Tampere University

PAULYN JEAN ACACIO-CLARO

Reserve Capacity and other Adolescent Pathways in Socioeconomic Inequalities in Mortality and Education

A Three-Generation Study in Finland

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A Three-Generation Study in Finland

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PunaMusta Oy – Yliopistopaino
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Dedicated to my parents, husband and sons who are the wind beneath my wings

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I am writing this section while the world is currently going through a pandemic, presenting many uncertainties and challenging our notions of normalcy. While we look forward to the day when the world is healed, my present-day realisation is that no person, society or government can solve this alone. Helping and supporting one another is crucial in navigating this public health crisis. Indeed, that is true for all other pursuits in life, particularly, this PhD journey. I would not have been able to stay committed in studying for almost seven years without the support of these key individuals and institutions.

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Last, I offer all the glory and honour that comes with this to the One who created me. His boundless blessings and gifts enabled me to be where I am now.

Pau Acacio-Claro
April 2020

ABSTRACT

Studies have extensively shown that low socioeconomic status (SES) has cumulative adverse effects on morbidity and mortality. In addition, recent evidence showed that grandparents' socioeconomic capital could also be transmitted to their grandchildren. This implies that transmission of low SES across generations of families could perpetuate a cycle of socioeconomic disadvantage, creating further health and socioeconomic inequalities.

The strong SES-health connection was theorised to occur through different intermediate pathways in the life-course. An integrative framework emphasised the role of psychosocial resources called "reserve capacity" in causing health inequalities. This framework specifies that low SES increases one's exposure to environmental stressors and depletes "reserve capacity." Thus, negative emotional and physiological responses are triggered, affecting health via altered biological and behavioural pathways. We propose that similar processes also affect attained educational level and ultimately, one's future SES. We also extend the reserve capacity framework to include health-promoting behaviours because of their underlying psychosocial resources.

Using 1985-1995 data from the Adolescent Health and Lifestyle Surveys (AHLS) linked with data from the registries of Statistics Finland, we determined the existence of socioeconomic inequalities in mortality and education. Moreover, we assessed the roles of reserve capacity and other intermediate pathways in adolescence such as pubertal timing and school achievement in these life-course trajectories. We further studied if grandparents' socioeconomic circumstances affected their grandchildren's education.

The AHLS dataset contained information on representative samples of 12- to 18-year old Finns while the linked data from the registries updated until 2009 had relevant mortality and socioeconomic information for the AHLS participants, their parents and grandparents. Cox and multinomial logistic regression models as well as structural equation models were fitted to answer the aims of this dissertation.

In general, we found the existence of socioeconomic inequalities in mortality and education in our setting. We also found that reserve capacity and school achievement were both good and independent predictors of mortality and education. In Study I,

high reserve capacity and good school achievement reduced the risk of mortality. Among boys, these also mitigated the negative effect of low SES on mortality. In Study II, both reserve capacity and school achievement independently predicted educational attainment and mediated the effect of family SES on education. We also found out that the socioeconomic circumstances of grandparents predicted their grandchildren's educational outcomes, providing evidence on the origin of socioeconomic inequalities. The direct effects of reserve capacity and school achievement on education were further confirmed in Study III. Additionally, we have shown that pubertal timing was a potential biological pathway which influenced adolescents' educational trajectories. Moreover, indirect pathways from family SES to education existed through reserve capacity and school achievement. In this study, we have also provided evidence that a low family SES increased the probabilities of low reserve capacity, delayed pubertal timing and low school achievement.

Consistent with previous research, all the studies clearly showed that family SES directly influenced health and one's future education. Important mechanisms in adolescence, however, namely, reserve capacity, pubertal timing and school achievement, mediated the relationships of family SES with these outcomes. Even though family SES also influenced these pathways, reserve capacity and school achievement are amenable to policies and public health measures and thus, could be improved to reduce health risks, avert untimely mortality and improve the socioeconomic status of subsequent generations. Our findings suggest that strengthening multiple generations of families and schools in building reserve capacities (e.g., improving perceived health, encouraging health-promoting behaviour and providing social support), and supporting the school performance of adolescents, particularly those with disadvantaged socioeconomic backgrounds, could likely reduce socioeconomic inequalities in health and education.

