

# **Timing of puberty and reserve capacity in adolescence as pathways to educational level in adulthood – a longitudinal study**

Paulyn Jean Acacio-Claro<sup>1</sup> Leena Kristiina Koivusilta<sup>2</sup>

David Teye Doku<sup>1,3</sup> Arja Hannele Rimpelä<sup>1,4</sup>

<sup>1</sup>*Faculty of Social Sciences, Health Sciences, **Tampere University** and PERLA (Tampere Centre for Childhood, Youth and Family Research), **Tampere University**, Finland*

<sup>2</sup>*Department of Social Research, Faculty of Social Sciences, University of Turku, Finland*

<sup>3</sup>*Department of Population and Health, University of Cape Coast, Cape Coast, Ghana*

<sup>4</sup>*Department of Adolescent Psychiatry, Tampere University Hospital, Nokia, Finland*

Correspondence

Paulyn Jean Acacio-Claro

Mailing address: Faculty of Social Sciences, Health Sciences, P.O. Box 100, 33014, **Tampere University**, Finland

Email address: Claro.Paulyn.J@student.uta.fi

# Timing of puberty and reserve capacity in adolescence as pathways to educational level in adulthood – a longitudinal study

Abstract

*Background.* Family socioeconomic status (SES) is related to a child's educational success. Intermediate pathways for this relationship, such as through **pubertal timing** and reserve capacity, occur in adolescence.

*Aim.* We studied whether family SES affected a child's adult education through a psychosocial and behavioural pathway (reserve capacity) and/or a biological pathway (pubertal timing) or only through school achievement in adolescence.

*Subjects and methods.* **Finnish adolescents** sampled in five cross-sectional surveys in 1985-1995 (N=37,876) were followed through the Registry of Completed Education and Degrees until 2009, when they were 29-43 years old. **Family SES data** also came from this registry. Structural equation modelling adjusted for ages at baseline and follow-up was used.

*Results.* Low family SES increased the probability of low adult education, delayed pubertal timing (in boys), weak reserve capacity and low school achievement. Reserve capacity and school achievement directly affected adult education and mediated the relationship of family SES with the outcome. Delayed pubertal timing predicted low adult education except when school achievement was added in the model.

*Conclusions.* We elucidated **the roles and interrelationships of family SES and important adolescent pathways in educational trajectories. Supporting adolescents during their critical developmental and learning transitions could help reduce educational inequalities.**

Keywords: socioeconomic status, puberty, education, reserve capacity, school achievement

## **Introduction**

From a developmental perspective, adolescence has a unique position in the life course because it could either lessen or aggravate the impact of early childhood disadvantages on adult outcomes (Johnson et al. 2011). Rapid biological and social changes such as puberty and increasing autonomy from one's family (Viner et al. 2012), along with childhood experiences and environmental influences shape young people's beliefs and actions which affect "successful" transitions into adulthood (Johnson et al. 2011). Hence, intermediate pathways from childhood exposures to educational trajectories may be elucidated in adolescence.

Socioeconomic status (SES) is an important exposure in early life which has been strongly linked to various developmental outcomes of children and adolescents, particularly, educational attainment (Conger et al. 2010; Merritt and Buboltz 2015; Acacio-Claro et al. 2017). Previous research focusing on SES as a predictor of child development explained that such links probably occur through family dynamics, parenting practices and investments for children (Martin et al. 2010). Accordingly, families with more economic resources tend to invest more in the health and education of their children than those with less resources (Conger et al. 2010). Research has also shown that economic hardship affects relationships between parents and children leading to poor parenting practices or poor communication in the family, which influence the cognitive, emotional and behavioural development of children (Kroenke 2008; Conger et al. 2010).

During adolescence, one salient marker of development with likely effects persisting until adulthood is puberty and its timing has been extensively studied due to its complex familial and environmental causes (Parent et al. 2003; Euling et al. 2008; Golub et al. 2008; Johnson et al. 2011; Graber 2013). The physical, behavioural and

hormonal effects of puberty, particularly when occurring earlier or later than in one's age mates, bring psychological and adjustment issues linked to elevated symptomatology and risks of psychopathology during adolescence and other disorders in adulthood (Golub et al. 2008; Graber 2013). Higher rates of depressive symptoms, especially in girls (Copeland et al. 2010; Keenan et al. 2014), and risky health behaviours (Koivusilta and Rimpelä 2006; Golub et al. 2008; Downing and Bellis 2009; Graber 2013) and higher risks for developing cardiovascular disease (Golub et al. 2008; Jacobsen et al. 2009; Lakshman et al. 2009; Bleil et al. 2013), type 2 diabetes, breast and testicular cancers (Golub et al. 2007) were associated with early maturation. On the other hand, late maturation increased fracture risk (Zhu and Chan 2017) and psychopathology in boys in terms of higher rates of depressive symptoms and disruptive behaviours (Graber 2013; Zhu and Chan 2017). Aside from its health impact, recent evidence suggests that pubertal timing has cognitive effects which may be reflected in academic performance (Cavanagh et al. 2007; Martin and Steinbeck 2017) and educational outcomes (Koivusilta and Rimpelä 2004; Koerselman and Pekkarinen 2017), influencing socioeconomic conditions in adulthood (Johnson et al. 2011; Koerselman and Pekkarinen 2017).

