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Extending decomposition analysis to account for socioeconomic background: income-related smoking inequality among Swedish women

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Abstract: This article suggests an extension of the standard decomposition of the concentration index that allows for an exploration of the pathways through which socioeconomic background relates to income-related health inequality. This novel approach is contrasted to the standard one using a panel of Swedish women in Statistics Sweden's Survey of Living Conditions for one vital health-related behavior, smoking. The decomposition uses an underlying model that considers both individual heterogeneity and smoking persistence, showing that the largest contributions to the pro-rich smoking inequality come from years of schooling and living in a single household. The contribution from socioeconomic background is close to negligible when using the standard approach. Once applying the suggested extension, socioeconomic background contributes indirectly to the inequality, primarily through an increase in years of schooling. These results highlight the potential importance of using this extension, especially for distinguishing between circumstances that individuals may affect themselves and these that are out of their control, which may be important for policy design.

Keywords: Health inequality; concentration index; decomposition; smoking; socioeconomic background.

JEL: I14; I12

I. INTRODUCTION

The last decades have seen considerable improvements in life expectancy and average health status in Europe, but socioeconomic inequalities in health have, despite egalitarian public health policy, been persistent in most countries (e.g., Kunst et al. 2004, 2005; Marmot et al. 2012). In Sweden, recent studies even find increasing inequality in mortality, morbidity, and health status (e.g., Burström et al. 2005; Fors et al. 2008; Shkolnikov et al. 2012). To understand health inequalities, it may be important to study the distributions of the underlying risk factors such as smoking, obesity, and alcohol use. In contrast to other risk factors, smoking is unambiguously an important health determinant. In fact, smoking is responsible for as much as 30% of the cancer deaths in the developed world and accounts for a substantial proportion of vascular- and respiratory-disease deaths (Vineis et al. 2004; Peto et al. 2006). As today's smoking behavior affects future health status, the relationship between smoking and socioeconomic status (SE-status) today may be a snapshot of future health inequalities.

Although the hazards of smoking are well known, individuals still engage in and initiate smoking. Smoking behavior also varies with SE-status (e.g., Kenkel 1991; Yen and Jones 1996; de Walque 2007; Kjellsson et al. 2011), which may affect smoking decisions through several channels. There may be differences in time preferences[1] or in reactions to health-risk information (e.g., Grossman 1972; Adda and Lechene 2011). Moreover, SE-status may be associated with higher access to resources such as (human) capital or social networks, which facilitates quitting an addictive habit (e.g., Hodgson 1997; Lindbladh and Lyttkens 2002; Kjellsson et al. 2011). The degree of peer acceptance or opportunity cost of smoking may also vary depending on the socioeconomic group one belongs to (e.g., Clark and Etilé 2002). Traits or intergenerational household factors that encourage human capital investments, or are

[1] Individuals from higher socioeconomic groups may have incentives to stay out of smoking to reap the returns of their higher human, or health, capital in the future.

important to success in life, may also be related to self-control – which in turn may be directly affected by education (e.g., Viscusi and Hersch 2001).

Exposing oneself to risky behavior may be considered a personal concern and should therefore not be discussed in terms of health inequality. Several reasons, however, may invalidate such an argument. The myopic and addictive dimensions of smoking may raise questions of internal time inconsistencies, or intrapersonal externalities, as there are long-term consequences of current smoking behavior (cf. Gruber and Köszegi 2001, Laux 2000).[2] The close link between smoking initiation and adolescence implies that individuals have to face the consequences of the myopic decisions taken as adolescents much later in life. In an inequality-of-opportunity context, most of us would further consider the socioeconomic background (SE-background) of individuals as circumstances rather than efforts (e.g., Dworkin 1981; Roemer 1998, 2002; Rosa Dias 2009; Trannoy et al. 2010). This perspective provides a clear rationale for studying how SE-background affects the smoking distribution.

The question of how childhood conditions and SE-status of the parents may affect health status later in life has also received increased attention in the literature (e.g., Marmot et al. 2001; Case et al. 2005; Currie 2009; Trannoy et al. 2010; Halleröd and Gustafsson 2011; Tubeuf et al. 2012;). One potential path is through participation in such risky health behavior as smoking (cf. Rosa Dias 2009; Francesconi et al. 2010; Göhlmann et al. 2010; Balia and Jones 2011). The mechanisms suggested above affecting the correlation between SE-status and smoking may also carry over to the next generation, so that parental SE-status directly affects the offspring's smoking behavior. Alternatively, SE-background may indirectly affect smoking behavior through other paths such as education or income. The actual smoking

[2] If smoking behavior may be modeled in accordance with the rational-addiction model suggested by Becker and Murphy (1988), there should be less concern about inequality.

behavior may also persist over generations; children of smokers are more likely to initiate smoking themselves (e.g., Keyes et al. 2008; Göhlmann et al. 2010; Balia and Jones 2011).

In this article, I improve standard decomposition techniques to explore the role of SE-background in income-related smoking inequality (IRSI). The contribution is both empirical and methodological. I first measure long-run IRSI in Sweden between 1988 and 2006 within an ageing female population using a rank-dependent index. Secondly, I contrast the standard decomposition with a novel approach (in this setting) that allows for an exploration of the pathways through which SE-background may affect the health variable – here, smoking – both directly and indirectly. The results also highlight the advantage and importance of using this approach.

I follow a Swedish cohort of females born between 1920 and 1961 over three consecutive waves from 1988/89/90 to 2004/05/06[3] in Statistics Sweden's Survey of Living Standards (ULF). The sample is restricted to females since the pervasive moist-snuff use among Swedish men complicates an analysis based on a joint sample. Swedish women initiate smoking at younger ages than their European counterparts (Oh et al. 2010).[4] As female smoking is a more mature phenomenon in Sweden than in other European countries, studying IRSI in a Swedish context is also relevant for research and policy outside of Sweden.[5] To examine whether smoking inequality within the study period is in favor of the rich or the poor, I compute a rank-dependent index of absolute IRSI suggested by Erreygers (2009a). To illustrate the development over time, I use a range of complementary indices. As smoking behavior is persistent and has life-long consequences, taking a long-term perspective is arguably appropriate. Using a time-stable measure of long-term income as the socioeconomic

[3] I observe four consecutive waves, but the first of the four waves is used in the dynamic specification of the underlying model. Therefore, I do not compute any inequality indices for this wave.

[4] Note that, the establishment of female smoking in Sweden was comparably early, and, in contrast to other European countries, smoking prevalence is higher among Swedish females than among Swedish males (Oh et al . 2000). The participation rate is comparably low and has been decreasing over the last 30 years.

[5] For applications in different contexts see Harper and Lynch (2007) for US and Bacigalupe et al. (2013) for the Basque Country.

ranking variable also ensures that changes in inequality stem from changes in smoking behavior rather than reranking of individuals.[6]

When decomposing the level of absolute IRSI during this period, I extend the standard decomposition techniques with a more realistic model of smoking behavior to address the problem of unobserved heterogeneity and to allow for both direct and indirect contributions of SE-background. A drawback of the standard decomposition analysis is that the underlying multivariate regression usually estimates the contribution of one covariate conditional on all other covariates without considering whether the covariate may mediate or confound. For example, if the effects of SE-background or education are mediated through income, the contribution of these covariates will be underestimated if income is included in the model. I address this drawback and extend the previous literature by adapting the Tubeuf et al. (2012) approach for decomposition of a univariate inequality index to a bivariate rank-dependent index. The approach, which may be used for any binary health variable, considers the pathways through which childhood conditions or schooling may affect smoking directly and indirectly. In an inequality-of-opportunity context, this may also help distinguish between circumstances and effort.

Exploiting the longitudinal dimension of the data, I use an underlying model that, in contrast to the standard decomposition, reduces unobserved heterogeneity and considers the role of smoking persistence. In short, I estimate a static and dynamic specification of a random effect probit (RE-probit). While the dynamic specification – which solves the initial-condition problem by estimating the model conditional on the first observed smoking status (cf., Wooldridge 2005) – considers the persistence of smoking, it may conceal a part of the long run relationship between SE-background and smoking. Using a static *and* a dynamic specification as complements, therefore, deepens the insights of how SE-background affects

[6] For discussion of the problems related to re-ranking of individuals, see Brekke and Kverndokk (2012)

IRSI in both long and short run. Both specifications are estimated conditional on the intraindividual mean of the time-variant variables to reduce the problem of unobserved heterogeneity (cf. Mundlak 1978). If unobserved traits such as self-control, risk aversion, and patience are still correlated with both smoking and such independent variables as education and income, these variables' contributions may be overestimated; I elaborate on this issue in the discussion.[7] From an inequality perspective, however, it may be less of a problem if these traits are correlated with the intergenerational household's characteristics that the SE-background variable may capture. As one cannot choose one's parents, most of us would consider SE-background as a circumstance – in contrast to effort – regardless of whether it affects smoking behavior through genetics or the environment.

The results show that absolute IRSI is pro-rich and the decomposition analysis using a conventional model further indicates that schooling and living in a single-adult household are the main contributors to IRSI (alongside the long-term income measure) – regardless if the underlying model accounts for individual heterogeneity and smoking persistence. The decomposition also shows that IRSI is largely driven by individual heterogeneity and the persistence of smoking behavior. The direct contribution of SE-background is small in a static specification and close to absent when accounting for smoking persistence in a dynamic specification – unless the pathways through which SE-background may affect smoking participation are considered. When allowing for an indirect contribution, the total contribution of the father's SE-status increases considerably, indicating that, while SE-background may not have a large direct contribution to IRSI, it affects the smoking distribution indirectly through an increase in years of schooling. That is, by showing the contrast to standard decomposition techniques, the results stress the importance of considering the indirect

[7] As the identifying assumption is still selection on observables, causality should be interpreted with care and the study should be interpreted as an explanatory study of the driving factors of IRSI. Although I do not claim to establish a true causal effect of a socioeconomic variable on smoking behavior – as is common in the inequality literature – I do believe that I reduce the unobserved heterogeneity.

pathways of childhood conditions and SE-background when decomposing income-related health inequality.

The article proceeds as follows. Section 2 discusses inequality measurement and presents the standard decomposition techniques, before elaborating on how to extend the underlying model and developing the decomposition to consider the SE-background pathways. Section 3 discusses data and how to measure income and SE-background. Section 4 presents the results, while Section 5 concludes and considers policy implications.

II. METHODS

II.A Measuring Inequalities

The standard health-economic tool for measuring income- or socioeconomic-related health inequalities is a rank-dependent index, such as the concentration index. Advantages of the concentration index (and its alternative versions) are that it considers the total population rather than the extremes of the distribution, and the intuitive, graphical interpretation in relation to the concentration curve. For a given population of n individuals, each denoted i and with an attached socioeconomic rank R_i, the concentration curve graphs the cumulative fraction of a health variable Y concentrated in the cumulative fraction of the population (cf. Kakwani et al. 1997). The concentration index (C, hereafter) equals twice the difference between the area below the line of equality (the 45° line) and above the concentration curve and the area above the line of equality and below the concentration curve. If the concentration curve is above the line of equality, Y is more concentrated among the poor and the index attains a negative value (and vice versa). As the health variable, smoking, is a bad, I refer to such a distribution as pro-rich. For C of nonsmoking, $C(1-Y)$, then negative (positive) values are referred to as pro-poor (pro-rich). C is a relative index as it is invariant to proportional changes of Y. An absolute counterpart denoted V (invariant to uniform changes) is based on

the absolute concentration curve, which plots the cumulative fraction of the population against the cumulative amount of Y in terms of the mean. While C attains values between −1 and 1, V is generally unbounded (but attains values between 0.25 and -0.25 for binary variables).