TIIVISTELMÄ

Laaja joukko tutkimuksia on osoittanut, kuinka matala sosioekonominen asema (SES) heikentää terveyttä ja kuinka vaikutus kasaantuu ajan myötä näkyen sairastavuudessa ja kuolleisuudessa. Lisäksi on saatu uutta näyttöä siitä, kuinka isovanhempien sosioekonominen pääoma siirtyy jopa lapsenlapsille. Näin voidaan olettaa, että matalan sosioekonomisen aseman siirtyminen yli sukupolvien voi pahentaa sosioekonomisen huono-osaisuuden kierrettä ja siten lisätä terveydellistä ja sosioekonomista eriarvoisuutta.

Tutkimuksessa käytetyssä viitekehyksessä sosioekonomisen aseman ja terveyden välisen yhteyden ajateltiin toteutuvan elämänkulunaikaisten polkujen välityksellä. Nämä polut yhdistävä viitekehys painotti psykososiaalisten resurssien ("reserve capacity") merkitystä terveyserojen synnyn taustalla. Tässä työssä käytetty englanninkielinen käsite "reserve capacity" käännetään suomenkieliseksi sanaksi "suorituskykyvaranto". Matalan sosioekonomisen aseman nähdään lisäävän yksilön altistumista ympäristön kuormitustekijöille ja pienentävän yksilön suorituskykyvarantoa. Täten negatiiviset emotionaaliset ja fysiologiset vasteet laukeavat vaikuttaen terveyteen biologisten tai käyttäytymiseen liittyvien muutosten kautta. Samanlaisten prosessien oletetaan vaikuttavan myös yksilön aikuisena saavuttamaan koulutustasoon ja lopulta hänen tulevaan sosioekonomiseen asemaansa. Suorituskykyvarantoa koskeva viitekehys laajenee koskemaan myös terveyttä tukevaa käyttäytymistä, koska myös sen taustalla ymmärretään olevan psykososiaalisia resursseja.

Tutkimuksessa käytettiin Nuorten terveystapatutkimuksen (NTTT) aineistoja, joihin oli liitetty Tilastokeskuksen rekisteritietoa. Yhdistetyn aineiston perusteella tutkittiin kuolleisuuden ja aikuisiässä saavutetun koulutuksen sosioekonomista eriarvoisuutta. Lisäksi arvioitiin suorituskykyvarannon sekä eräiden muiden välittävien polkujen kuten puberteetin ajoittumisen sekä koulumenestyksen roolia tutkituille elämänkulunaikaisille prosesseille. Tutkimuksen kohteena oli myös isovanhempien sosioekonomisen aseman vaikutus lastenlasten sosioekonomisiin asemiin.

NTTT sisälsi koko maata edustavat aineistot 12-18-vuotiaista suomalaisista. Yhdistetty aineisto sisälsi kunkin NTTT:n kyselyihin vastanneen henkilön tietojen

lisäksi hänen vanhempiensa ja isovanhempiensa kuolemaa ja sosioekonomista asemaa koskevat tiedot vuoteen 2009 asti. Coxin regressiota, multinomiaalista logistista regressioanalyysia sekä rakenneyhtälömallinnusta käytettiin vastaamaan tutkimuskysymyksiin.

Tutkimustulokset osoittivat kuolleisuuden ja aikuisiän koulutuksen sosioekonomisen eriarvoisuuden käytetyssä aineistossa. Lisäksi osoitettiin, että suorituskykyvaranto ja koulumenestys olivat voimakkaita ja toisistaan riippumattomia kuolemanriskin ja aikuisiän koulutuksen ennustajia. Ensimmäisessä osatyössä korkea suoritusvaranto ja hyvä koulumenestys vähensivät kuolemanriskiä. Pojilla nämä lisäksi lievensivät matalan sosioekonomisen aseman vaikutusta kuolleisuuteen. Toisessa osatyössä suorituskykyvaranto ja koulumenestys toisistaan riippumatta ennustivat aikuisiän koulutustasoa, ja näiden kautta välittyi myös perheen sosioekonomisen aseman vaikutus aikuisuuden koulutustasoon. Lisäksi osoitettiin, kuinka isovanhempien sosioekonomiset olosuhteet ennustivat lastenlasten koulutusta, mikä antoi näyttöä sosioekonomisen eriarvoisuuden syntymekanismeista.