Secular changes observed regarding pubertal timing have been attributed mainly to improvements in nutrition and health, including increase in body fat (de Muinck Keizer-Schrama and Mul 2001; Parent et al. 2003). Pubertal timing is also influenced by certain gene regulators, gender, race/ethnicity (Obeidallah et al. 2000; Parent et al. 2003; Euling et al. 2008) and exposure to endocrine disrupting chemicals (Parent et al. 2003; Aksglaede et al. 2008). A stressful family environment characterized by family conflict (Bleil et al. 2013) and stressful life events (Sun et al. 2017), father absenteeism, divorce and single parent families (Bellis et al. 2006) is likewise linked to altered

pubertal timing. Notably, the onset of puberty also depends on socioeconomic conditions in the family. Research has documented socioeconomic inequalities in timing of puberty with mixed findings (de Muinck Keizer-Schrama and Mul 2001; Parent et al. 2003; Downing and Bellis 2009; James-Todd et al. 2010; Sun et al. 2017). On one hand, high SES or “privileged conditions” were shown to have shifted pubertal timing towards earlier ages (de Muinck Keizer-Schrama and Mul 2001; Parent et al. 2003) possibly due to improved childhood health status (de Muinck Keizer-Schrama and Mul 2001; Bellis et al. 2006) and nutrition (Parent et al. 2003; Bellis et al. 2006; Kyweluk et al. 2017). On the other hand, low SES or childhood socioeconomic disadvantage was also found to accelerate pubertal onset (James-Todd et al. 2010; Sun et al. 2017) likely due to environmental stress which hastens reproductive maturation (Obeidallah et al. 2000; James-Todd et al. 2010; Xu et al. 2017).

The mechanisms through which pubertal timing occurs and causes adverse health outcomes likely represent the interplay of socioeconomic, psychosocial and biobehavioural pathways in the life-course (Gallo et al. 2009; Matthews and Gallo, 2011). An integrative framework overarching this is the reserve capacity model proposed by Gallo and Matthews (2003). This model posits that low SES increases one’s exposure to environmental stressors and depletes psychosocial resources such as self-efficacy, mastery and social support, triggering negative emotional and physiological responses, affecting health via altered biological and behavioural pathways (Gallo et al. 2009; Matthews et al. 2010; Matthews and Gallo 2011). Initially designed to understand how the psychosocial pathway links SES with physical health (Gallo and Matthews 2003), research which tested this model among adults produced inconclusive results about the hypothesized relationships (Matthews et al. 2010). However, studies conducted among children and adolescents yielded clearer directions

on the connections of childhood SES and adult health outcomes through reserve capacity and biobehavioural pathways (Matthews et al. 2010). In addition, low SES and poor psychosocial functioning early in life placed children and adolescents at risk of lower educational outcomes compared to those with high SES and/or strong reserve capacity (Matthews et al. 2010).

We adopt this framework to assess if pubertal timing and reserve capacity are such pathways through which SES influenced educational trajectories. We further extend the reserve capacity framework to include health-promoting behaviours, particularly, tooth brushing and physical activity, as these underlie psychosocial resources such as perceived control and self-efficacy (Robbins et al. 2004; Cinar et al. 2009; Pakpour and Sniehotta 2012). Moreover, both behaviours were found to serve as pathways from childhood socioeconomic position to adult education level (Koivusilta et al. 2013), hence, we included these variables in the present study within the context of reserve capacity. Reserve capacity, in this study, covers three dimensions, namely: perceived health, health-promoting behaviour and social support, all of which have been previously demonstrated to predict adult education (Acacio-Claro et al. 2017). We also add another factor, school achievement, as several studies have shown this to be one of the strongest predictors of adult education (Slominski et al. 2011; Brekke 2015; Acacio-Claro et al. 2017). Further, we propose that the pathways occurring in adolescence might interact with each other to affect adult education (Figure 1).

In general, we studied whether family SES affects a child's adult education through a psychosocial and behavioural pathway (reserve capacity) and/or a biological pathway (timing of puberty) or only through school achievement in adolescence. Specifically, we want to test the following hypotheses: (1) family SES is related to pubertal timing, reserve capacity and school achievement; (2) pubertal timing and

reserve capacity influence adult education level; and, (3) family SES relates to adult education level either directly or indirectly (i.e., mediated by pubertal timing and reserve capacity). Understanding these mechanisms will help clarify the links among SES, adolescent pathways and adult education and point to new ways of supporting young people to achieve their full potential in learning – a recognized important life stage transition (Viner et al. 2012).

## **Materials and methods**

### ***Study design and sample***

A longitudinal study design was constructed using two data sources linked through unique national personal identification numbers. Baseline data were obtained from the Adolescent Health and Lifestyle Surveys (AHLS) of 1985, 1987, 1991, 1993 and 1995. The AHLS, monitors the health and health-related lifestyle of adolescents in Finland. Nationally representative samples of 14-, 16-, and 18-year-old Finns born on certain days in June, July and August **between 1966 to 1980** were drawn each study year from the Population Register Centre. Even though the AHLS was conducted biennially since 1977, the variables suitable for measuring reserve capacity were included only in the above-mentioned years. A self-administered questionnaire, to be voluntarily answered, was sent by post in February, followed by two re-inquiries to non-respondents. Overall response rate was 79.1 % (N=37,876), with 71.9% (N=17,531) for boys and 86.6% (N=20,345) for girls, respectively.

Follow-up data on adult education as well as socioeconomic information for the parents of AHLS participants were obtained from the Registry of Completed Education and Degrees of Statistics Finland. **The data from Statistics Finland covered censuses**

every fifth year from 1970 to 1995 and yearly registry data from 2000 until the end of 2009. At the end of 2009, the AHLS participants were aged 29 to 43 years.

Statistics Finland performed the data linkage according to a contract specifying the rights and duties of both parties. The Institutional Review Board of Statistics Finland and the Data Protection Ombudsman approved the study protocol. A specific university and hospital review board also stated that no human rights were violated in the research protocol and approved it. Identification of the study participants was withheld from the investigators at all stages of the study.

### ***Variables from Statistics Finland***

#### *Adult education level of the survey respondents*

This is the main outcome of interest and based on the attained highest educational level of the adolescent. The exact degree codes according to the Finnish Standard Classification of Education was obtained (Statistics Finland 2018). **We classified two groups** according to years of schooling: low ( $\leq 9$  years) to middle (10-12 years) and high education ( $>12$  years).

#### *Family SES*

Family SES was based on parents' education and categorized in the same way as that of the adolescents'. Data were obtained nearest to the year when the adolescent was aged 15 years and based on both mother's and father's education levels. If parents belonged to different categories, the highest was selected. If one parent had missing data, the available parent's data was used. **The minimum age of parents was 30 years at the time their children participated in the surveys.**



## *Variables from the surveys*

### *Pubertal timing*

To obtain an indicator of pubertal timing (biological pathway), boys were asked about their age at first ejaculation while girls were asked about their age at menarche.