The binary nature of smoking prevalence complicates matters. To preserve the meaning of the relative and absolute vertical value judgments, let us consider the distributional change of the prevalence over income quantiles. That is, an absolute index remains constant when the prevalence changes uniformly over the quantiles, while a relative index, such as C, remains constant when the prevalence changes proportionally over the quantiles. Whereas the measured degree of absolute inequality is the same whether one codes smoking as zero or one, the measured degree of relative inequality is not (Clarke et al. 2002; Erreygers and van Ourti 2011a). The normative and technical implications of binary variables have been intensely debated in the literature and several versions of the concentration index has been suggested – all included in the family of rank-dependent indices (cf. Wagstaff 2005, 2009, 2011a,b; Erreygers 2009a,b; Erreygers and van Ourti 2011a,b; Kjellsson and Gerdtham 2013a,b; Allanson and Petrie 2013a, 2013b). Formally, I express any rank-dependent index as a normalized weighted sum of smoking levels:

$$I(Y) = f(\mu, n) \sum_{i=1}^{n} r_i Y_i, \qquad (1)$$

where $r_i = \frac{n+1}{2} - R_i$ and the normalization function $f(\mu, n) > 0$. Thus, the direction of inequality is the same for any rank-dependent index: For the main analysis, the choice of index is of semantic importance only. I focus on absolute inequalities using the index suggested by Erreygers (2009a): For a binary variable,

$$E(Y) = \frac{8}{n^2} \sum_{i=1}^{n} r_i Y_i. \qquad (2)$$

The choice of index matters when comparing populations with different means. For the illustration of the development over time, I therefore complement the absolute index with a range of indices bounded by the two relative indices, *C* with respect to smoking and nonsmoking:

$$C(Y) = \frac{2}{n^2\mu}\sum_{i=1}^{n} r_i Y_i, \quad (3)$$

$$C(1-Y) = \frac{2}{n^2(1-\mu)}\sum_{i=1}^{n} r_i (1-Y_i). \quad (4)$$

The recent literature suggests these two are the polar cases of the range of indices that may be considered to represent intuitive or defensible vertical value judgments (cf. Allanson and Petrie 2013a, 2013b; Kjellsson and Gerdtham 2013b). a normalization of *C* for binary variables suggested by Wagstaff (2005)[8], denoted as

$$W(Y) = \frac{2}{n^2\mu(1-\mu)}\sum_{i=1}^{n} r_i Y_i, \quad (5)$$

W intermediate the two relative indices and is identical (with opposing signs) for smoking and nonsmoking.

II.B Decomposition of Inequalities

Wagstaff et al. (2003) propose a way to explain the inequalities in period *t* by decomposing a rank-dependent index using an underlying linear regression of the health variable, Y_t, on *J* explanatory variables, X_{jt},

$$Y_{it} = \alpha_t + \sum_{j=1}^{J} \beta_{jt} X_{jit} + \varepsilon_{it}, \quad (6)$$

[8]Wagstaff (2005) suggests normalizing the concentration index of the most unequal society possible given the smoking prevalence: *C* can only reach its maximum value if there is only one (non)smoker in the population. Wagstaff's index further equals the sum the indices capturing the relative vertical value judgments.

where α_t and β_{jt} are the coefficients and ε_{it} is the error term in a given period t. If defining the absolute concentration index of a variable X_j as

$$V_{jt}(X) = \frac{2}{n^2}\sum_{i=1}^{n} r_i X_{jit}, \tag{7}$$

one may, given the relationship in Eq(6), express any rank-dependent index in period *t* as

$$I_t(Y) = \sum_{j=1}^{J}\left(\frac{\beta_{jt}V_{jt}}{g(\mu_t)}\right) + \frac{V_t^{\varepsilon}}{g(\mu_t)}, \tag{8}$$

where V_{jt} and V_t^{ε} are the absolute concentration index of the j^{th} explanatory variable, X_{jit}, and the error term, respectively. $g(\mu_t) > 0$ and relates to the normalization function of any rank-dependent index as $f(\mu_t, n) = 2/n^2 g(\mu_t)$. Eq(8) suggests that the contribution of each covariate equals the product of the coefficient from the underlying model, β_{jt}, and the absolute concentration index V_{jt} weighted by a function of the mean of smoking participation, $g(\mu_t)$. Although the actual value of the contributions of the variables varies depending on the chosen index, the contribution of each covariate as a percentage of the total index is equal for all rank-dependent indices.[9] Therefore, I will only decompose $E(Y)$ – by setting $g(\mu_t)$ = ¼ in Eq(8).

As the scope of the paper is to measure and decompose long-run IRSI during the study period, I denote the mean of Y over all *i* and *t* as $\mu_{\cdot}$ and use a panel-data model that restricts the time-invariant coefficients, $\beta_{jt} = \beta_j$, as the underlying model (compare Wildman 2003). Computing the rank-dependent index as

$$I(Y) = f(\mu_{\cdot}, nt)\sum_{t=1}^{T}\sum_{i=1}^{n} r_i Y_{it} \tag{9}$$

[9]Expressing a rank-dependent index as $I_t = V_t/g(\mu_t)$, where $V_t = \frac{2}{n^2}\sum_{i=1}^{n} r_i Y_{it}$, illustrates that $(\beta_{jt}V_{jt}/g(\mu_t))/(V_t/g(\mu_t)) = (\beta_{jt}V_{jt})/V_t$ for any $g(\mu_t)$. For the concentration index, the explained part of Eq(9) is generally referred to as a weighted sum of the concentration indices of the explanatory variables weighted by the elasticities of Y_t with respect to X_{jt}: $C_t = \sum_{k=1}^{K}\left(\beta_{jt}\mu_{jt}^{x}/\mu_t\right) C_{jt} + V_t^{\varepsilon}/\mu_t$, where μ_{jt}^{x} is the mean of X_{jt}.

is essentially equivalent to both treating all observations independently and using an intraindividual average of smoking participation. The same applies to the decomposition of $E(Y)$, which then may be expressed as

$$E(Y) = 4\sum_{j=1}^{J}\left(\beta_j\left(\frac{2}{(nt)^2}\right)\sum_{t=1}^{T}\sum_{i=1}^{n} r_i x_{it}\right) + 4V^{\varepsilon}$$

$$= 4\sum_{j=1}^{J}\beta_j V_j + 4V^{\varepsilon} \qquad (10)$$

II.C The Underlying Regression

The decomposition of the rank-dependent inequality index was originally developed for a cross-sectional linear additive model (Wagstaff et al. 2003). For a binary outcome such as smoking, van Doorslaer et al. (2004) suggest approximating the β-coefficients from the linear regression in Eq(6) by the partial effects evaluated at the mean from a binary choice model.[10] To estimate the smoking-participation decision, I use, in line with their suggestion, a binary choice model where the underlying latent variable equals

$$Y_{it}^{*} = \boldsymbol{X_{it}}\boldsymbol{\lambda} + u_i + e_{it}, \qquad (11)$$

and vector $\boldsymbol{X_{it}}$ contains all independent variables. e_{it} and u_i represent a time-variant error term and the individual time-invariant unobserved effect, respectively. The latent variable in Eq(11) relates to the actual smoking behavior as $Y_{it} = 1$ if $Y_{it}^{*} \geq 0$ and $Y_{it} = 0$ if $Y_{it}^{*} < 0$. By assuming that e_{it} and u_i are independent and normally distributed (with variance denoted σ_e^2 and σ_u^2), a RE-probit – denoting the standard normal CDF (PDF) as $\boldsymbol{\Phi}$ (ϕ) – may be used to examine the probability of smoking participation in a given period:

$$\Pr(Y_{it} = 1|\boldsymbol{X_{it}}) = \boldsymbol{\Phi}(\boldsymbol{X_{it}}\boldsymbol{\lambda}). \qquad (12)$$

[10]The partial effects at the mean, compared to the average partial effect, require less-restrictive assumptions on the representability of the sample distribution of the covariates.

However, the assumption of independence between u_i and e_{it} is very restrictive and unlikely to hold. The specification also ignores the addictive dimension of smoking. To better account for unobserved heterogeneity, I use a Mundlak-type specification to parameterize the individual effect using the within-individual mean of the time-variant variables, $\bar{\boldsymbol{X}}_{i.}$ (cf. Mundlak 1978; Contoyannis et al. 2004). That is, specifying the latent variable, Y_{it}^*, as

$$Y_{it}^* = \boldsymbol{X_{it}}\boldsymbol{\lambda} + \bar{\boldsymbol{X}}_{\boldsymbol{i}}\boldsymbol{\rho} + u_i + e_{it}, \tag{13}$$

where the vector $\bar{\boldsymbol{X}}_{\boldsymbol{i}}$ contains the Mundlak variables. To also account for the role of smoking persistence in IRSI, I complement the static specification in Eq(13) with a dynamic model using a state-dependency approach established in the literature (cf. Gilleskie and Strumpf 2005; Kjellsson et al. 2011). I estimate a dynamic RE-model that allows individuals to be locked into an unwanted smoking behavior caused by addiction. In contrast to rational-addiction models, this model does not include a lead of consumption.[11] The introduction of the lagged dependent variable Y_{it-1} to capture state dependency is likely to reduce unobserved heterogeneity, but consistency requires that the initial smoking condition Y_{i0} is independent of u_i. To handle this problem, I use a convenient solution suggested by Wooldridge (2005),[12] estimating the model conditional on the initial smoking status Y_{i0}. The latent smoking variable, Y_{it}^*, is now

$$Y_{it}^* = \delta Y_{it-1} + \boldsymbol{X_{it}}\boldsymbol{\lambda} + \bar{\boldsymbol{X}}_{\boldsymbol{i}}\boldsymbol{\rho} + \omega Y_{i0} + u_i + e_{it}. \tag{14}$$

[11]While the rational-addiction theory, despite of its flaws (cf. Suranovic et al. 1999; Wangen 2004; Gruber and Köszegi 2001), may be appealing, the econometric application of the theory is at least problematic; antithesis tests have shown milk and eggs to be addictive goods (Auld and Grootendorst 2004), and, as Gilleskie and Strumpf (2005) point out, observed future value is not enough to apply the assumption of perfect foresight.

[12]There are alternative estimators suggested by Heckman (1981) and Orme (2001). Arulampalam and Stewart (2009) show that none of these estimators are unambiguously preferred above the others, but conclude that using a Mundlak-type specification is advantageous. There are also fixed-effects estimators available, but the FE-model suggested by Honoré and Kyriazidou (2000) is very data demanding and allows for neither computing marginal effects nor including binary independent variables such as time dummies.

The assumption of independence between the remaining individual time-invariant unobserved effect u_i and a normally distributed time-variant error term e_{it} is more likely to hold after the inclusion of $\bar{\boldsymbol{X}}_{\boldsymbol{i}}$, Y_{i0}, and Y_{it-1}.

In the decomposition analysis, I use the partial effects of the RE-probit. Denoting the coefficients from the RE-probit rescaled by $(1+\hat{\sigma}_u^2)^{-0.5}$ as $\tilde{\delta}, \tilde{\boldsymbol{\lambda}}, \tilde{\boldsymbol{\rho}}$ and $\tilde{\omega}$, the partial effect of X_j, which are evaluated at the mean, is

$$\frac{\delta(\Pr(Y_{it}=1|\boldsymbol{X_{it}},\bar{\boldsymbol{X}}_{\boldsymbol{i}},Y_{i0}Y_{it-1})}{\delta X_{jt}} = \phi\left(\tilde{\delta}Y_{it-1} + \boldsymbol{X_{it}}\tilde{\boldsymbol{\lambda}} + \bar{\boldsymbol{X}}_{\boldsymbol{i}}\tilde{\boldsymbol{\rho}} + \tilde{\omega}Y_{i0}\right) * \tilde{\lambda}_j \qquad (15)$$

(cf. Wooldridge 2010). For binary variables, the partial effect is computed as $\Pr\left(Y_{it}=1\middle|\boldsymbol{X_{it}},\bar{\boldsymbol{X}}_i,Y_{i0},Y_{it-1},X_{jit}=1\right) - \Pr\left(Y_{it}=1\middle|\boldsymbol{X_{it}},\bar{\boldsymbol{X}}_i,Y_{i0},Y_{it-1},X_{jit}=0\right)$. For categorical variables, such as the child dummies, the other dummies within the category are set to zero.