Suorituskykyvarannon ja koulumenestyksen suorasta vaikutuksesta aikuisiän koulutukseen saatiin näyttöä kolmannessa osatyössä. Osoitettiin myös, että puberteetin ajoittuminen oli eräs mahdollinen biologinen polku, joka vaikutti nuoren koulu-uraan. Lisäksi epäsuorat polut perheen sosioekonomisesta asemasta aikuisiän koulutustasoon kulkivat suorituskykyvarannon ja koulumenestyksen kautta. Tutkimus osoitti myös, että perheen matala sosioekonominen asema lisäsi matalan suorituskykyvarannon, myöhäisen puberteetin ja heikon koulumenestyksen mahdollisuutta.

Aikaisempien tutkimusten kanssa yhtäpitävästi saatiin näyttöä siitä, että perheen sosioekonominen asema vaikuttaa suoraan terveyteen ja lapsen tulevaan koulutukseen. Kuitenkin nuoruudessa tärkeät mekanismit, nimittäin suorituskykyvaranto, puberteetin ajoitus ja menestys koulussa, välittävät perheen sosioekonomisen aseman ja lopputulosten välisiä yhteyksiä. Vaikka perheen sosioekonomisen aseman merkitystä ei voida unohtaa, suorituskykyvaranto ja menestys koulussa ovat asioita, joihin poliittisin toimin ja terveyspoliittisin keinoin voidaan vaikuttaa. Näin tekemällä voidaan vähentää terveysriskejä ja kuolleisuutta ja parantaa tulevien sukupolvien sosioekonomista asemaa. Tulosten perusteella voidaan päätellä, että sosioekonomisia terveys- ja koulutuseroja voidaan todennäköisesti vähentää tukemalla suorituskykyvarannon kehittymistä sekä perheissä yli sukupolvien että kouluissa (esim. parantamalla koettua terveyttä, kannustamalla terveyttä edistävään käyttäytymiseen ja tarjoamalla sosiaalista tukea) ja

tukemalla nuorten koulumenestystä, erityisesti heidän jotka tulevat huono-osaisista perheistä.

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ABBREVIATIONS

ABP	Ambulatory blood pressure
AHLS	Adolescent Health and Lifestyle Surveys
AIC	Akaike Information Criteria
BMI	body mass index
CFA	confirmatory factor analysis
CFI	comparative fit index
CI	confidence interval
CVD	cardiovascular disease
FSM	family stress model
HBM	Health Belief Model
HR	hazard ratio
IM	investment model
OECD	Organisation for Economic Co-operation and Development
OR	odds ratio
PIAAC	Programme for the International Assessment of Adult Competencies
PISA	Programme for International Student Assessment
RMSEA	root mean square error of approximation
SCT	Social Cognitive Theory
SDG	Sustainable Development Goal
SEM	structural equation model
SES	Socioeconomic status
SRH	self-rated health
TPB	Theory of Planned Behaviour
UN	United Nations

LIST OF ORIGINAL PUBLICATIONS

This dissertation is based on the three original articles presented below. These studies are referred to in this summary as Study I, II and III, respectively.

I. Acacio-Claro PJ, Koivusilta LK, Borja JR, Rimpelä AH (2017). Adolescent reserve capacity, socioeconomic status and school achievement as predictors of mortality in Finland – a longitudinal study. BMC Public Health. 17(1), 980.

II. Acacio-Claro PJ, Doku DT, Koivusilta LK, Rimpelä AH (2018). How socioeconomic circumstances, school achievement and reserve capacity in adolescence predict adult education level: a three-generation study in Finland. International Journal of Adolescence and Youth. 23(3), 382-397.

III. Acacio-Claro, P., Koivusilta, L. K., Doku, D. T., & Rimpelä, A. H. (2019). Timing of puberty and reserve capacity in adolescence as pathways to educational level in adulthood—a longitudinal study. Annals of Human Biology, 46(1), 35-45.

1 INTRODUCTION

Socioeconomic status (SES), commonly measured by income, education, or occupation (Cheng, Goodman, & The Committee on Pediatric Research, 2015; Matthews & Gallo, 2011), influences human capital formation. This includes one's health and education, especially during adolescence and early adulthood (Bird, 2007). Indeed, evidence pointed to the impact of SES on human morbidity and mortality (Adler & Newman, 2002; Mackenbach et al., 2015; Matthews & Gallo, 2011) as well as educational attainment through cognitive development and school performance (Bird, 2007) implying socioeconomic inequalities among populations. Despite the advances in medicine and technology and changing disease pathways over time, the SES-health relationship persisted because individuals may either have or not have the socioeconomic resources to decrease their risks and protect their health. Thus, SES has been considered as a "fundamental cause" of health inequalities (Phelan, Link, & Tehranifar, 2010). Similarly, disparities in education prevailed because socioeconomic background, rather than one's own intellect and abilities, predicts academic achievement and other educational outcomes (Broer, Bai, & Fonseca, 2019).