Classification of pubertal timing as early, average and late, followed those groupings used by Koivusilta and Rimpelä (2004). In boys, the categories were chosen to be at age 12 or earlier (early), at 13 or 14 (average), at 15 or later or if not occurred by the time of enquiry (late). In girls, the categories were at age 11 or earlier (early), at 12 or 13 (average), at 14 or later or if not occurred by the time of enquiry (late).

### *Reserve capacity*

Reserve capacity, spanning an underlying strong or weak construct, referred to a latent variable measured by nine observed variables in three distinct dimensions:

1. Perceived health dimension included three items: reported chronic disease, injury or disability that restricts daily activities (no/yes); a summary index of weekly perceived stress symptoms (stomach aches, tension or nervousness, irritability or outbursts of anger, trouble falling asleep or waking at night, headache, trembling of hands, feeling tired or weak, feeling dizzy) categorized as no symptoms, one symptom/week, 2-3/week, 4-8/week; and self-rated health categorized as very good, average/good or poor.
2. Health-promoting behaviour dimension included frequency of tooth brushing (several times a day, once a day, 1-5 times/week or less) and efficiency of physical activity. Efficiency of physical activity was measured by combining information from two variables: frequency of physical activity in leisure time and intensity of exercise (shortness of breath/sweating). This combination used

the following categories: does not exercise, exercises with low/occasional efficiency, active efficient exerciser, very active efficient exerciser.

3. Social support dimension was measured by four items: nuclear family (living with both parents or not); ease of talking about troubling issues to father, to mother and to friends (easy, difficult or very difficult). Those who did not have a father (5.2%), mother (1%) or friends (0.5%) were included in the “very difficult” category.

### *School achievement*

For school achievement, adolescents were categorized based on self-assessment of their school performance as having: highest, 2<sup>nd</sup> highest, 2<sup>nd</sup> lowest or lowest academic achievement. The 12-14-year-old respondents (all in comprehensive schools) were asked to assess whether their end-of-term school report was much better (highest), slightly better (2<sup>nd</sup> highest), average (2<sup>nd</sup> lowest), slightly poorer or much poorer (lowest) than the class average. For 16-18-year-olds, in addition to their self-assessment, school status (academic upper secondary school/vocational school/not attending school) was also used. Their achievement was classified as follows: highest (in academic upper secondary school with better performance); 2<sup>nd</sup> highest (in vocational school with better performance or academic upper secondary school with average performance); 2<sup>nd</sup> lowest (in vocational school with poor to average performance or high school with poor performance); and lowest (not at school).

### *Statistical Analysis*

We used confirmatory factor analysis (CFA) to estimate the underlying construct of “reserve capacity” and create a general, continuous latent variable from the nine

measured variables: presence of chronic disease, perceived stress symptoms, self-rated health, physical activity, regular tooth brushing, nuclear family, talking to father, talking to mother and talking to friends. We included covariances among variables within each dimension. We also fixed the value of the variance of the latent variable at one to freely estimate the factor loadings for all the variables.

To analyse the mechanisms by which SES, puberty, reserve capacity and school achievement influence adult education level, we used structural equation modelling (SEM). This enabled the inclusion of latent effects and testing of multiple pathways simultaneously (Grace and Bollen 2005). SEM is composed of both a measurement model and a structural model. The measurement model is given by CFA which shows how observed or measured variables relate to latent variables. The structural model describes the relationships among the variables, including the latent variables, through a set of regression equations (Muthén and Muthén 2012). In our study, the resulting estimates were probit coefficients which are effects on a cumulative normal function of the probabilities that the response variable equals one (Muthén and Muthén 2012). We assigned a value of one to an outcome of low to middle adult education, thus, we predict this probability given a low family SES, delayed pubertal timing, weak reserve capacity and low school achievement.

Models were fitted separately for each sex group and adjusted for both baseline age and age at follow-up. Since we wanted to assess if pubertal timing independently influenced the outcome, we initially tested for the effects of SES and puberty only (Model 1), then added reserve capacity (Model 2) and finally, school achievement (Model 3). All models were estimated using a robust weighted least squares estimator, under missing data theory which used all available data. In such analyses, missingness was allowed to be a function of the observed covariates but not the observed outcome

(Muthén and Muthén 2012). Fit of the CFA and full models (Model 3) were assessed using the root mean square error of approximation (RMSEA) and the comparative fit index (CFI). RMSEA values  $<0.08$  and  $<0.06$  imply acceptable and good fits, respectively. Similarly, CFI values  $>0.90$  and  $>0.95$  imply acceptable and good fits, respectively (Hooper et al. 2008). Mplus 7.11 was used for both CFA and SEM analyses.

## **Results**

### *Sample characteristics*

Table 1 presents the descriptive characteristics of the adolescents in the sample according to the main variables. The proportions of those who had low to middle adult education largely exceeded those who had high education among boys (70.1%) and girls (59.3%). Majority of adolescents with low to middle adult education had parents with similarly attained education. Among those with available data, average age of pubertal onset for boys was  $13.1 \pm 1.3$  years while for girls was  $12.6 \pm 1.1$  years. In terms of reserve capacity, there were higher proportions of adolescents with very good self-rated health, better health-promoting behaviours, presence of nuclear families and ease of communication with parents and friends among those with high adult education compared to those with low education. The same pattern was observed in the distribution of school achievement.

### *CFA results*

Preliminary analyses showed that all factor loadings of the nine variables were statistically significant and the positive coefficients implied that each observed variable directly relates with latent reserve capacity (Table 2). Larger factor loadings reflect

greater degree of relationship with the latent variable. Among the nine variables, perceived stress symptoms and self-rated health, both included in the perceived health dimension, contributed most to the measurement of the latent reserve capacity in both boys and girls. The estimated coefficients for the covariances indicate the relationship of variables with one another. Table 2 showed that grouped variables had statistically significant covariances implying that the observed variables were related within each dimension. RMSEA and CFI values signified good fit for our measurement models. Thus, the hypothesized reserve capacity framework in our study was consistent with observed data and provided support for our models in both boys and girls. The relationship of latent reserve capacity with other variables in the study was also illustrated in the bottom parts of Figures 2 and 3.