II.D Using a Full Model Allowing for Intermediate Effects

In line with the previous literature, the model in Eq(14) estimates the contribution of one covariate conditional on all other covariates without considering whether one of them may act as a mediator or a confounder. For example, if a covariate, such as childhood conditions or schooling, is mediated through income, the contribution of that covariate is underestimated. To design policy in line with an inequality-of-opportunity viewpoint, it is important to not underestimate the contribution of covariates related to circumstances an individual cannot affect by her own effort (cf. Roemer 1998; Rosa Dias 2009; Trannoy et al. 2010). By replacing the standard model with a full nonlinear pathway model developed by Karlson et al. (2012) and Karlson and Holm (2011) and already applied in the health inequality context by Tubeuf et al. (2012), I can identify the total contribution of SE-background, differentiate between the direct and indirect contributions, and further explore the mediating role of

schooling in smoking decisions. For notational ease, I exchange the vectors $\boldsymbol{X}_{it}$ and $\bar{\boldsymbol{X}}_i$ for single variables of SE-background B_i, demographic D_i, schooling S_i, and a mediator, Z_{it}, and its intraindividual mean, $\bar{Z}_i$. Using this notation, the latent smoking variable in Eq(14) is now

$$Y_{it}^* = \psi B_i + \vartheta D_i + \tau S_i + \lambda Z_{it} + \rho \bar{Z}_i + \delta Y_{it-1} + \omega Y_{i0} + u_i + e_{it}. \quad (16)$$

We may suspect that the effects of SE-background and demographic variables are mediated through schooling, S_i, and other variables (e.g., income) denoted as Z_{it}. There may also be an additional layer of mediating effects; the effect of education may be mediated through income. I, therefore, complement the analysis with a pathway model that identifies these two layers of mediating effects and distinguishes between the direct and indirect effect of SE-background B_i, demographic D_i, and schooling S_i. The model is estimated in two steps starting with a set of auxiliary regressions that captures the effect of the demographic variables and SE-background on schooling

$$S_i = \varphi_1^S B_i + \varphi_2^S D_i + s_{it} \quad (17)$$

and the effect of demographic variables, SE-background, and schooling on the remaining time-varying variables and their within-individual mean:

$$Z_{it} = \varphi_1^Z B_i + \varphi_2^Z D_i + \varphi_3^Z S_i + z_{it} \quad (18)$$

$$\bar{Z}_i = \varphi_1^{\bar{Z}} B_i + \varphi_2^{\bar{Z}} D_i + \varphi_3^{\bar{Z}} S_i + \bar{z}_i. \quad (19)$$

By substituting in Eq(17), Eq(18), and Eq(19) for S_i, Z_{it}, and $\bar{Z}_i$ in Eq(16), the latent smoking variable is now

$$Y_{it}^* = \psi B_i + \vartheta D_i + \tau(\varphi_1^S B_i + \varphi_2^S D_i + s_{it}) + \lambda(\varphi_1^Z B_i + \varphi_2^Z D_i + \varphi_3^Z S_i + z_{it})$$

$$+\rho(\varphi_1^{\bar{Z}} B_i + \varphi_2^{\bar{Z}} D_i + \varphi_3^{\bar{Z}} S_i + \bar{z}_i) + \delta Y_{it-1} + \omega Y_{i0} + u_i + e_{it} \quad (20)$$

Defining $\Psi^B = (\varphi_1^s\tau + \varphi_1^z\lambda + \varphi_1^{\bar{z}}\rho)$, $\Psi^D = (\varphi_2^s\tau + \varphi_2^z\lambda + \varphi_2^{\bar{z}}\rho)$, and $\Psi^S = (\varphi_3^z\lambda + \varphi_3^{\bar{z}}\rho)$, rearranging Eq(20) into

$$Y_{it}^* = (\psi + \Psi^B)B_i + (\vartheta + \Psi^D)D_i + \Psi^S S_i + \tau s_i + \lambda z_{it}$$

$$+\rho\bar{z}_i + \delta Y_{it-1} + \omega Y_{i0} + u_i + e_{it}, \qquad (21)$$

and again substituting in Eq(17) makes it possible to distinguish between the direct and indirect effects of SE-background, demographics, and schooling on the latent smoking variable (cf. Karlson and Holm 2011; Karlson et al. 2012; Tubeuf et al. 2012):

$$Y_{it}^* = (\psi + \Psi^B + \Psi^S\varphi_1^s)B_i + (\vartheta + \Psi^D + \Psi^S\varphi_2^s)D_i + (\Psi^S + \tau)s_i$$

$$+\lambda z_{it} + \rho\bar{z}_i + \delta Y_{it-1} + \omega Y_{i0} + u_i + e_{it}. \qquad (22)$$

Here, Ψ^B, Ψ^D, and Ψ^S denote the first layer indirect effect – i.e., the effect of SE-background, demographics, and schooling, respectively – mediated through Z_{it}, $\bar{Z}_i$, and schooling. $\Psi^S\varphi_1^s$ and $\Psi^S\varphi_2^s$ denote the second layer mediated through education and, in turn, Z_{it} and $\bar{Z}_i$. In practice, Eq(19) is estimated by replacing s_i, z_{it} and $\bar{z}_i$ by the estimated residuals of the auxiliary regressions $\hat{s}_i$, $\hat{z}_{it}$, and $\hat{\bar{z}}_i$ in Eq(17)–Eq(19).

Analogous to the decomposition of the univariate inequality of the latent variable y_{it}^* suggested by Tubeuf et al. (2012), it is possible to decompose a bivariate rank-dependent index using the specification of the latent variable as the underlying regression (cf. van Doorslaer and Jones 2003). Using Eq (16) as the underlying variable, the decomposition of $E(Y^*)$ is specified as

$$E(Y^*) = 4\left(\vartheta V_D + \varphi V_B + \tau V_S + \lambda V_Z + \rho V_{\bar{Z}} + \delta V_{y_{t-1}} + \omega V_{y_0} + V^{\varepsilon}\right).$$

(23)

To compare the contribution of the each covariate with and without considering the indirect effect, compare Eq(23) with the decomposition using Eq (22) as the underlying model with the total coefficient divided into the direct effect and the indirect effect as

$$E(Y^*) = 4(\vartheta V_D + (\Psi^D + \Psi^S \varphi_2^S) V_D + \psi V_B + (\Psi^B + \Psi^S \varphi_1^S) V_B +$$

$$\tau V_s + \Psi^S V_s + \lambda V_z + \rho V_{\bar{z}} + \delta V_{y_{t-1}} + \omega V_{y_0} + V^{\varepsilon}), \qquad (24)$$

where V_s, V_z, and $V_{\bar{z}}$, denote the absolute rank-dependent index with respect to the residual of the auxiliary regressions of S_i, Z_i, and $\bar{Z}_i$ respectively.

In contrast to Tubeuf et al. (2012), who interpret Y_{it}^* as an indicator of the latent health of the individual, I am interested in the actual distribution of Y_{it} (in relation to social position). Therefore, I compute the partial effects (at the mean), in analogy to Eq(10), using the specification in Eq(19).[13] To compare the contribution to the long-run IRSI of the covariates using a specification considering the indirect effects to a specification not considering the indirect effects, I compute the contribution of variable j – i.e., $4\beta_j V_j$– using the relevant terms from the specification in Eq(22) as substitutes for the counterparts in Eq(10). That is, I let the new partial effects replace β_j. To obtain the net contribution of the covariates Z_{it} and $\bar{Z}_i$ when accounting for the indirect effects of the SE-background, demographics, and schooling, I substitute V_j of Z_{it} and $\bar{Z}_i$ for the counterpart of the residuals of the auxiliary regressions in Eq(18) and Eq(19), z_{it} and $\bar{z}_i$ – i.e., substituting V_z and $V_{\bar{z}}$ for V_Z and $V_{\bar{Z}}$ as in the example of the latent variable in Eq(24). By comparing the contributions using the two specifications, it is possible to distinguish between the indirect and direct contributions.

III. DATA

III.A Survey of Living Conditions

[13]Karlson et al. (2012) illustrate how to use partial effects in a nonlinear pathway model.

The ULF 1980–2006 is a yearly cross-sectional survey including rotating panels where a subset of the respondents is interviewed every 8th year (Statistics Sweden 2010). I use twelve ULF cross sections to construct a panel of four waves (1980/81/82, 1988/89/90, 1996/97/98, and 2004/05/06). ULF is further linked to Swedish registry data on income and education from the Longitudinal Integration Database for Health Insurance and Labour Market Studies (LISA) 1990–2006. I use a balanced panel of 1,631 women born between 1924 and 1963 – i.e., the age groups 27–64 in 1988/89/90 and 43–80 in 2004/05/06.[14] When performing tests for attrition suggested by Verbeek and Nijman (1992), I cannot reject the null of no attrition (See appendix).

III.B Measuring SE-Status

The rank-dependent index requires ordering the population after a socioeconomic variable. While income is most often a continuous and finely distributed variable, occupational and educational measures may be unordered or distributed with considerable amount of ties. It is, therefore, also standard in the literature to measure income-related inequality. I use a measure of *disposable income* (income from capital, employment and transfers net of taxes) aggregated to the household level, converted to a common scale by the consumer price index and weighted by the OECD equivalence scale.[15] As the scope of the paper is studying long-run IRSI, I use the individual mean of the disposable income from LISA from 1990 to 2006 to obtain a robust and constant measure of SE-status. (This income measure also ensures that IRSI changes are due to changes in smoking status rather than income mobility.[16]) However, as individuals' productivity and income varies over the lifecycle, such a measure of long-term

[14]Figure A1 in the appendix presents an overview of the results for an unbalanced panel. These are similar to those of a balanced panel.

[15]The OECD equivalence scale weights children as 0.3 and adults (including children older than 18) as 0.5. Note that the main results are robust for not using the weight of children in the equivalence scale, although the inequality index increases slightly.

[16]Thus, we avoid a problem of bivariate inequality measures pointed out by Brekke and Kverndokk (2012).

income is still sensitive to the individuals' age during the observed period. Therefore, I age-standardize the long-term income analogously to the indirect standardization of a health variable (O'Donnell et al. 2008) using a Mincer (1974)-inspired equation. I first regress the logarithm of long-term income M_i^{long} on a vector of independent variables,[17] $\boldsymbol{w_i}$, and a higher-order polynomial of the average age of the individual during the observed years:

$$M_i^{long} = \boldsymbol{w_i}\boldsymbol{\pi} + \widehat{\kappa_1} age_i + \widehat{\kappa_2} age_i^2 + \widehat{\kappa_3} age_i^3 + \widehat{\kappa_4} age_i^4 + v_i \qquad (25)$$

A higher order polynomial of the average age allows for a less restrictive functional form of the age effect. Then, I use the parameters from this regression to obtain an age-expected income measure using the individual value of the age variables and the sample mean of the other variables, $\bar{\boldsymbol{w}}$.:

$$\widehat{M}_i^{longAGE} = \bar{\boldsymbol{w}}.\widehat{\boldsymbol{\pi}} + \widehat{\kappa_1} age_i + \widehat{\kappa_2} age_i^2 + \widehat{\kappa_3} age_i^3 + \widehat{\kappa_4} age_i^4. \qquad (26)$$

Then, I compute the standardized long-term income as the difference between the actual long-term income and the age-predicted long-term income plus the overall sample mean of the long term income, $\bar{M}_{\cdot}^{\text{long}}$.:[18]

$$M_i^{stand} = M_i^{long} - \widehat{M}_i^{longAGE} + \bar{M}_{\cdot}^{long}. \qquad (27)$$

Thus, I compute a measure of lifetime or long-term disposable income conditioned on a higher-order polynomial function of the individual average age during these years.

III.C Variables

Table 1a presents the definitions of all variables used in the analysis.[19]

[17] SE-background, level of education, immigrant status, and the individual mean (during the years we observe the individual in LISA) of both the number of children in the household (squared) and dummies indicating living in a single-adult household and living in a (large) city. Table A1b in the appendix reports the results of the regression.

[18] Eq(27) may be reduced to $M_i^{\text{stand}} = M_i^{\text{long}} - (\widehat{\kappa_1} age_i + \widehat{\kappa_2} age_i^2 + \widehat{\kappa_3} age_i^3 + \widehat{\kappa_4} age_i^4) + (\hat{\kappa}_1 \overline{age} + \hat{\kappa}_2 \overline{age^2} + \hat{\kappa}_3 \overline{age^3} + \hat{\kappa}_4 \overline{age^4})$

1. Daily Smoking. The dependent variable, Y_{it}, is an indicator of being a daily smoker in period *t*. To model the state dependency, the lag of the binary smoking variable, Y_{it-1}, is included in the model. The model is also estimated conditional on the individual's smoking status in 80/81/82, Y_{i0}.

2. Disposable Income. As information on current income is not available for all waves in LISA,[20] I use the ULF income information for current income in the underlying regression.[21] However, instead of including the individual mean of current income in the underlying regression as suggested in Section 4.3, I include the standardized measure of long-term income used as a ranking variable. The interpretation of the two income variables is best understood as how current income, which is plausibly fluctuating, relates to the smoking decisions conditional on the long-term income (and vice versa). The long-term income measure will, however, also capture a large degree of the unobserved heterogeneity that the Mundlak approach attempts to address. Based on the model's goodness of fit guided by appropriate information criteria (i.e., AIC, BIC), I use the logarithm of income.[22]

3. Demographics, Socioeconomic Background, Schooling and Household Variables. The SE-background is measured using categorical dummies indicating whether the father was a blue-collar worker (omitted), a farmer (*fath_farm*) or a white-collar worker of lower (*fath_white_low*) or higher grades (*fath_white_high*).[23] Schooling enters the model as a time-invariant variable measuring years of schooling.[24] Moreover, I include indicators of the

[19]The underlying regression includes such socioeconomic variables as education, household variables, and parental socioeconomic status, but due to potential endogeneity problems we exclude any additional lifestyle factors.

