in Sururban(yes/no)	0.08(0.27)	0.34(1.41)
County/town capital	-0.13(-0.32)	0.41(1.24)
Constant	0.99(0.88)	0.55(1.04)
Sigma for parent	1.51^{***}(9.22)	
Sigma for child	1.21^{***}(12.12)	
Theta	0.78^{***}(10.60)	
Rho corr	0.44^{***}(4.46)	
Log likelihood	-1211.3026	
Number of observations	1124	
t statistics in parentheses	* p<0.10, ** p<0.05, *** p<0.01	

Table 12: Random sample 2 (Parent-child inter-generational smoking behaviour)

A.3 Phase diagram and system dynamics

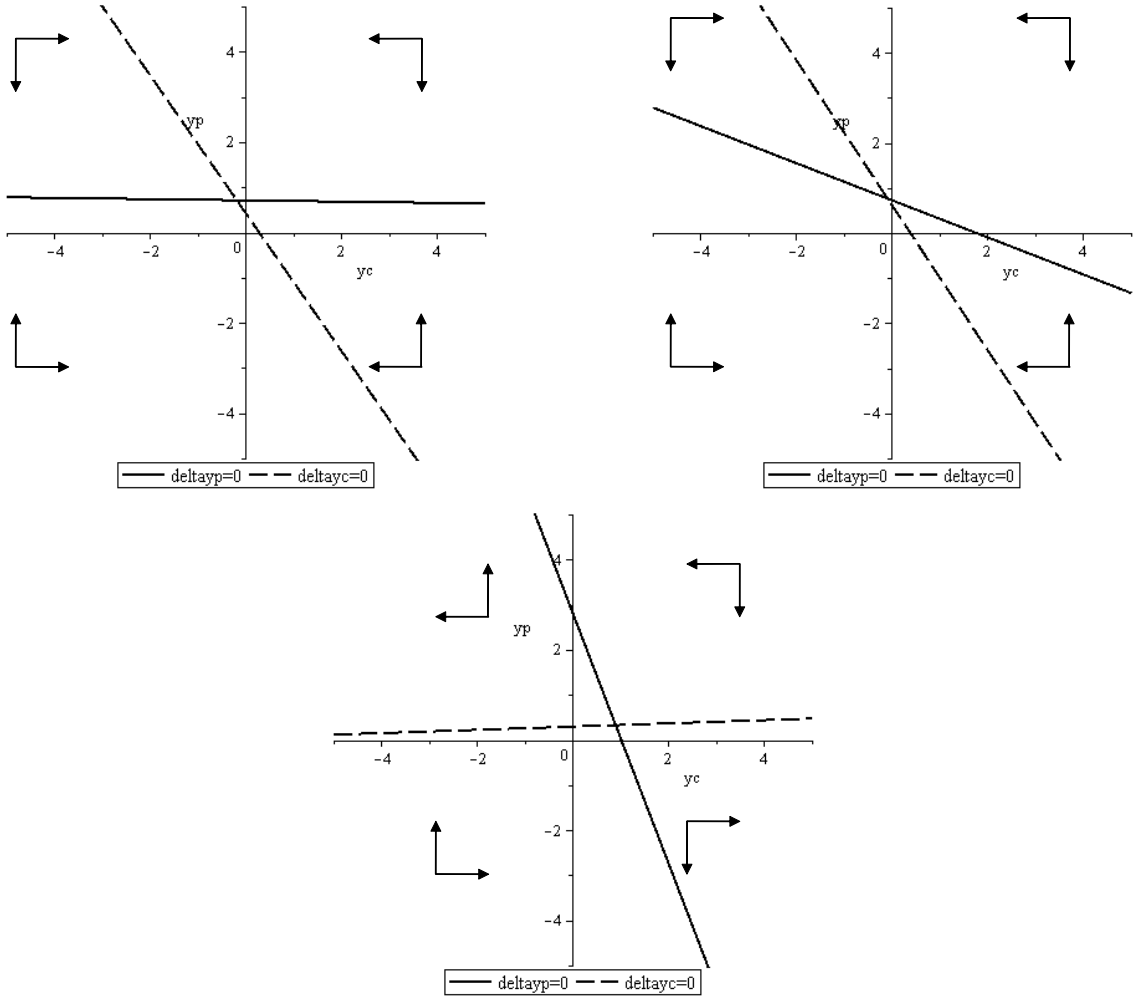

Figure 4: Phase diagrams: long run (top left), medium run (top right) and short run coefficients (bottom)

B Appendix: detailed description of the econometric model

B.1 The initial condition

In order to estimate the model described in section 4.1, let us first consider the initial condition problem. This problem arises because the start of the observation period in the sample data may not coincide with the start of the stochastic process generating individuals' smok-

ing behaviour. The sample data used in the estimation can refer to periods that are later than the first smoking experience. Therefore, the first observation in the sample can be caused by an earlier history of smoking behaviour (state dependence) or by some observed exogenous variables and/or unobserved individual characteristics. To account for this problem, we follow Heckman (1981a, 1981b), Arulampalam (2000), Stewart (2007) and Plum (2013, 2014). The equation for the initial observation (period 1) is specified in the following way:

$$Y_{j,i,1} = \begin{cases} 1, \text{ if } y^*_{j,i,1} = \lambda_j Z_{j,i} + \theta_j a_{j,i} + u_{j,i,1} & \geq 0 \\ 0, & otherwise \end{cases} \tag{6}$$

where $Z_{j,i}$ is a vector of strictly exogenous variables at any individual, family and community level, including the variables relevant in period 1; coefficients $\theta = (\theta_p, \theta_c) \in \mathbb{R}^2$ stand for free parameters (factor loadings) that allow any type of correlation between $Y_{p,i,1}$, $Y_{c,i,1}$, $Y_{p,i,t}$ and $Y_{c,i,t}$; to simplify the estimation, we set $\theta_p = \theta_c$ (this restriction can be easily relaxed).