### *SEM Analyses*

In Table 3, models 1 and 2 disentangle the effect of the biological pathway. Results showed that delayed pubertal timing loses statistical significance only when school achievement was added into the model. On the other hand, family SES remained a strong predictor of adult education level regardless of adolescent factors added into the model.

Detailed results from SEM analyses of the full model (Model 3 in Table 3) depicting relationships among family SES, pubertal timing, school achievement and reserve capacity while additionally controlling for age at baseline and at follow-up showed that the models in both population groups fit the data well based on the presented fit indices in Figures 2 and 3. To simplify the model presentations, estimates relating to age variables and their covariances, along with covariances among adolescent pathways and among variables within same dimension of reserve capacity

were not shown. The hypothesized pathways are described in detail below.

*Hypothesis 1: family SES is related to pubertal timing, reserve capacity and school achievement*

This hypothesis was fully supported by the model in boys (Figure 2). Direct paths from family SES to the following factors: pubertal timing ( $\beta = 0.03$ ), reserve capacity ( $\beta = 0.10$ ) and school achievement ( $\beta = 0.26$ ) were all statistically significant ( $p < 0.001$ ). The results in girls (Figure 3) partially supported this hypothesis which showed only the pathways from family SES to reserve capacity ( $\beta = 0.13$ ,  $p < 0.001$ ) and from family SES to school achievement ( $\beta = 0.25$ ,  $p < 0.001$ ) as statistically significant. On the other hand, the relationship of family SES to girl's pubertal timing differed from that found in boys. Among girls, a low family SES ( $\beta = -0.02$ ,  $p = 0.05$ ) decreased the probability of delayed pubertal timing.

*Hypothesis 2: pubertal timing and reserve capacity influence adult education level*

This hypothesis was also partially supported by the results. Figures 2 and 3 illustrate statistically significant paths from reserve capacity to adult education in boys ( $\beta = 0.10$ ,  $p < 0.001$ ) and girls ( $\beta = 0.12$ ,  $p < 0.001$ ), respectively. Although, the paths from pubertal timing to adult education were not statistically significant, a positive coefficient ( $\beta = 0.01$ ) pointed a direct relationship between delayed pubertal timing and low to middle education in both boys and girls.

*Hypothesis 3: family SES relates to adult education level either directly or indirectly*

The results for boys (Figure 2) and girls (Figure 3) fully support this hypothesis as direct pathways from family SES to adult education in both boys ( $\beta = 0.16$ ,  $p < 0.001$ )

and girls ( $\beta = 0.14$ ,  $p < 0.001$ ) were statistically significant. Estimation of indirect paths in Table 4 showed that the effect of family SES on adult education is significantly mediated by reserve capacity (boys:  $\beta = 0.01$ ; girls:  $\beta = 0.02$ ;  $p < 0.001$ ) and school achievement (boys:  $\beta = 0.14$ ; girls:  $\beta = 0.12$ ;  $p < 0.001$ ) in the two groups. No mediation via pubertal timing was observed.

#### *How school achievement fits*

Direct paths from school achievement to adult education level as shown in Figures 2 and 3 were statistically significant in both boys ( $\beta = 0.52$ ;  $p < 0.001$ ) and girls ( $\beta = 0.48$ ;  $p < 0.001$ ), respectively. We also found statistically significant covariances among pubertal timing, reserve capacity and school achievement in boys, while in girls, similar statistically significant covariances existed except between pubertal timing and school achievement (Table 4).

The covariances indicate the direction of the relationship between the variables. As shown in Table 4, pubertal timing had a negative relationship with reserve capacity but positive relationship with school achievement. In our study, this means that delayed pubertal timing was related with better reserve capacity in both boys and girls but lower school achievement in boys. On the other hand, a weak reserve capacity was related with low school achievement.

## **Discussion**

### *Summary and interpretation of results*

We investigated the relationships between family SES, the intermediate pathways in adolescence and adult education. We found that family SES directly predicted the measured adolescent pathways (except biological pathway in girls) and adult education.

Reserve capacity and school achievement directly influenced adult education and mediated its relationship with family SES. Although we did not find statistical significance for the path between pubertal timing and adult education, unadjusted results suggested that delayed pubertal timing might be a risk for having low to middle adult education in both boys and girls. Additionally, we found that pubertal timing, reserve capacity and school achievement were interrelated, providing empirical evidence on how mechanisms in adolescence work to influence educational outcomes.

Clearly, our study showed that family SES predicted the adolescents' educational outcomes, either directly or indirectly via pathways of reserve capacity and school achievement. The direct relationship of SES to adult education implies that educational inequalities existed in our setting. This is comparable with analyses of more current data attributed to the rising income inequality observed in the region within recent years (OECD 2018). On the other hand, research also showed that higher levels of social mobility occur in welfare Scandinavian societies such as Finland where economic inequality gap is narrower than in other countries (OECD 2018). Indeed, we observed greater upward social mobility where children born into low SES families ended up in higher SES than their parents (Table 1).