[20]As LISA ranges from 1990 to 2006, it includes no income information for 1988 and 1989.

[21]Both variables are based on register data, but the household definition differs between the two datasets.

[22]As the logarithm of zero is undefined, one krona is added to all observations of household disposable income.

[23]*fath_white_low* includes white-collar workers in lower and medium positions. *fath_white_high* includes white-collar workers in high or leading positions as well as academics and entrepreneurs. Note, that for these cohorts, the father's SE-status is probably a better indicator of the childhood conditions than the mother's.

[24]We treat the years of schooling as fixed at the entry into the panel (at least aged 29). Including a time-variant schooling variable would identify a local effect of (nonrepresentative) individuals who increase their level of education later in life.

household structure; a dummy for living in a single-adult household, *alone*, and two dummies indicating the number of children in the household (*childd2*, 1 child; *childd3*, two or more children). As further demographic variables, I include a dummy for being a first-generation immigrant (*im2*) and variables controlling for age[25] and birth cohort. The model is also estimated conditional on wave-specific effects and the intraindividual mean of the time-varying variables. Note, however, that I consider the individual mean of age as another indicator of the birth cohort and therefore exclude it from the model.

[About here: Table 1a-b about here]

IV. RESULTS

The following section reports the results of the analysis, first presenting the level of IRSI as measured by the rank-dependent indices. To give a broader view, I present both the total level and the development over the waves. The following sections present the results of the decomposition using the standard technique (Section IV.B) and the full pathway model (Section 4.C).

IV.A Measuring IRSI

1. Level of Inequality. Table 2 shows that, regardless of the chosen index, the total IRSI during the study period is pro-rich. So is also the wave-specific inequality for each of the three waves. Figure 1 graphs the smoking concentration curve for the whole period and for each wave separately; they are all above the 45° line of equality for (nearly) the whole distribution. Consequently, $C(Y)$, $E(Y)$, and $W(Y)$ are all negative while $C(1-Y)$ is positive. Also, all indices are significantly different from zero (see Table 2). The picture of smoking

Such an effect is however found to be positive (although insignificant). Results are robust to using the maximum years of schooling obtained during the study period.

[25]Using polynomials of age does not change the conclusions. However, age becomes more important at the expense of the cohort dummies.

being less common higher up in the income distribution is confirmed by simply graphing the smoking prevalence over income quintiles in Figure 2. However, the two graphs also exhibit that the proportion of smokers is lower in the very bottom than slightly higher in the income distribution.

[About here: Table 2 about here]

[About here: Figure 2 & Figure 3 about here]

2. Changes in Inequality over Time. The smoking prevalence within these cohorts has declined steadily from 29% to 23% to 18% during the three waves. The decrease in prevalence between the first two waves translates into a general increase in IRSI, irrespective of the index – although the change in the inequality indices is significant at conventional levels for $C(Y)$ and $W(Y)$ only. How the decrease in prevalence between the last two waves translates into changes in IRSI differs between the indices; although IRSI decreases according to all indices, this decrease is largest for absolute inequality (i.e., E) and relative inequality in nonsmoking (i.e., $C[1 - Y]$) and smallest for relative inequality in smoking $C(Y)$ and $W(Y)$. Thus, the conclusion of how the decrease in prevalence over the whole period translates into changes in IRSI also varies between the indices: IRSI increases according to $C(Y)$ and $W(Y)$, but remains at the same level (or slightly below) as in 88/89/90 according to $E(Y)$ and $C(1 - Y)$ as the reduction between the later waves offset the increase between the first two. A reasonable interpretation of this pattern is that smokers from the higher percentiles quit smoking earlier during their life course, while the smokers from the lower percentiles quit later on. During the years from 96/97/98 to 04/05/06, the decrease in the percentiles is close to proportional, implying that the absolute decrease is larger within the lower percentiles and the increase in the proportion of nonsmokers is skewed to the poor. This interpretation is also supported by Figure 2, which graphs the smoking prevalence over income quintiles.

VI.B Decomposing IRSI

This section presents the results from the static and dynamic underlying model and decomposition using the standard decomposition approach, whereas Section 4.3 presents the results from the pathway specifications illustrating the implications of allowing for both direct and indirect effects of SE-background. The sections follow the order of the methodological section, first presenting the results from the underlying models before discussing the contributions of the variables to the total inequality.

1 Results of the Underlying Model. Table 3 reports results for the static and dynamic RE-probits corresponding to Eq(11), Eq(13), and Eq(14). As the size of the coefficients is only identified to a scale dependent on σ_u^2, partial effects evaluated at the mean accompany the coefficients for comparisons between models. Paired bootstrap clustering on the individual level renders statistical inference possible. (Throughout, I use a conventional level of 0.05 for statistical significance.) The whole process, including the computation of the standardized long-term income, is performed in each bootstrap sample.

The partial effects of the three SE-background dummies, which indicate the difference with respect to the reference group of blue-collar fathers, are all negative and the size of all three is considerably lower (in absolute terms) when estimated conditional on Y_{it-1} and Y_{i0} in the dynamic specification than in the static specification. Coefficients and partial effects of both *fath_farm* and *fath_white_high* are also significant in the static models, but not in the dynamic specification. The partial effect of years of schooling decreases by one percentage point (from 2.6% to 1.5%), but remains comparatively large and statistically significant. The living-alone indicator is statistically significant in all models, but the partial effect is larger in the dynamic specification (and in the static specification that does not address the unobserved heterogeneity). Further note that the coefficients and partial effects of current income, *lninc*,

are insignificant throughout, but change sign when the measure of long-term income, *ln_LIFEinc*, is included. In turn, the partial effect of *ln_LIFEinc* is somewhat larger (although insignificant) in the dynamic model.[26] The coefficients and partial effects of both Y_{it-1} and Y_{i0} are of considerable size and highly significant.

[About here: Table 3 about here]

2 Decomposition Analysis of Dynamic and Static Model. The partial effects from Table 3 are further used in the decomposition of the absolute IRSI. Table 4 reports these results. Column 1 presents the absolute concentration index for each of the independent variables, while Columns 2–7 present the contribution to $E(Y)$ in levels and percentage of the total index for the three specifications (as in Table 3).

In general, the results of the three specifications are similar but the magnitude of the contributions is smaller in the dynamic model, which condition the contributions on previous smoking behavior. This is specifically the case for the contribution of SE-background. For example, having a white-collar father of high grade contributes 8% in the static and 3% in the dynamic specification. (To anticipate the results from section 4.3, the contribution of the SE-background increases when considering the indirect contribution channeled through schooling.)

Years of schooling and living in a single-adult household are the covariates with the largest contribution (alongside the socioeconomic ranking variable). The magnitude of the percentagewise contribution of years of schooling is 67 and 71% in the two static specifications, and is still substantial (39%), although considerably lower, in the dynamic specification. The contribution is driven by the fact that the well-educated have higher long-

[26] The coefficient, and contribution of *ln_LIFEinc*, which is included in the underlying model as a Mundlak variable, shall be interpreted with care as there may be reverse causal relationship between smoking and (future) income. sd

term income (the high V_j) and that they are less likely to smoke. Although the contribution is of considerable magnitude even when controlling for individual heterogeneity, care must be taken when interpreting the causality of the schooling effect as schooling may correlate with the unobserved heterogeneity. Living alone also contributes to inequality, but not to the same extent as schooling. Women living in single households are more prone to smoke and financially worse off than women living with a partner. (Because the living standard measure is on a household level and men tend to earn more than women, the negative correlation between living alone and long-term income is expected and, thus, V_j for *alone* and *m_alone* are both negative.) Given that the intraindividual mean of living in a single-adult household, *m_alone*, captures the unobserved characteristics of women who tend to live alone, the contribution of *alone* reflects the effect of living in a single household rather than potential related characteristics. Further, *m_alone* contributes considerably to IRSI in the static specification, but not at all in the dynamic model where the association between characteristics and smoking is captured by the lagged and initial smoking variable.

The lagged smoking variable contributes 10% of the total index under the dynamic specification, indicating that IRSI persists in part due to smoking persistence of smoking. The initial smoking condition makes an even larger contribution – potentially capturing a large part of the unobserved heterogeneity. Although the level of inequality is smaller in 80/81/82 than in the later waves, the contribution is large since being a smoker in this period is a strong predictor of being a smoker in later waves. Due to the residual term in Eq(8) and Eq(10), the contributions of the specific variables do not sum to the inequality index. This term represents the part of inequalities that are still unexplained after calculating the contribution of the independent variables and equals 0.1% in the dynamic and 21% and 14% in the static specifications.

4.3 Allowing for Intermediating Effects

1. Underlying Model. The results discussed in the previous sections do not consider the two layers of mediating effects described in Section 2.3: schooling, SE-background, and demographic variables (the latter two partly channeled through schooling) may affect smoking behavior indirectly by affecting income and household variables that in turn affect smoking behavior. Table 5a reports the results for the dynamic mediating specification in Eq(22), and Table 5b reports results for the static version. The statistical tests of the coefficients in Table 5a-b, as well as the results of the decomposition in Table 6a-b, are based on a bootstrapped procedure including all the steps from the auxiliary regressions to the decomposition exercise. All estimations of the auxiliary regressions are included in Table A3 in the appendix.

Column 1 presents the direct effects that correspond to the coefficients of the dynamic and static RE-probits in Table 3, Column 2 reports the coefficients corresponding to the total effects in the mediating specification, while Column 3 reports the total indirect effects, which amounts to the difference between the coefficients in Columns 2 and 1. Consider the SE-background variables, which also constitute the major differences between the models: $\Psi^B + \Psi^S \varphi_1^s$ denotes the total indirect effect. The coefficient of *fath_white_high* in Column 2 is significantly different from zero and more than double the size of its counterpart in Column 1, while the coefficient of *fath_white_low* is large but insignificant in the dynamic specification (but significant in the static specification in Table 5b). The total indirect effect, $\Psi^B + \Psi^S \varphi_1^s$ (the difference between Columns 1 and 2), is significant, large, and similarly sized for both *fath_white_low* and *fath_white_high*. The remainders of the coefficients are of similar size as in the standard model, except for the elimination of the negative age coefficient.

Columns 4 and 5 divide the first part of the total indirect effect of each covariate into the indirect effect from schooling and indirect effect from Z_{it} and $\bar{Z}_i$, denoted as $\Psi^B = \tau\varphi_1^s + (\varphi_3^z\lambda + \varphi_3^{\bar{z}}\rho)$. Column 6 reports the second-layer indirect effects mediated through education and, in turn, Z_{it} and $\bar{Z}_i$, denoted as $\Psi^S\varphi_1^z = (\varphi_3^z\lambda + \varphi_3^{\bar{z}}\rho)\varphi_1^z$. The results show that it is the indirect effect via schooling $\tau\varphi_1^s$ that, above all, drives the increase of the coefficients of *fath_white_high* and *fath_white_low*. That is, having a white-color father increases the level of education which in turn affects smoking.

[About here: Table 5a Dynamic RE-probit: Total and indirect effects (dynamic)]

[About here: Table 5b: Static RE-probit: Total and indirect effects (static)]

2 Decomposition Analysis of Dynamic and Static Pathway Models. Tables 6a-b report the decomposition of the full pathway model using a dynamic (a) and a static (b) specification. Columns 1 to 3 present the partial effects, contributions to *E*(*Y*) in levels and percentages; Columns 4 to 6 present the differences in partial effects and contributions compared to the standard specification (not accounting for the indirect contribution). The differences in the percentagewise contributions are also illustrated for both dynamic and static specifications in Figure 3, summarizing the contributions of the categorical dummies per category. Figure 3 illustrates that the contribution of SE-background increases from 2% to 8% in the dynamic specification and from 9% to 15% in the static specification. Table 6a further reports that both the contribution and the change in comparison to the specification without the mediating effect (Table 4) of *fath_white_high* are statistically significant, whereas only the latter is significant for *fath_white_low*. In the static specification in Table 6b, the contribution of *fath_white_low* is also significant. The contribution increase is driven by the increase of the partial effects of *fath_white_high* and *fath_white_low*. The results previously presented Table

5 illustrate that this increase is due to a higher level of education among women with a white-collar father.[27]

The pattern in Figure 3 and the results in Table 6a (and b) also support the hypothesis that the increased contribution of SE-background is mediated through an increase in years of schooling (and life-time income). The contribution of *yrsschool* (four percentage points) and *ln_LIFEinc* (three percentage points) decrease, while the contribution of *alone* increases slightly – these changes are, however, all insignificant. Figure 3b further illustrates that among the demographic variables the contribution of age and cohort decreases. Also, the partial effects and contribution of *fath_farm* (the father being a farmer) are statistically significant and of considerable size in the static model and do not change at all when allowing for the indirect pathways (in neither the dynamic nor the static specification). Thus, *fath_farm* relates to smoking by decreased initiation rates before the observed time span rather than indirectly through increased education.