The composite error for period 1 is

$$v_{j,i,1} = \theta_j a_{j,i} + u_{j,i,1} \tag{7}$$

The covariance of the composite error term between the initial period and the subsequent periods is $cov(v_{j,i,1}, v_{j,i,t}) = \theta_j \sigma_j^2$. The idiosyncratic error ($u_{j,i,1}$) is uncorrelated with the random effect ($a_{j,1}$), and with the idiosyncratic shock ($u_{j,i,s}$, $s = 2, ..., T$) at other times.

B.2 The covariance matrix

To estimate the complete model for period 1 and the subsequent periods, we estimate the initial condition equation and the subsequent equations as a system. This allows us to model the correlation of the composite error term between the initial and subsequent periods. The covariance matrix includes 4 blocks:

block 1: covariance matrix for p	block 2: covariance matrix between p and c
block 3: covariance matrix between c and p	block 4: covariance matrix for c

Using the unbalanced panel, the size ($2T_i$ by $2T_i$) of the covariance matrix varies with the maximum number of the waves that each parent-child pair is present in the survey. For

those present in all 8 waves, the covariance matrix of the full sample of composite errors is written as

$$\Omega^*_{8\times 8} = \begin{array}{c|cccc|cccc} & v_{p,i,1} & v_{p,i,2} & \cdots & v_{p,i,8} & v_{c,i,1} & v_{c,i,2} & \cdots & v_{c,i,8} \\ \hline v_{p,i,1} & \theta^2\sigma_p^2+1 & & & & \rho\theta^2\sigma_p\sigma_c & & & \\ v_{p,i,2} & \theta\sigma_p^2 & \sigma_p^2+1 & & & \rho\theta\sigma_p\sigma_c & \rho\sigma_p\sigma_c & & \\ \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots \\ v_{p,i,8} & \theta\sigma_p^2 & \sigma_p^2 & \cdots & \sigma_p^2+1 & \rho\theta\sigma_p\sigma_c & \rho\sigma_p\sigma_c & \cdots & \rho\sigma_p\sigma_c \\ \hline v_{ci,1} & \rho\theta^2\sigma_p\sigma_c & & & & \theta^2\sigma_c^2+1 & & & \\ v_{c,i,2} & \rho\theta\sigma_p\sigma_c & \rho\sigma_p\sigma_c & & & \theta\sigma_c^2 & \sigma_c^2+1 & & \\ \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots \\ v_{c,i,8} & \rho\theta\sigma_p\sigma_c & \rho\sigma_p\sigma_c & \cdots & \rho\sigma_p\sigma_c & \theta\sigma_c^2 & \sigma_c^2 & \cdots & \sigma_c^2+1 \end{array}$$

where Ω^*_8 is the covariance matrix for 8 waves.

B.3 The log likelihood

The joint probability of the complete model for person p and c living in household i over time is

$$\begin{array}{cc} \prod_{t=2}^{T_i}\Phi\left[\left(2Y_{p,i,t}-1\right)\left(\beta_p X'_{p,i,t}+\alpha_p Y_{p,i,t-1}+\gamma_p Y_{p,i,t-1}\right)\right] & \Phi\left[\left(2Y_{p,i,1}-1\right)\left(\lambda_p Z_{p,i,1}\right)\right] \\ \Phi\left[\left(2Y_{c,i,t}-1\right)\left(\beta_c X'_{c,i,t}+\alpha_c Y_{c,i,t-1}+\gamma_c Y_{c,i,t-1}\right)\right] & \Phi\left[\left(2Y_{c,i,1}-1\right)\left(\lambda_c Z_{c,i,1}\right)\right] \end{array}$$

The complete model is easily estimated by noticing that the distribution of $y^*_{j,i,t}$ conditional on the random effect (a) is bivariate normal. The likelihood can be written as

$$\begin{aligned} L \;=\; & \prod_{i=1}^{N}[\int_{-\infty}^{(2Y_{p,i,1}-1)(\lambda_p Z_{p,i,1})}][\int_{-\infty}^{(2p_{c,i,1}-1)(\lambda_c Z_{c,i,1})}]... \\ & [\int_{-\infty}^{(2Y_{p,i,t}-1)(\beta_p X'_{p,i,t}+\alpha_p Y_{p,i,t-1}+\gamma_p Y_{c,i,t-1})}][\int_{-\infty}^{(2Y_{c,i,t}-1)(\beta_c X'_{c,i,t}+\alpha_c Y_{c,i,t-1}+\gamma_c Y_{p,i,t-1})}]... \\ & [\int_{-\infty}^{(2Y_{p,i,T}-1)(\beta_p X'_{p,i,T}+\alpha_p Y_{p,i,T-1}+\gamma_p Y_{c,i,T-1})}][\int_{-\infty}^{(2Y_{c,i,T}-1)(\beta_c X'_{c,i,T}+\alpha_c Y_{c,i,T-1}+\gamma_c Y_{p,i,T-1})}] \\ & g(v|\Omega^*_{2t})dv_{c,i,T}dv_{p,i,T}...dv_{c,i,t}dv_{p,i,t}...dv_{c,i,1}dv_{p,i,1} \end{aligned} \tag{8}$$

where Ω^*_{2T} is the covariance matrix and where $g(.)$ is the multivariate normal density; $T = 1, ..., 8$. The log-likelihoood is simply $log(L)$.