The revealed indirect pathways of SES supported previous knowledge that SES affects life-course developments such as psychosocial, behavioural and cognitive functioning (Kroenke 2008; Conger et al. 2010). We can infer that the parents' SES influenced the reserve capacity and school achievement of the adolescents probably through family dynamics such as family stress processes, parenting practices including cognitive stimulation and parental investments for education (Conger et al. 2010; Martin et al. 2010). The adolescents with stronger reserve capacity and higher school achievement than their peers may have utilized their cognitive abilities, psychosocial



and behavioural resources to cope with academic transitions and attain higher education, and consequently, better SES in the future. **As one study proved, the pursuit of higher education, controlling for social origin, was dependent on academic motivation and abilities and subjective expectations and evaluations of return of investments on higher education (Becker and Hecken 2009).**

Like Obeidallah and colleagues (2000), we did not observe a statistically significant direct effect of family SES on menarche. On the other hand, we found that a low family SES increased the probability of delayed pubertal timing in boys. Our results supported previous findings which had documented inverse associations between SES and pubertal onset within populations (de Muinck Keizer-Schrama and Mul, 2001; Parent et al. 2003). Living in low socioeconomic conditions might delay puberty because of higher likelihood of malnutrition, acute or chronic illnesses and presence of other adverse physical or psychological conditions compared to those living in privileged environments (Parent et al. 2003). In contrast, recent evidence revealed that low family SES markedly increased rates of early puberty in both boys and girls (Downing and Bellis 2009; Sun et al. 2017) possibly through interactions with biological systems regulating pubertal timing (Sun et al. 2017) or other risk factors such as having higher body mass index (BMI) or being overweight (Downing and Bellis 2009; James-Todd et al. 2010) and stressful life events (James-Todd et al. 2010). Yet, a meta-analysis of studies among males found no significant association between family SES and pubertal timing (Xu et al. 2018). Since there is limited research on determinants of pubertal onset among boys, the processes influencing male pubertal development were much less understood (Graber 2013). We conclude that the inconsistent relationship of family SES with pubertal timing probably reflected **inherent differences in study populations which account for ethnic and geographic variations,**

gender and genetic predisposition and changes in underlying mechanisms influenced by SES to activate puberty such as intrauterine conditions, health, nutrition, stress and environmental exposures (Parent et al. 2003). Other methodological issues including differences in study designs and measurement of SES and pubertal timing indicators (Xu et al. 2017) might have contributed to this inconsistency.

In our study, low family SES increased the probability of having weak reserve capacity brought about by poor perceived health, health-promoting behaviour and social support. Our findings were congruent with previous evidence, albeit, reserve capacity was measured as purely psychosocial resources (Kroenke 2008; Matthews and Gallo 2011). According to Gallo and Matthews (2003), low-SES individuals have weaker reserve capacity due to frequent exposure to situations requiring use of their psychosocial resources and their environments which inhibit them from developing and replenishing these resources “to be kept in reserve”. While reserve capacity was initially conceptualized as a potential mediating pathway in SES-health inequalities (Gallo et al. 2009), we have shown that it also served as a pathway connecting one’s family SES to future adult education. Indeed, an indirect effect of family SES through this pathway was statistically significant in both boys and girls. We believe that dealing with school transitions, along with puberty during adolescence constantly required the use of one’s reserve capacity. This may be implied in the reported covariances between reserve capacity and pubertal timing. Thus, those with low SES and weak reserve capacity might have educational transition difficulties. It has also been suggested that individuals with weak reserve capacity may lack the coping skills needed to attain higher education (Matthews et al. 2010). The observed direct effect of reserve capacity on adult education in our study supported this logic.

Partitioning the full model showed that delayed pubertal timing, along with

family SES and reserve capacity, increased the probability of having low to middle education in both boys and girls. However, when we included school achievement in the model, pubertal timing **lost its** statistically significant effect on adult education. Instead, pubertal timing was more related with school achievement, especially in boys (based on the reported covariance), than with adult education. One study explained that pubertal status did not directly predict academic achievement but rather influenced academic motivation, which then affected academic achievement (Martin and Steinbeck 2017). Our results replicated the findings from a British cohort study which showed that late pubertal development was associated with lower levels of educational attainment, but the said association weakened when test scores at age 16 years were factored in (Koerselman and Pekkarinen 2017).

While our results in girls showed no association between pubertal timing and adult education, **evidence presented contrary findings (Hendrick et al. 2016; Gill et al. 2017). Research showed that early** maturing girls had higher probability of being a high school dropout (Cavanagh et al. 2007; Hendrick et al. 2016) or having a low-grade point average (GPA) at the end of high school (Cavanagh et al. 2007). However, these studies have suggested that beyond high school, the impact of early pubertal timing seemed to cease on educational outcomes (Copeland et al. 2010; Hendrick et al. 2016). Still, as research on educational outcomes related to pubertal timing was relatively scarce, variations in the results of these studies imply that pubertal timing coincides with cognitive development in adolescence (Viner et al. 2012; Koerselman and Pekkarinen 2017) and likely interacts with structural and behavioural mechanisms to predict educational attainment (Johnson et al. 2011).

As shown in previous studies (Slominski et al. 2011; Brekke 2015; Acacio-Claro et al. 2017), school achievement had the largest effect on adult education. This was

expected as good grades obtained in high school strongly predicted enrolment in higher education (Brekke 2015). In fact, the ground-breaking work of Entwisle and colleagues (2005) demonstrated that **as early as first grade** academic performance influenced educational attainment. Our results also pointed to the direct role of family SES in predicting school achievement. Indeed, socioeconomic disparities in school achievement probably occur because material deprivation and low SES may have reduced human capital investments of parents for their children, including cognitive stimulation, affecting their cognitive development (Kroenke 2008; Conger et al, 2010).

### *Strengths and limitations of the study*

Using large, nationwide samples with good response rates, long follow-up period and reliable register-based data allowed us to test our hypotheses about multiple direct and mediating pathways for the outcome of interest. **Since no specific set of psychosocial resources comprise reserve capacity, our study expanded the concept of reserve capacity with the addition of health-promoting behaviour. Although we needed to use proxy indicators while related studies had used psychological scales or other structured tools, because reserve capacity was conceptualized at a much later time than when our surveys were conducted. Nonetheless, we have measured a valid construct as proven by the good fit indices obtained for this latent variable. Moreover, we have obtained consistent results even with different methodologic techniques (i.e., using a longer follow-up period, different analytic procedures), adding to the reliability of our study.**

Indeed, much empirical research finds that countries with higher levels of income inequality tend to show lower levels of social mobility across generations, with more egalitarian Scandinavian countries having higher levels of social mobility than more unequal countries, such as Italy, the United