In general, the large contribution of Y_{i0} may conceal a part of SE-background's (long-run) relation to smoking if SE-background affects the initial smoking condition, Y_{i0}. For that reason, Figure 3a (and Tables 5b and 6b) reports the contribution of each set of covariates using the static specification. Although the overall pattern of the results is similar, the contributions of the covariates and the differences between the full and standard models are generally larger in the static model. The difference between the two graphs in Figure 3 further suggests that the small direct relationship between SE-background and smoking is primarily captured by the smoking status in the initial period, and persists due to smoking persistence. A standard probit estimating the relationship between Y_{i0} and $\boldsymbol{X_{i0}}$ (presented in appendix Table A4) confirms a considerable association between SE-background and the initial

[27] The results of the auxiliary regressions in Table A5 in the appendix also show that the coefficients of *fath_white_high* and *fath_white_low* are of large magnitude in the regression of *yrsschool* indicating that SE-background influences smoking participation through the increased education.

smoking condition[28] – even when including the Mundlak variables. Although the indirect contribution of SE-background is slightly larger in the static specification, the graph still suggests that a higher level of education among the women with a white-collar father relates negatively to the smoking behavior even when accounting for the persistence and the initial smoking condition.

[About here: Figure 3. Contributions to total inequality]

3. Sensitivity Analysis. Because the relationship between SE-background and smoking may differ between cohorts, the decomposition exercise is preferably performed within a specific cohort (compare Tubeuf et al. 2012). As this application includes cohorts from a comparably long period, I perform sensitivity analysis using three subsamples: I perform the estimations splitting the sample into older and younger samples, 1924–1944 and 1945–1963, as well as including only women from the middle of the period (1935–1954). Figure A2 in appendix shows graphs analogous to Figure 3 for three subsamples. The pattern for the samples with women born 1935–1954 and 1945–1963 resembles the pattern in Figure 3, while the pattern for the sample born 1924–1944 differs slightly. The indirect contribution of schooling increases, rather than decreases. In general, the contribution of education and SE-background is larger for a younger sample while the contribution of the long-term income measure is larger for an older sample.

V. DISCUSSION AND POLICY IMPLICATIONS

Using a measure of long-term income as a time-stable indicator of SE-status, I find IRSI to be pro-rich. The decomposition analysis offers useful insights to explain the pro-rich IRSI during the period. The largest contributions to the level of inequality (alongside the long-term income measure) come from schooling and living in a single-adult household. The

[28] The association is even stronger if applying a pathway model similar to that of the main results.

contribution from SE-background is small in the static and even negligible in the dynamic specification without considering the pathways through which the father's SE-status may affect smoking participation. Although SE-background does not contribute directly to the inequality index, there is a considerable indirect contribution as the father's SE-status affects smoking participation through an increase in the years of schooling. Note, however, that my empirical strategy does not allow me to discriminate between the effect of the parents' low SE-status per see and any related unobserved variable or intergenerational traits such as self-control or norms of how smoking and education are regarded. Nor can I give any answer to whether the contribution is a result of nature or nurture. Nevertheless, as a child cannot choose its parents, this is not a necessary distinction in an inequality context. These are all circumstances that the individual are unable to affect by effort alone.

The absence of a direct contribution of SE-background in the dynamic model together with the minor contribution in the static model indicates that a possible direct effect of SE-background comes early in life (or at least prior to the observed time span[29]), and inequality then persists due to smoking persistence and the indirect effect of SE-background. The small or absent direct contribution of SE-background further indicates that the mechanisms causing socioeconomic differences in smoking in one generation affect the offspring's smoking behavior to only a limited extent. As most of the contribution of SE-background is channeled through increased education, it is more likely the individual's own network, skills, and peer acceptance, rather than those of the parents, that explains the differences in smoking participation.[30]

[29]In the initial period 80/81/82 individuals are aged between 19 and 56. The results of the sensitivity analysis of younger samples also indicate that the effect comes early in life.

[30]These results are compatible with the findings in Balia and Jones (2011), which show that smoking behavior carries over between generations, if the socioeconomic gradient in smoking for the generation(s) of parents were negligible. Indeed, there are suggestions that the socioeconomic gradient was negligible or reversed for previous generations (SOU 1999:137).

How to design policies to address the IRSI and the contribution from SE-background depends on how we interpret these results. Schooling is highlighted as the covariate with the largest contribution to inequality, and as an important mediator for the contribution of SE-background. Even though variation is not truly exogenous, I have at the very least controlled for a large part of the unobserved individual heterogeneity making the assumption of independence between the time-invariant unobserved effect and error term more likely to hold, and there appears to be something in the relationship between schooling and smoking that drives IRSI. A true causal interpretation would call for a policy directed at increasing education among the least well off. To what extent do we believe that such a policy would affect smoking behavior? The evidence of an actual causal effect of education from the recent literature is mixed.[31] Additionally, Etilé and Jones (2011), who find schooling to causally decrease smoking participation, conclude that the mechanisms are rather socialization patterns and differences in opportunity costs than more-educated individuals reacting more quickly to health information. In this light, my results imply that policies should directly address smoking prevalence within certain groups (e.g., lower educated from a lower socioeconomic background) by affecting the opportunity cost of smoking, either by changing actual relative prices or by affecting perceptions, and peer acceptance, of smoking among the less educated. Alternatively, as education potentially is related to lower self-control (or lower access to networks), these groups may need support to execute quitting plans.

Although I am bolder regarding the causal interpretation of living in single-adult households, the policy implications are similar. A plausible mechanism that relates living alone to higher smoking prevalence is the lack of a social network needed to break a smoking habit, which

[31]Additionally, the recent literature shows mixed results for the estimate of a causal effect of education on smoking behavior. Arendt (2005), Kenkel et al. (2006), Tenn et al. (2010), and Kemptner et al. (2011), all find little or no evidence for a causal effect of education on smoking behavior. Etilé and Jones (2011) find support for a significant causal effect of education on smoking. De Walque (2007), Grimard and Parent (2007), and Jürges et al. (2011) all find a causal effect on smoking, but not on quitting.

suggests policy promoting services that can ease the burden of quitting within this group. The large contribution from the lagged smoking variable, which indicates that inequalities are persistent over time, further calls for directed efforts to affect decisions to initiate smoking (or to quit early in life) among women with a lower SE-background.

Finally, I must stress that this paper presents a first approach to include the pathways that the childhood conditions and SE-background may have in income-related health inequality. Nevertheless, it leaves room for future research to extend on this approach to further develop a framework that better accounts for causality issues (and is less strict in terms of the functional form). I encourage such a development as it may further help to distinguish between individuals' own effort and circumstances.

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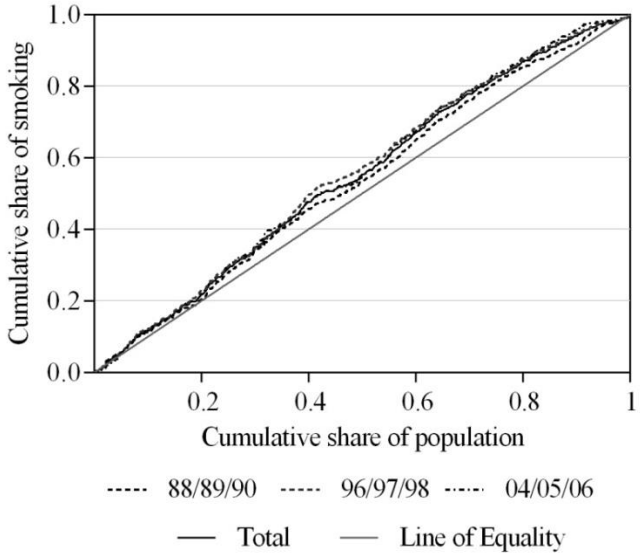
Appendix: Attrition and Sensitive analysis

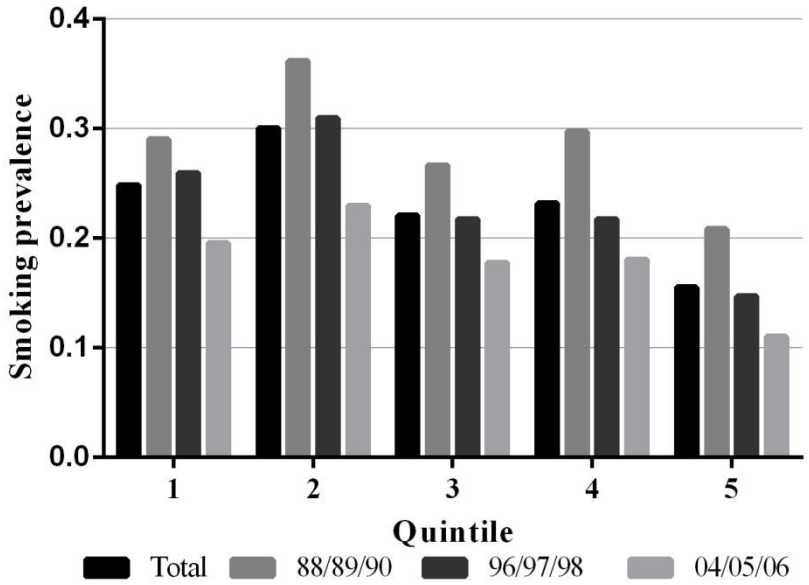
To test for attrition bias in the RE-probit, I perform two versions of a test suggested by Verbeek and Nijman (1992). In each test, a variable reflecting attrition, A, is included in the original model (RE-probit). The null hypothesis of no attrition bias, $E(Y|X,A) = E(Y|X)$, cannot be rejected in either of the tests. The variables used are the number of waves the respondent appears in ($\chi2(1)$ 1.22; $p = 0.27$) and a dummy indicating appearance in all waves ($\chi2(1)$ 1.17; $p = 0.28$). The decomposition analysis (Figure A1) and the level of inequality (Table A2) are alike using both a balanced and unbalanced panel, see and Figure A1.

Figure 1: Concentration Curve of smoking



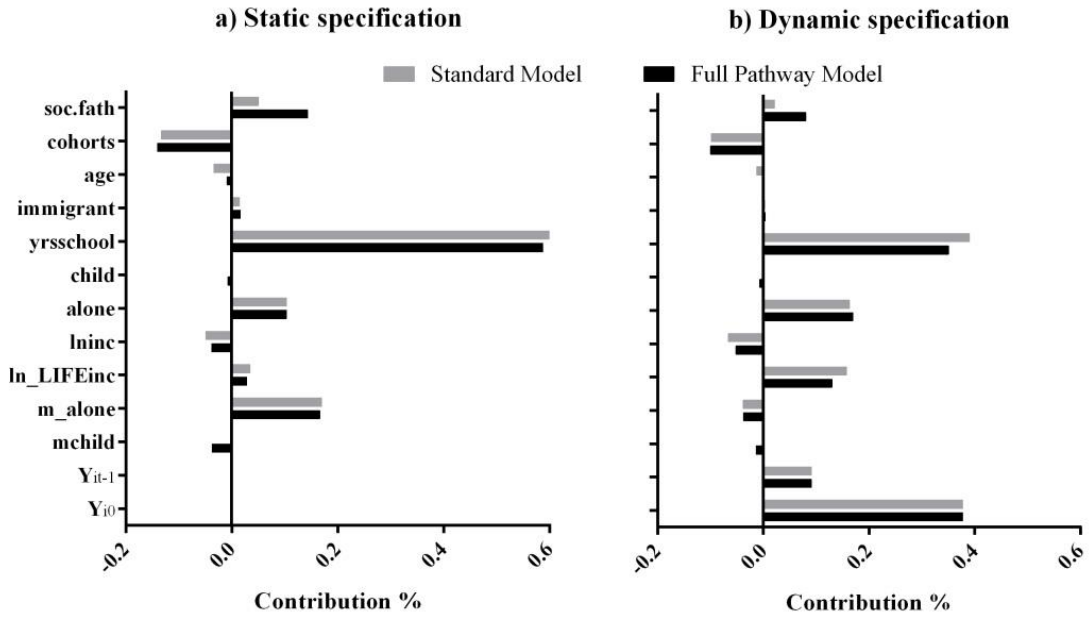
Note: The smoking concentration curve for the total period and each of the three waves, separately.

Figure 2: Smoking participation per quintile



Note: The smoking prevalence per income quintile (lowest first) for each of the three years.

Figure 3. Contributions to total inequality



Note: The contributions per category of variables when accounting – full pathway model in Eq(20) – and not accounting – standard model in Eq(11) – for the indirect effect of SE-background for a static (a) and dynamic (b) specification.

Figure A1: Sensitivity analysis of unbalanced panel

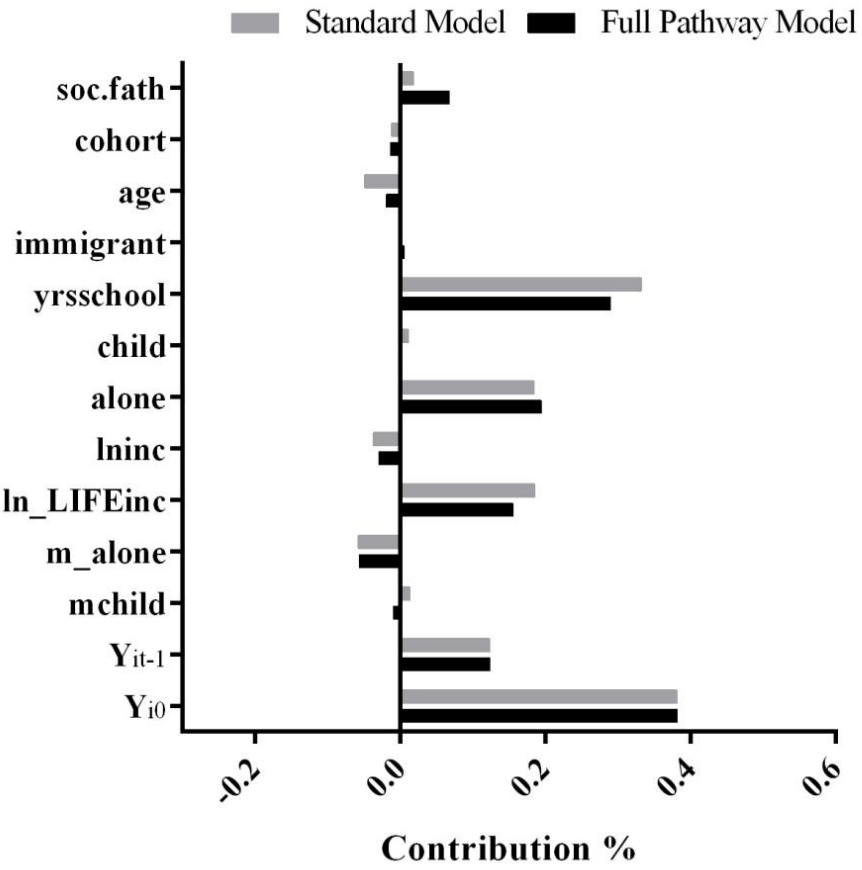
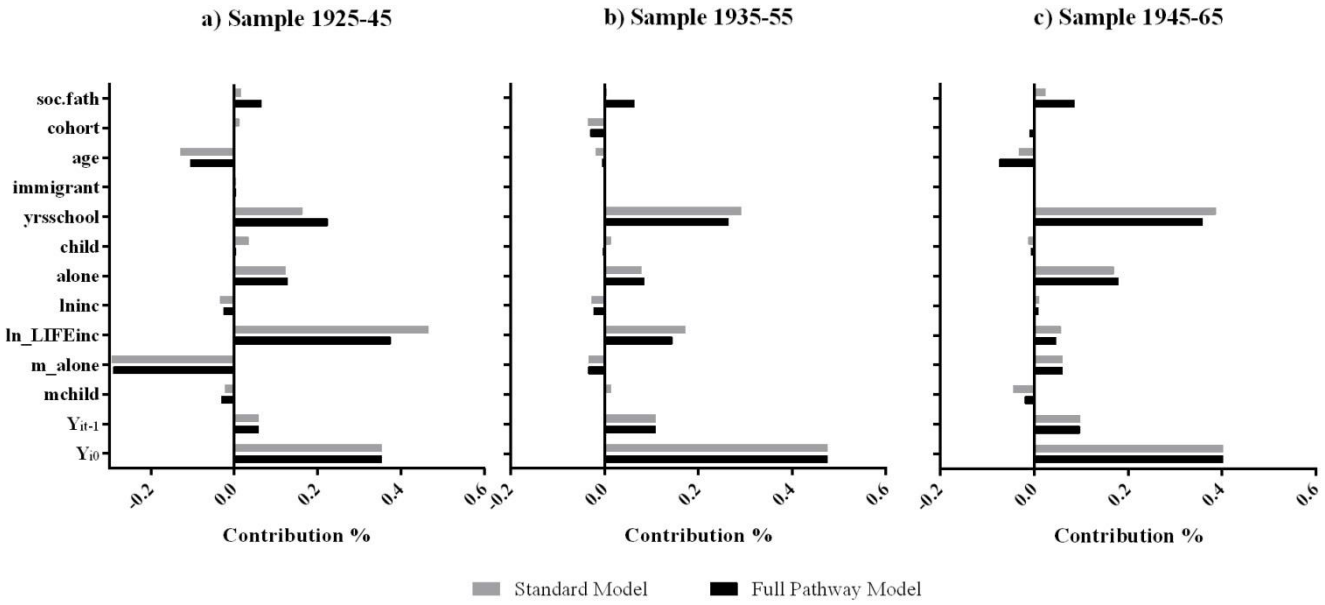


Figure A2: Sensitivity analysis of subsamples



Tables

Table 1a: Definitions of the Variables

Variable	Definition
Y_{it}	= 1 if daily smoker, 0 otherwise
Y_{it-1}	= 1 if the respondent was a daily smoker in the previous wave ($t-1$)
Y_{i0}	= 1 if daily smoker in 80/81/82 (Initial smoking condition)
inc	= disposable income from ULF (in SEK 1,000)
LIFEinc	= standardized mean of disposable income 1990–2005 from LISA (in SEK 1,000)
lninc	= natural logarithm of *inc*
ln_LIFEinc	= natural logarithm of *LIFEinc*
yrsschool	= years of schooling
Children	
childd1	= 1 if no children in the household (omitted)
childd2	= 1 if 1 child in the household
childd3	= 1 if greater than 1 child in the household
alone	= 1 if living in a single-adult household
Im2	= 1 if first-generation immigrant
age	= age of the individual
cohort20	= 1 if born 1924–1934 (omitted)
cohort40	= 1 if born 1934–1944
cohort50	= 1 if born 1945–1954
cohort60	= 1 if born 1955–1963
SE-background	
fath_blue	= 1 if father was a blue-collar worker (omitted)
fath_white_high	= 1 if father was a highly graded white-collar worker
fath_white_low	= 1 if father was a lowly graded white-collar worker
fath_farm	= 1 if father was a farmer
m_childd2	= Individual mean of childd2
m_childd3	= Individual mean of childd3
m_alone	= Individual mean of alone
wave2	= 1 if 1988/89/90 (omitted)
wave3	= 1 if 1996/97/98
wave4	= 1 if 2004/05/06

Table 1b: Descriptive statistics

	Total Sample	
Variable	Mean	Std. Dev.
fath_white_high	0.208	0.406
fath_white_low	0.180	0.384
fath_farm	0.148	0.356
im2	0.054	0.226
cohort40	0.254	0.436
cohort50	0.321	0.467
cohort60	0.235	0.424
Age	51.605	12.153
Yrsschool	11.243	2.502
childd2	0.151	0.358
childd3	0.208	0.406
alone	0.237	0.425
lninc	5.003	0.577
ln_LIFEinc	5.104	0.331
m_childd2	0.151	0.215
m_childd3	0.208	0.283
m_alone	0.237	0.354
Y_{i0}	0.330	0.470
Y_{it-1}	0.282	0.450
Observations	4,893	

Table 2: Levels and changes in income-related inequality in smoking

Wave	Mean	$E(Y)$	$C(Y)$	$C(1-Y)$	$W(Y)$
Total	0.232	−0.084	−0.090	0.027	−0.118
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
88/89/90	0.285	−0.077	−0.067	0.027	−0.094
	(0.000)	(0.002)	(0.002)	(0.002)	(0.001)
96/97/98	0.231	−0.101	−0.109	0.033	−0.142
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
04/05/06	0.179	−0.074	−0.103	0.023	−0.126
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	Δmean	ΔE(Y)	Δ$C(Y)$	Δ$C(1-Y)$	Δ$W(Y)$
88/89/90–96/97/98	−0.055	−0.024	−0.042	0.006	−0.047
		(0.193)	(0.022)	(0.353)	(0.049)
96/97/98–04/05/06	−0.052	0.027	0.006	−0.010	0.016
		(0.121)	(0.781)	(0.068)	(0.535)
88/89/90–04/05/06	−0.106	0.003	−0.036	−0.004	−0.032
		(0.896)	(0.134)	(0.553)	(0.298)

Note: The level and changes in the inequality indices in Eq(2)–Eq(5) using standardized long-term income as the ranking variable. *P*-values obtained by paired bootstrap with 4,999 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap sample (including the standardization of long-term income).

Table 3: Underlying regressions: static and dynamic RE-probit

	(1)	(2)	(3)	(4)	(5)	(6)
	Static RE-probit		Static RE-probit (Mundlak)		Dynamic RE-probit	
Variable	Coef	P.E.	Coef.	P.E.	Coef.	P.E.
fath_white_high	−0.581	−0.050	−0.606	−0.051	−0.132	−0.015
	(0.031)	(0.024)	(0.030)	(0.024)	(0.424)	(0.403)
fath_white_low	−0.260	−0.023	−0.337	−0.029	−0.014	−0.002
	(0.402)	(0.391)	(0.284)	(0.270)	(0.933)	(0.932)
fath_farm	−0.613	−0.052	−0.684	−0.057	−0.043	−0.005
	(0.076)	(0.061)	(0.050)	(0.038)	(0.815)	(0.812)
im2	0.595	0.054	0.628	0.057	0.088	0.011
	(0.211)	(0.222)	(0.185)	(0.198)	(0.694)	(0.699)
cohort40	1.241	0.092	1.021	0.074	0.407	0.037
	(0.023)	(0.005)	(0.066)	(0.030)	(0.169)	(0.113)
cohort50	1.783	0.142	1.705	0.135	0.960	0.108
	(0.038)	(0.018)	(0.049)	(0.025)	(0.045)	(0.018)
cohort60	1.264	0.094	1.325	0.100	0.697	0.071
	(0.288)	(0.274)	(0.268)	(0.253)	(0.298)	(0.290)
age	−0.042	−0.004	−0.081	−0.007	−0.020	−0.002
	(0.282)	(0.275)	(0.054)	(0.048)	(0.380)	(0.368)
yrsschool	−0.319	−0.028	−0.303	−0.026	−0.127	−0.015
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
childd2	−0.333	−0.029	−0.228	−0.019	−0.122	−0.015
	(0.022)	(0.018)	(0.126)	(0.116)	(0.372)	(0.353)
childd3	−0.713	−0.059	−0.495	−0.041	−0.311	−0.035
	(0.000)	(0.000)	(0.009)	(0.006)	(0.057)	(0.040)
alone	0.554	0.049	0.366	0.032	0.392	0.050
	(0.000)	(0.000)	(0.027)	(0.032)	(0.010)	(0.018)
lninc	0.051	0.004	0.077	0.007	0.075	0.009
	(0.675)	(0.675)	(0.572)	(0.572)	(0.551)	(0.547)
ln_LIFEinc			−0.049	−0.004	−0.160	−0.019
			(0.906)	(0.905)	(0.479)	(0.470)
m_childd2			−0.891	−0.076	−0.302	−0.036
			(0.147)	(0.141)	(0.354)	(0.347)
m_childd3			−2.294	−0.195	−0.623	−0.074
			(0.000)	(0.000)	(0.070)	(0.064)
m_alone			0.613	0.052	−0.098	−0.012
			(0.088)	(0.079)	(0.676)	(0.674)
Y_{i0}					2.895	0.450
					(0.000)	(0.000)
Y_{it-1}					0.697	0.092
					(0.000)	(0.001)
wave3	−0.430	−0.039	−0.079	−0.007	−0.394	−0.053
	(0.179)	(0.193)	(0.818)	(0.818)	(0.053)	(0.074)
wave4	−0.868	−0.075	−0.174	−0.015	−0.763	−0.092
	(0.171)	(0.168)	(0.799)	(0.798)	(0.048)	(0.047)
Constant	2.353		−1.706		−3.039	
	(0.331)		(0.478)		(0.025)	
Observations	4,893		4,893		4,893	
Number of *lpnr*	1,631		1,631		1,631	
Model	RE-probit		RE-probit		RE-probit	
$G-H$	24		24		24	
P	0.912		0.913		0.653	
Σ	3.220		3.239		1.372	
Pseudo-R^2	0.117		0.144		0.540	
BIC	3,607		3,618		2,794	
AIC	3,496		3,482		2,645	
Logl	−1,731		−1,720		−1,299	

Note: The coefficients and partial effects evaluated at the mean for the three specifications in Section 2.3. P-values obtained by paired bootstrap with 499 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap samples.