We have identified intermediate adolescent pathways (pubertal timing, reserve capacity and school achievement) which accounted for the relationship of family SES with adult education. Even though our models had good fit, we recognize that there are other structural and individual factors **unmeasured** in our study which **might be probable** pathways through which SES influences adult education. **For instance, schools, neighbourhood and peers also affect adolescents' learning potential and consequently, one's transition to adulthood (Viner et al. 2012). However, our data was not obtained from school-based nor community-based surveys, so analysing those effects were beyond the scope of our study.**

We acknowledge a limitation of our study related to the measurement of one of our main variables. While age at menarche has been extensively used in studies of pubertal timing, age at spermarche or first ejaculation may not be an accurate indicator of pubertal onset due to a high number of false negative results (Euling et al. 2008) which possibly diluted the effect of boys' pubertal timing on adult education level in our study. The use of additional puberty markers, such as Tanner staging based on appearance of secondary sexual characteristics, either through self-assessment or staging by a professional was recommended for collection of puberty data (Euling et al. 2008), although this was not possible through **mailed questionnaires. Yet, the pubertal timing ages estimated in our study population closely resemble those described in other European countries which used more accurate staging methods for the same period (de Muinck Keizer-Schrama and Mul 2001).**

### ***Conclusion***

Our study underscores the role of family SES in predicting intermediate pathways in adolescence and adult education. Moreover, we elucidated the interplay of these

pathways (pubertal timing, reserve capacity and school achievement) in influencing educational trajectories and mediating the effect of family SES on adult education. As important learning and school transitions occur during adolescence, which impact future adult education, support should be given to young people to help them adjust and cope well with various physical, behavioural and psychosocial developmental changes.

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### **Disclosure of interest**

The authors report no conflict of interest.

### **Data Availability Statement**

The data that support the findings of this study are not available for replication outside members of the research group due to contract specifications with Statistics Finland.

The computing code or syntax for analyses are not useful without the data but these may

be shared upon request. However, we welcome other researchers to join our team in Tampere for further analyses of the data.

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## Tables

Table 1. Characteristics of participants according sex group and adult education level

Personal factors, family SES, reserve capacity and school achievement in adolescence	Boys (n=17,531)				Girls (n=20,345)			
	Low/Middle		High		Low/Middle		High	
	No.	%	No.	%	No.	%	No.	%
<b>Age at baseline (years)</b>								
14	4182	34.0	1828	34.9	3624	30.1	2951	35.6
16	4412	35.9	1873	35.8	4325	35.8	2972	35.9
18	3701	30.1	1535	29.3	4107	34.1	2366	28.5
<b>Pubertal timing</b>								
Early	2731	22.2	1211	23.1	1684	14.0	1213	14.6
Average	4884	39.7	2449	46.8	7709	63.9	5327	64.3
Late	3067	25.0	1127	21.5	2565	21.3	1714	20.7
No data	1613	13.1	449	8.6	98	0.8	35	0.4
<b>Parents' education</b>								
High	1227	10.0	1659	31.7	1011	8.4	2178	26.3
Low/Middle	11063	90.0	3577	68.3	11039	91.6	6108	73.7
No data	5	0.0	0	0.0	6	0.0	3	0.0
<b>Reserve Capacity</b>								
<b>Perceived health dimension</b>								
<i>Chronic disease</i>								
No	11194	91.0	4796	91.6	10759	89.2	7521	90.7
Yes	1101	9.0	440	8.4	1297	10.8	768	9.3
<i>Perceived stress symptoms</i>								
None	6221	50.6	2647	50.6	3636	30.2	2724	32.9
1/week	2576	21.0	1119	21.4	2657	22.0	1906	23.0
2-3/week	2435	19.8	1117	21.3	3535	29.3	2426	29.2
4-8/week	1063	8.6	353	6.7	2228	18.5	1233	14.9
<i>Self-rated health</i>								
Very good	4502	36.6	2061	39.4	2882	23.9	2525	30.5
Average/good	7511	61.1	3080	58.8	8833	73.3	5606	67.6
Poor	236	1.9	77	1.5	302	2.5	144	1.7
No data	46	0.4	18	0.3	39	0.3	14	0.2
<b>Health-promoting behaviour dimension</b>								
<i>Physical activity</i>								
Very active efficient exerciser	2938	23.9	1677	32.0	1824	15.1	1805	21.8
Active efficient exerciser	3554	28.9	1735	33.2	3242	26.9	2740	33.1
Occasional/low efficient exerciser	3020	24.6	1094	20.9	3966	32.9	2513	30.3
Does not exercise	2740	22.3	719	13.7	3000	24.9	1219	14.7
No data	43	0.3	11	0.2	24	0.2	12	0.1
<i>Regular tooth brushing</i>								
Several times/day	2101	17.1	1584	30.2	5644	46.8	4601	55.5
About once/day	5967	48.5	2794	53.4	5358	44.4	3309	39.9
About 1-5 times/week or less	4151	33.8	846	16.2	1031	8.6	360	4.4
No data	76	0.6	12	0.2	23	0.2	19	0.2
<b>Social support dimension</b>								
<i>Nuclear family (with both parents)</i>								
Yes	9268	75.4	4471	85.4	8577	71.1	6838	82.5
No	2937	23.9	748	14.3	3406	28.3	1419	17.1
No data	90	0.7	17	0.3	73	0.6	32	0.4
<i>Talking about issues to father</i>								