Table 4: Decomposition of income-related inequality in smoking

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
		Static RE-probit		Static RE-probit (Mundlak)		Dynamic RE-probit	
	V_k	Cont	%	Cont	%	Cont	%
fath_white_high	0.034	−0.007	8.2%	−0.007	8.4%	−0.002	2.5%
	(0.000)	(0.047)		(0.045)		(0.417)	
fath_white_low	0.020	−0.002	2.2%	−0.002	2.8%	−0.000	0.2%
	(0.001)	(0.426)		(0.312)		(0.936)	
fath_farm	−0.023	0.005	−5.6%	0.005	−6.2%	0.000	−0.6%
	(0.000)	(0.108)		(0.082)		(0.817)	
im2	−0.005	−0.001	1.4%	−0.001	1.5%	−0.000	0.3%
	(0.113)	(0.373)		(0.356)		(0.753)	
cohort40	0.013	0.005	−5.9%	0.004	−4.7%	0.002	−2.4%
	(0.036)	(0.077)		(0.113)		(0.202)	
cohort50	0.025	0.014	−16.7%	0.013	−15.9%	0.011	−12.7%
	(0.000)	(0.040)		(0.050)		(0.038)	
cohort60	−0.015	−0.006	6.9%	−0.006	7.3%	−0.004	5.2%
	(0.037)	(0.345)		(0.326)		(0.391)	
age	−0.104	0.002	−1.8%	0.003	−3.4%	0.001	−1.2%
	(0.611)	(0.723)		(0.653)		(0.745)	
yrsschool	0.540	−0.059	70.9%	−0.056	66.6%	−0.033	39.0%
	(0.000)	(0.000)		(0.000)		(0.000)	
childd2	0.004	−0.000	0.6%	−0.000	0.4%	−0.000	0.3%
	(0.224)	(0.317)		(0.392)		(0.533)	
childd3	−0.002	0.000	−0.5%	0.000	−0.4%	0.000	−0.3%
	(0.749)	(0.750)		(0.753)		(0.761)	
alone	−0.068	−0.013	16.0%	−0.009	10.3%	−0.014	16.3%
	(0.000)	(0.001)		(0.034)		(0.020)	
lninc	0.156	0.003	−3.3%	0.004	−4.9%	0.006	−6.6%
	(0.000)	(0.674)		(0.571)		(0.546)	
ln_LIFEinc	0.172			−0.003	3.4%	−0.013	15.7%
	(0.000)			(0.904)		(0.467)	
m_childd3	0.004			−0.001	1.5%	−0.001	0.7%
	(0.224)			(0.411)		(0.524)	
m_childd2	−0.002			0.001	−1.7%	0.001	−0.7%
	(0.749)			(0.766)		(0.791)	
m_alone	−0.068			−0.014	16.9%	0.003	−3.8%
	(0.000)			(0.082)		(0.676)	
Y_{i0}	−0.018					−0.032	37.8%
	(0.006)					(0.008)	
Y_{it-1}	−0.021					−0.008	9.1%
	(0.000)					(0.014)	
Residual		-0.022	23.9%	−0.012	14.7%	0.000	0.2%

Note: Column 1 reports the absolute concentration index for each explanatory variable in Eq(9). Columns 2–7 present the contribution to absolute inequality in levels and in percentage of the total inequality index E(Y) for the three specifications. P-values obtained by paired bootstrap with 499 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap samples. Note that the p-values of the contribution to E(Y) are close to equivalent to the contribution to other indices.

Table 5a Dynamic RE-probit: Total and indirect effects

Variable	(1) Direct effect	(2) Mediating specification	(3) Total indirect effect	(4) Indirect via $\boldsymbol{Z}$ and $\bar{\boldsymbol{Z}}$	(5) Indirect via schooling (1)	(6) Indirect via schooling (2)
fath_white_high	−0.132	−0.340	−0.208	−0.007	−0.183	−0.017
	(0.424)	(0.039)	(0.000)	(0.762)	(0.000)	(0.245)
fath_white_low	−0.014	−0.187	−0.172	0.037	−0.191	−0.018
	(0.933)	(0.268)	(0.001)	(0.160)	(0.000)	(0.248)
fath_farm	−0.043	−0.036	0.006	0.025	−0.017	−0.002
	(0.815)	(0.841)	(0.859)	(0.367)	(0.447)	(0.614)
im2	0.088	0.116	0.028	−0.002	0.027	0.003
	(0.694)	(0.611)	(0.542)	(0.952)	(0.365)	(0.558)
cohort40	0.407	0.414	0.006	0.080	−0.068	−0.006
	(0.169)	(0.159)	(0.924)	(0.129)	(0.103)	(0.328)
cohort50	0.960	0.817	−0.142	0.002	−0.131	−0.013
	(0.045)	(0.084)	(0.122)	(0.976)	(0.047)	(0.289)
cohort60	0.697	0.485	−0.211	−0.064	−0.134	−0.013
	(0.298)	(0.467)	(0.074)	(0.408)	(0.119)	(0.339)
age	−0.020	0.000	0.020	0.017	0.003	0.000
	(0.380)	(0.995)	(0.003)	(0.007)	(0.285)	(0.494)
yrsschool	−0.127	−0.139	−0.012	−0.012		
	(0.000)	(0.000)	(0.236)	(0.236)		
m_alone	−0.098	−0.098				
	(0.676)	(0.676)				
ln_LIFEinc	−0.160	−0.160				
	(0.479)	(0.479)				
m_childd2	−0.302	−0.302				
	(0.354)	(0.354)				
m_childd3	−0.623	−0.623				
	(0.070)	(0.070)				
alone	0.392	0.392				
	(0.010)	(0.010)				
lninc	0.075	0.075				
	(0.551)	(0.551)				
childd2	−0.122	−0.122				
	(0.372)	(0.372)				
childd3	−0.311	−0.311				
	(0.057)	(0.057)				
Y_{it-1}	0.697	0.697				
	(0.000)	(0.000)				
Y_{i0}	2.895	2.895				
	(0.000)	(0.000)				
wave3	−0.394	−0.482				
	(0.053)	(0.015)				
wave4	−0.763	−0.934				
	(0.048)	(0.014)				

Note: For the SE-background variables, the coefficients in Column 2 correspond the total effect $\psi + \Psi^B + \Psi^S\varphi_1^z$. The estimate of the direct effect ψ corresponds to the coefficient of the dynamic RE-probit presented in Column 5 of Table 3. The total indirect effect in Column 3 corresponds to $\Psi^B + \Psi^S\varphi_1^z$. The first part of the total indirect effect Ψ^B is divided into the indirect effects from Z_{it} and $\bar{Z}_i$ – $\varphi_3^z\lambda + \varphi_3^{\bar{z}}\rho$ – (Column 3), and the indirect effect from schooling, $\tau\varphi_1^s$, (Column 4). The second part, $\Psi^S\varphi_1^z = (\varphi_3^z\lambda + \varphi_3^{\bar{z}}\rho)\varphi_1^z$, denotes the second-layer indirect effects mediated through education and, in turn, Z_{it} and $\bar{Z}_i$ (Column 5). P-values obtained by paired bootstrap with 499

replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap samples.

Table 5b: Static RE-probit: Total and indirect effects

Variable	(1) Direct effect	(2) Mediating specification	(3) Total indirect effect	(4) Indirect via $\boldsymbol{Z}$ and $\bar{\boldsymbol{Z}}$	(5) Indirect via schooling (1)	(6) Indirect via schooling (2)
fath_white_high	−0.606	−1.074	−0.469	0.003	−0.439	−0.033
	(0.030)	(0.000)	(0.000)	(0.957)	(0.000)	(0.276)
fath_white_low	−0.337	−0.712	−0.376	0.116	−0.458	−0.034
	(0.284)	(0.025)	(0.001)	(0.081)	(0.000)	(0.281)
fath_farm	−0.684	−0.659	0.025	0.068	−0.040	−0.003
	(0.050)	(0.062)	(0.751)	(0.245)	(0.445)	(0.625)
im2	0.628	0.693	0.065	−0.005	0.065	0.005
	(0.185)	(0.156)	(0.573)	(0.954)	(0.369)	(0.582)
cohort40	1.021	1.093	0.072	0.246	−0.162	−0.012
	(0.066)	(0.047)	(0.628)	(0.028)	(0.101)	(0.359)
cohort50	1.705	1.377	−0.328	0.011	−0.315	−0.023
	(0.049)	(0.113)	(0.125)	(0.938)	(0.041)	(0.320)
cohort60	1.325	0.805	−0.520	−0.175	−0.321	−0.024
	(0.268)	(0.507)	(0.069)	(0.389)	(0.111)	(0.368)
Age	−0.081	−0.021	0.060	0.053	0.007	0.001
	(0.054)	(0.609)	(0.000)	(0.000)	(0.284)	(0.526)
Yrsschool	−0.303	−0.326	−0.023	−0.023		
	(0.000)	(0.000)	(0.270)	(0.270)		
m_alone	0.613	0.613				
	(0.088)	(0.088)				
ln_LIFEinc	−0.049	−0.049				
	(0.906)	(0.906)				
m_childd2	−0.891	−0.891				
	(0.147)	(0.147)				
m_childd3	−2.294	−2.294				
	(0.000)	(0.000)				
Alone	0.366	0.366				
	(0.027)	(0.027)				
Lninc	0.077	0.077				
	(0.572)	(0.572)				
childd2	−0.228	−0.228				
	(0.126)	(0.126)				
childd3	−0.495	−0.495				
	(0.009)	(0.009)				
wave3	−0.079	−0.452				
	(0.818)	(0.167)				
wave4	−0.174	−0.924				
	(0.799)	(0.157)				

Note: P-values obtained by paired bootstrap with 499 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap samples.

Table 6a: Comparison with a full pathway model. Dynamic specification

Variable	(1) P.E.	(2) Cont E	(3) %-cont	(4) ΔPE	(5) Δcont E	(6) Δ%-cont
fath_white_high	−0.039	−0.005	6.4%	−0.024	−0.003	3.9%
	(0.025)	(0.048)		(0.000)	(0.006)	
fath_white_low	−0.023	−0.002	2.1%	−0.021	−0.002	2.0%
	(0.245)	(0.305)		(0.001)	(0.037)	
fath_farm	−0.005	0.000	−0.5%	0.001	−0.000	0.1%
	(0.839)	(0.844)		(0.906)	(0.911)	
im2	0.014	−0.000	0.4%	0.004	−0.000	0.1%
	(0.621)	(0.698)		(0.541)	(0.674)	
cohort40	0.041	0.002	−2.6%	0.004	0.000	−0.2%
	(0.102)	(0.188)		(0.563)	(0.592)	
cohort50	0.093	0.009	−10.9%	−0.015	−0.001	1.7%
	(0.048)	(0.070)		(0.110)	(0.152)	
cohort60	0.049	−0.003	3.6%	−0.022	0.001	−1.6%
	(0.461)	(0.525)		(0.060)	(0.165)	
Age	0.000	−0.000	0.0%	0.002	−0.001	1.2%
	(0.995)	(0.998)		(0.002)	(0.632)	
yrsschool	−0.017	−0.029	35.1%	−0.001	0.003	−3.9%
	(0.000)	(0.000)		(0.224)	(0.291)	
childd2	−0.014	−0.000	0.2%	0.001	0.000	−0.1%
	(0.344)	(0.581)		(0.547)	(0.608)	
childd3	−0.033	0.001	−0.8%	0.002	0.000	−0.5%
	(0.029)	(0.154)		(0.346)	(0.536)	
Alone	0.053	−0.014	17.0%	0.003	−0.001	0.7%
	(0.025)	(0.027)		(0.232)	(0.483)	
Lninc	0.009	0.004	−5.2%	0.000	−0.001	1.5%
	(0.547)	(0.543)		(0.596)	(0.558)	
ln_LIFEinc	−0.019	−0.011	13.0%	0.000	0.002	−2.8%
	(0.470)	(0.464)		(1.000)	(0.488)	
m_childd2	−0.036	−0.000	0.4%	−0.000	0.000	−0.3%
	(0.347)	(0.587)		(0.354)	(0.609)	
m_childd3	−0.074	0.001	−1.7%	0.000	0.001	−1.1%
	(0.064)	(0.218)		(1.000)	(0.546)	
m_alone	−0.012	0.003	−3.7%	0.000	−0.000	0.1%
	(0.674)	(0.674)		(1.000)	(0.872)	
Y_{i0}	0.450	−0.032	37.8%	−0.000	0.000	0.0%
	(0.000)	(0.008)		(0.174)	(0.205)	
Y_{it-1}	0.092	−0.008	9.1%	0.000	0.000	0.0%
	(0.001)	(0.014)		(1.000)	(1.000)	
wave3	−0.067	0.000	0.0%	−0.014	0.000	0.0%
	(0.029)	(0.868)		(0.152)	(0.882)	
wave4	−0.112	−0.000	0.0%	−0.021	−0.000	0.0%
	(0.014)	(0.155)		(0.159)	(0.250)	

Note: Columns 1–3 present the partial effects and the contribution to absolute inequality, *E*, in levels and as percentages of the total index using the full model allowing for intermediating effects. Columns 4–6 present the difference to the panel model not allowing for intermediate effects. P-values obtained by paired bootstrap with 499 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap samples.