Easy	6375	51.8	2763	52.8	4003	33.2	3026	36.5
Difficult	3762	30.6	1780	34.0	4477	37.1	3435	41.4
Very difficult/No father	1794	14.6	613	11.7	3326	27.6	1762	21.3
No data	364	3.0	80	1.5	250	2.1	66	0.8
<i>Talking about issues to mother</i>								
Easy	8454	68.7	3692	70.5	8593	71.3	5984	72.2
Difficult	2875	23.4	1246	23.8	2622	21.7	1876	22.6
Very difficult/No mother	737	6.0	232	4.4	727	6.0	401	4.8
No data	229	1.9	66	1.3	114	1.0	28	0.4
<i>Talking about issues to friends</i>								
Easy	9432	76.7	3945	75.4	10392	90.7	7540	91.0
Difficult	2093	17.0	1058	20.2	872	7.2	631	7.6
Very difficult/No friends	493	4.0	158	3.0	147	1.2	89	1.1
No data	277	2.3	75	1.4	105	0.9	29	0.3
<b><i>School achievement</i></b>								
Highest	1026	8.3	1972	37.6	1539	12.8	3611	43.6
2 <sup>nd</sup> highest	2987	24.3	2046	39.1	3718	30.8	3204	38.6
2 <sup>nd</sup> lowest	5081	41.3	1014	19.4	4453	36.9	1231	14.8
Lowest	3009	24.5	182	3.5	2212	18.4	221	2.7
No data	192	1.6	22	0.4	134	1.1	22	0.3

Table 2. Results from confirmatory factor analyses (CFA) of reserve capacity model regressed on nine observed variables presented as standardized ( $\beta$ ) coefficients

Observed variable	Boys		Girls	
	$\beta$	p-value	$\beta$	p-value
Chronic disease	0.15	<0.001	0.07	0.003
Perceived stress symptoms	0.55	<0.001	0.46	<0.001
Self-rated health	0.70	<0.001	0.58	<0.001
Physical activity	0.32	<0.001	0.33	<0.001
Regular tooth brushing	0.17	<0.001	0.17	<0.001
Nuclear family	0.18	<0.001	0.26	<0.001
Talking about issues to father	0.38	<0.001	0.40	<0.001
Talking about issues to mother	0.34	<0.001	0.36	<0.001
Talking about issues to friends	0.22	<0.001	0.23	<0.001
Covariances	Boys		Girls	
	$\beta$	p-value	$\beta$	p-value
<i>Perceived health</i>				
Chronic disease with				
Perceived stress symptoms	0.11	<0.001	0.23	<0.001
Self-rated health	0.18	<0.001	0.17	<0.001
Perceived stress symptoms with				
Self-rated health	-0.08	0.002	0.08	<0.001
<i>Health-promoting behavior</i>				
Physical activity with				
Regular tooth brushing	0.12	<0.001	0.10	<0.001
<i>Social support</i>				
Nuclear family with				
Talking about issues to father	0.33	<0.001	0.24	<0.001
Talking about issues to mother	0.10	<0.001	0.01	0.43
Talking about issues to friends	-0.03	0.071	-0.06	0.001
Talking about issues to father with				
Talking about issues to mother	0.55	<0.001	0.39	<0.001
Talking about issues to friends	0.24	<0.001	0.16	<0.001
Talking about issues to mother with				
Talking about issues to friends	0.28	<0.001	0.28	<0.001
<b>Fit indices:</b>				
RMSEA		0.04		0.03
CFI		0.97		0.97



Table 3. Direct effects of family SES and biological pathway on adult education level in a structural equation model presented as standardized ( $\beta$ ) coefficients

<b>Direct effects based on different models</b>	<b>Boys</b>		<b>Fit indices</b>	<b>Girls</b>		<b>Fit indices</b>
	SES	Puberty	RMSEA/CFI	SES	Puberty	RMSEA/CFI
Model 1 <sup>a</sup>	0.30*	0.03*	-	0.28*	0.00	-
Model 2 <sup>b</sup>	0.29*	0.05*	0.05/0.89	0.25*	0.03**	0.04/0.90
Model 3 <sup>c</sup>	0.16*	0.01	0.05/0.90	0.14*	0.01	0.04/0.91

Note: All models were adjusted for age at baseline and follow-up

\*statistically significant at  $p < 0.001$  \*\* $p = 0.001$

<sup>a</sup> Model with family SES and puberty <sup>b</sup> Model 1 plus reserve capacity <sup>c</sup> Model 2 plus school achievement

Table 4. Estimated indirect effects of family SES through adolescent pathways and the covariances among these pathways in the final structural equation model presented as standardized ( $\beta$ ) coefficients

<b>Indirect effect of family SES through</b>	<b>Boys</b>	<b>Girls</b>
Pubertal timing	0.00	0.00
School achievement	0.14*	0.12*
Reserve capacity	0.01*	0.02*
<b>Covariance between pathways</b>	<b>Boys</b>	<b>Girls</b>
Pubertal timing and reserve capacity	-0.11*	-0.12*
Pubertal timing and school achievement	0.05*	0.01
Reserve capacity and school achievement	0.35*	0.37*

Note: \*statistically significant at  $p < 0.001$

## Figures

Figure 1. Conceptual model for the relationship of family SES with adult education level through adolescent pathways (biological, reserve capacity and school achievement)

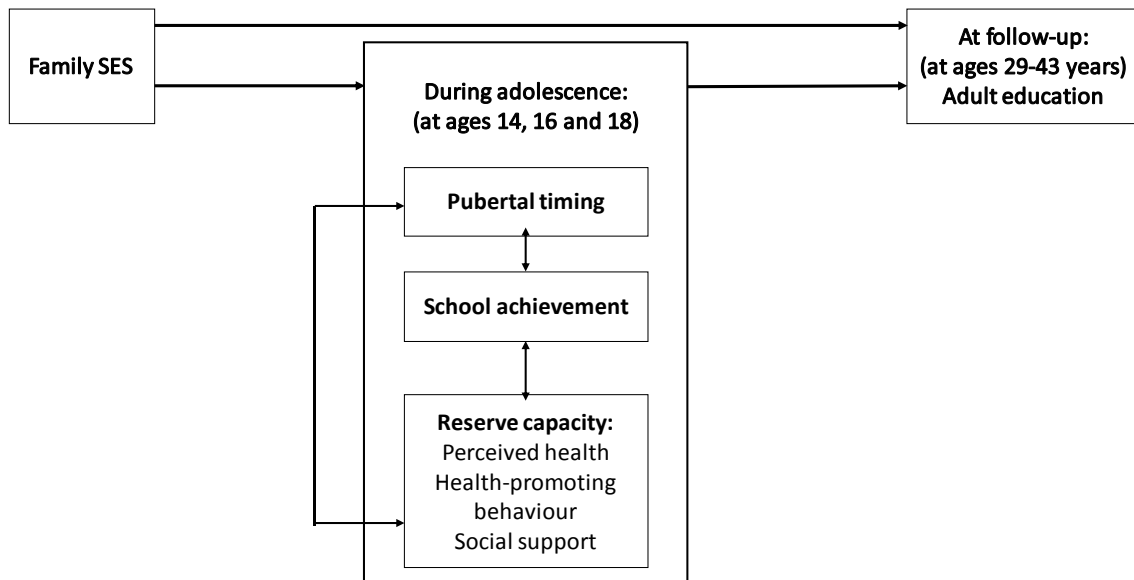
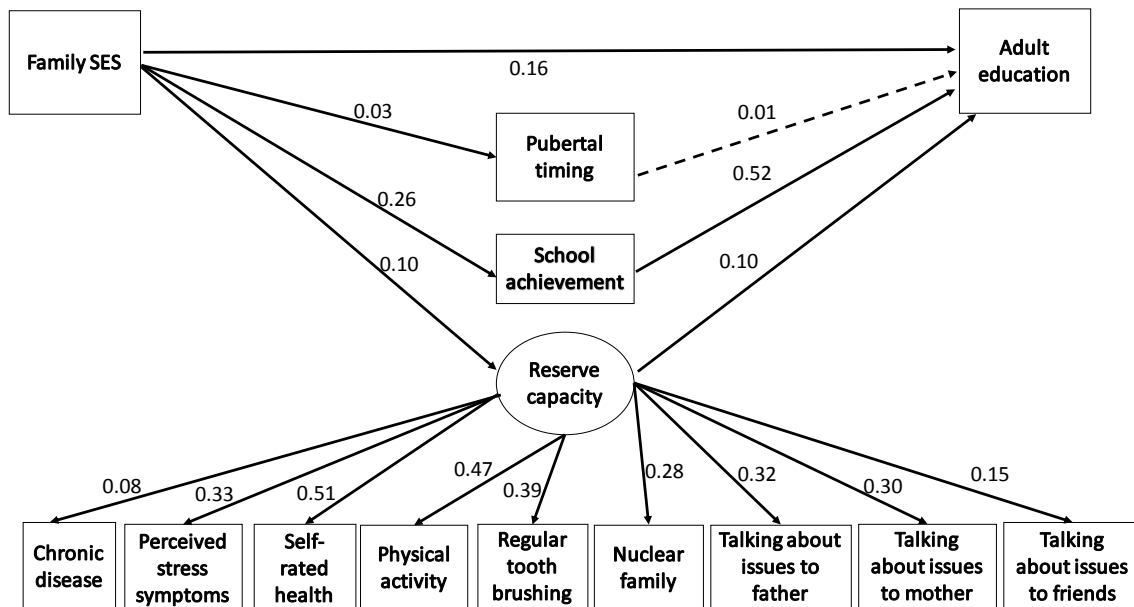
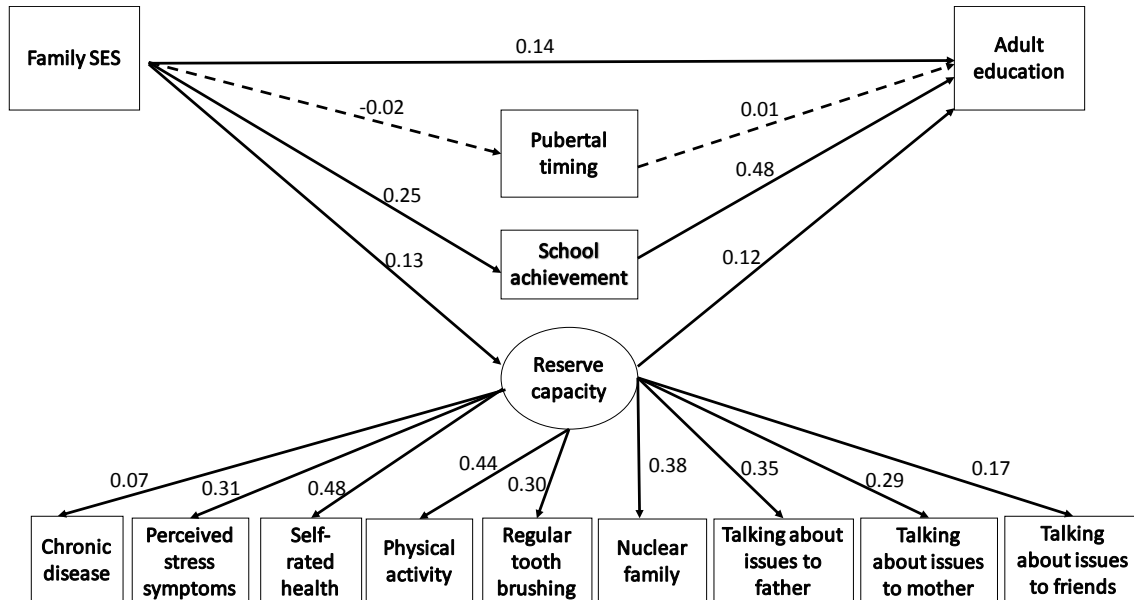


Figure 2. Boys: Structural equation model depicting relationships among family socioeconomic status (SES), pubertal timing, school achievement and reserve capacity in adolescence, and adult education level (RMSEA=0.05; CFI=0.90)



**Note:** The values along the paths are standardized regression coefficients. Solid lines indicate statistically significant paths ( $p < 0.001$ ).

Figure 3. Girls: Structural equation model depicting relationships among family socioeconomic status (SES), pubertal timing, school achievement and reserve capacity in adolescence, and adult education level (RMSEA=0.04; CFI=0.91)



**Note:** The values along the paths are standardized regression coefficients. Solid lines indicate statistically significant paths ( $p < 0.001$ ).