Table 6b: Comparison with a full pathway model. Static specification

Variable	(1) P.E.	(2) Cont *E*	(3) %-cont	(4) Δ PE	(5) Δcont *E*	(6) Δ%-cont
fath_white_high	−0.089	−0.012	15%	−0.038	−0.005	6%
	(0.000)	(0.002)		(0.000)	(0.004)	
fath_white_low	−0.062	−0.005	6%	−0.032	−0.003	3%
	(0.018)	(0.068)		(0.001)	(0.042)	
fath_farm	−0.058	0.005	−6%	−0.000	0.000	0%
	(0.049)	(0.091)		(0.980)	(0.981)	
im2	0.063	−0.001	2%	0.006	−0.000	0%
	(0.171)	(0.350)		(0.567)	(0.681)	
cohort30	0.085	0.005	−5%	0.011	0.001	−1%
	(0.019)	(0.094)		(0.300)	(0.359)	
cohort40	0.111	0.011	−13%	−0.025	−0.002	3%
	(0.081)	(0.107)		(0.114)	(0.160)	
cohort50	0.060	−0.004	4%	−0.040	0.002	−3%
	(0.498)	(0.530)		(0.053)	(0.147)	
Age	−0.002	0.001	−1%	0.005	−0.002	3%
	(0.605)	(0.837)		(0.000)	(0.623)	
Yrsschool	−0.028	−0.049	59%	−0.002	0.007	−8%
	(0.000)	(0.000)		(0.267)	(0.159)	
childd2	−0.019	−0.000	0%	0.001	0.000	0%
	(0.110)	(0.472)		(0.330)	(0.493)	
childd3	−0.040	0.001	−1%	0.001	0.000	−1%
	(0.004)	(0.116)		(0.193)	(0.504)	
Alone	0.033	−0.009	10%	0.001	−0.000	0%
	(0.036)	(0.037)		(0.299)	(0.971)	
Lninc	0.007	0.003	−4%	0.000	−0.001	1%
	(0.572)	(0.569)		(1.000)	(0.583)	
ln_LIFEinc	−0.004	−0.002	3%	0.000	0.001	−1%
	(0.905)	(0.903)		(1.000)	(0.909)	
m_childd2	−0.076	−0.001	1%	0.000	0.001	−1%
	(0.141)	(0.491)		(1.000)	(0.533)	
m_childd3	−0.195	0.004	−5%	0.000	0.002	−3%
	(0.000)	(0.101)		(0.151)	(0.483)	
m_alone	0.052	−0.014	17%	0.000	0.000	0%
	(0.079)	(0.081)		(1.000)	(0.668)	
wave3	−0.041	0.000	0%	−0.034	0.000	0%
	(0.185)	(0.885)		(0.002)	(0.864)	
wave4	−0.078	−0.000	0%	−0.064	−0.000	0%
	(0.155)	(0.207)		(0.002)	(0.166)	

Note: Columns 1–3 present the partial effects and the contribution to absolute inequality, *E*, in levels and as percentages of the total index using the full model allowing for intermediating effects. Columns 4–6 present the difference in the panel model not allowing for intermediate effects. *P*-values obtained by paired bootstrap with 499 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap samples.

Table A1a: Definitions of variables

m_LISA_child	= the mean of the number of children in the household 1990–2006 from LISA
m_LISA_child_sq	= *m_LISA_child* squared
m_LISA _alone	= the individual mean of living in a single household according to LISA
LISA _largecity	= the individual mean of living in a large city (Stockholm, Gothenburg, Malmoe)
LISA _city	= the individual mean of living in another city

Table A1b: Regression underlying the standardization of income variable

Variable	(1) *ln_lifeincome*
Yrsschool	0.042
	(0.000)
fath_white_high	0.063
	(0.001)
fath_white_low	0.009
	(0.652)
fath_farm	−0.043
	(0.044)
im2	−0.034
	(0.269)
Inkage	−25.838
	(0.083)
Inkagesq	26.404
	(0.076)
Inkagecube	−11.530
	(0.076)
inkage4	1.810
	(0.084)
m_stad	0.027
	(0.121)
m_storstad	0.133
	(0.000)
m_LISA_alone	−0.273
	(0.000)
m_LISA_child_sq	−0.001
	(0.884)
m_LISA_child	−0.063
	(0.024)
Constant	13.872
	(0.012)
Observations	1,631
R^2	0.347

Note: The table present results for the regression that underlies the standardized income measure. P-values presented in parenthesis.

Table A2: Standardized long-term income (unbalanced panel)

Wave	Mean	$C(y)$	$C(1-y)$	$W(y)$	$E(y)$	n
88/89/90	0.306	−0.070	0.031	−0.101	−0.086	2,458
		(0.000)	(0.000)	(0.000)	(0.000)	
96/97/98	0.243	−0.122	0.039	−0.161	−0.118	2,119
		(0.000)	(0.000)	(0.000)	(0.000)	
04/05/06	0.183	−0.107	0.024	−0.131	−0.079	1,764
		(0.000)	(0.000)	(0.000)	(0.000)	
Total	0.251	−0.098	0.033	−0.131	−0.098	6,341
		(0.000)	(0.000)	(0.000)	(0.000)	
	Δmean	$\Delta C(y)$	$\Delta C(1-y)$	$\Delta W(y)$	$\Delta E(y)$	
88/89/90–96/97/98	0.062	−0.050	0.009	−0.059	−0.033	
		(0.002)	(0.111)	(0.006)	(0.044)	
88/89/90–04/05/06	0.122	−0.041	−0.003	−0.039	−0.002	
		(0.098)	(0.706)	(0.211)	(0.904)	
96/97/98–04/05/06	0.059	0.0089	−0.0115	0.0205	0.0306	
		(0.714)	(0.064)	(0.499)	(0.116)	

Note: The level and changes in the inequality indices in Eq(2)–Eq(5) using standardized long-term income as the ranking variable for an unbalanced panel. *P*-values obtained by paired bootstrap with 4,999 replications are presented in parenthesis. Each bootstrap sample is drawn at the individual level and all estimates are computed within the same bootstrap sample (including the standardization of long-

Table A3: Auxiliary regressions

Variable	(1) *yrsschool*	(2) *alone*	(3) *lninc*	(4) *childd2*	(5) *childd3*	(6) *m_alone*	(7) *ln_LIFEinc*	(8) *m_childd2*	(9) *m_childd3*
fath_white_high	1.447	0.001	0.055	−0.016	0.006	0.001	0.082	−0.016	0.006
	(0.000)	(0.943)	(0.008)	(0.219)	(0.657)	(0.932)	(0.000)	(0.030)	(0.468)
fath_white_low	1.508	0.056	0.051	−0.017	−0.014	0.056	0.025	−0.017	−0.014
	(0.000)	(0.001)	(0.022)	(0.230)	(0.322)	(0.000)	(0.048)	(0.035)	(0.106)
fath_farm	0.132	0.032	−0.157	−0.008	−0.013	0.032	−0.069	−0.008	−0.013
	(0.183)	(0.074)	(0.000)	(0.571)	(0.388)	(0.032)	(0.000)	(0.318)	(0.160)
im2	−0.215	0.035	−0.017	0.023	0.005	0.035	−0.020	0.023	0.005
	(0.138)	(0.184)	(0.626)	(0.295)	(0.826)	(0.111)	(0.300)	(0.065)	(0.720)
cohort40	0.534	−0.078	0.215	0.012	−0.116	−0.078	0.101	0.012	−0.116
	(0.001)	(0.006)	(0.000)	(0.620)	(0.000)	(0.001)	(0.000)	(0.383)	(0.000)
cohort50	1.039	−0.093	0.259	0.036	−0.046	−0.093	0.111	0.036	−0.046
	(0.000)	(0.039)	(0.000)	(0.327)	(0.214)	(0.013)	(0.001)	(0.084)	(0.043)
cohort60	1.059	−0.053	0.168	0.021	0.038	−0.053	0.102	0.021	0.038
	(0.002)	(0.400)	(0.038)	(0.682)	(0.462)	(0.313)	(0.027)	(0.470)	(0.231)
age	−0.023	0.003	0.006	−0.008	−0.015	0.003	0.007	−0.008	−0.015
	(0.049)	(0.239)	(0.023)	(0.000)	(0.000)	(0.158)	(0.000)	(0.000)	(0.000)
yrsschool		0.002	0.040	0.002	0.008	0.002	0.043	0.002	0.008
		(0.349)	(0.000)	(0.335)	(0.000)	(0.261)	(0.000)	(0.090)	(0.000)
wave3	0.183	0.011	0.092	−0.007	−0.023	−0.020	−0.057	0.061	0.119
	(0.136)	(0.641)	(0.001)	(0.697)	(0.218)	(0.284)	(0.001)	(0.000)	(0.000)
wave4	0.366	0.060	0.252	0.018	−0.011	−0.040	−0.113	0.123	0.238
	(0.071)	(0.109)	(0.000)	(0.555)	(0.712)	(0.195)	(0.000)	(0.000)	(0.000)
Constant	10.944	0.101	3.943	0.508	0.928	0.145	4.212	0.450	0.798
	(0.000)	(0.437)	(0.000)	(0.000)	(0.000)	(0.183)	(0.000)	(0.000)	(0.000)
Observations	4,893	4,893	4,893	4,893	4,893	4,893	4,893	4,893	4,893
R^2	0.165	0.027	0.136	0.077	0.279	0.025	0.147	0.174	0.443

Note: p-values presented in parenthesis.

Table A4: Probit of initial smoking condition Y_{i0}.

Variable	(1) Probit	(2) Probit
fath_white_high	−0.155	−0.165
	(0.081)	(0.070)
fath_white_low	−0.128	−0.152
	(0.171)	(0.096)
fath_farm	−0.276	−0.292
	(0.007)	(0.006)
im2	0.235	0.239
	(0.096)	(0.108)
cohort40	0.441	0.344
	(0.007)	(0.036)
cohort50	0.423	0.358
	(0.093)	(0.146)
cohort60	0.290	0.289
	(0.405)	(0.392)
age	−0.007	−0.020
	(0.553)	(0.088)
yrsschool	−0.068	−0.063
	(0.000)	(0.000)
childd2	0.049	0.059
	(0.593)	(0.531)
childd3	−0.203	−0.169
	(0.034)	(0.082)
alone	0.189	0.087
	(0.033)	(0.359)
lninc	0.024	0.014
	(0.682)	(0.851)
missinc	0.447	0.491
	(0.220)	(0.225)
ln_LIFEinc		0.050
		(0.683)
m_childd2		−0.179
		(0.336)
m_childd3		−0.609
		(0.000)
m_alone		0.227
		(0.025)
Constant	0.229	0.589
	(0.737)	(0.439)
Observations	1,631	1,631
pseudo_R2	0.0756	0.0949
Logl	−997.4	−986.7

Note: For 12 observations with missing value on *lninc* in 80/81/82 the value from 88/89/90 is used. *missinc* is a dummy indicating this. P-value presented in parentheses.