Moreover, these health and development outcomes likely occurred due to the social stratification created by SES and the intersection of SES with various factors or conditions at the personal, family, community and national levels (Mackenbach et al., 2015; Organisation for Economic Co-operation and Development [OECD], 2018; Phelan et al., 2010). Among these factors identified as underlying mechanisms linking SES to health included health behaviour, differential access to health care services and exposures to environmental hazards and conditions (Adler & Newman, 2002). On the other hand, the relationship of SES with education was attributed to the use of economic, cultural and social capital as well as intrinsic characteristics of educational systems (Broer et al., 2019).

Researchers have also proposed that multiple psychosocial factors within the neighborhood, family and individual levels were shaped by SES and linked to biological mechanisms that influence health (Chen & Miller, 2013). Likewise, life-course studies have recognised the influence of psychosocial mechanisms on youth development, including socioeconomic trajectories (Kroenke, 2008; Murasko, 2007).

With growing evidence on the reciprocal relationship between health and education, particularly in the context of youth development (Brekke, 2015; Freudenberg & Ruglis, 2007; Koivusilta, Rimpelä, & Vikat, 2003; Spittel, Riley, & Kaplan, 2015), understanding which pathways connect SES to both health and educational trajectories is crucial for improving both outcomes, and maximising healthy transitions into adulthood.

In recent years, the "reserve capacity" framework, named after a concept in aging literature (Gallo & Matthews, 2003), integrated the psychosocial pathway with bio-behavioural pathways to understand the SES-health relationship and its potential effect on socioeconomic outcomes such as education and income (Matthews & Gallo, 2011; Matthews, Gallo, & Taylor, 2010). Reserve capacity refers to aggregate intrapersonal and interpersonal psychosocial resources that individuals maintain and use in response to stress. Specific examples of interpersonal resources are social support and integration while intrapersonal characteristics include self-efficacy, mastery, or a sense of perceived control (Matthews & Gallo, 2011). Based on the framework, individuals living in low SES environments have fewer psychosocial resources kept in reserve; hence, one's "reserve capacity" may not be replenished in time to buffer repeated stressful situations. Consequently, low SES individuals are more likely to experience negative emotions and psychological distress, which in turn, influence intermediate behavioural and physiological pathways leading to poor health (Gallo & Matthews, 2003; Matthews et al., 2010).

High reserve capacities may decrease morbidity and mortality risks by regulating stress response, promoting positive emotions and facilitating adaptive coping which dampen pathogenic processes (Chen & Miller, 2012; Gallo, Espinosa de los Monteros, & Shivpuri, 2009). Low SES individuals with strong control beliefs and social connectedness had health outcomes comparable to those of higher SES individuals (Chen & Miller, 2012; Gallo & Matthews, 2003; Gallo et al., 2009; George, 2013). On the other hand, increased risk to stroke and mortality were seen in those with reduced social resources (Gallo & Matthews, 2003; Holt-Lunstad, Smith, & Layton, 2010) and perceived low control (Bosma, Schrijvers, & Mackenbach, 1999). Self-efficacy was shown to modify the associations between SES and perceived health and SES and waist-to-hip ratio, a measure of obesity and a risk factor for cardiovascular disease (CVD) and type II diabetes (Gallo & Matthews, 2003).

Studies have also shown that psychosocial resources, namely, coping planning (Pakpour & Sniehotta, 2012; Scheerman et al., 2016), perceived behavioural control (Pakpour & Sniehotta, 2012) and self-efficacy (Feltz & Magyar, 2006; Robbins,

Pender, Ronis, Kazanis, & Pis, 2004; Scheerman et al., 2016; Schwarzer & Luszczynska, 2006) explained engagement in health-promoting behaviours. Thus, we have expanded the reserve capacity definition to include health-promoting behaviours. We now adopt the reserve capacity framework and propose that similar processes also affect educational attainment, a measure of one's future SES.

It was suggested that individuals with high reserve capacity gain the coping skills necessary to attain higher education while those with low reserve capacity may lack these skills and attain lower education and income (Matthews et al., 2010). Locus of control significantly increased the probability of continued schooling after age 16 years, and of obtaining a degree by age 29 years (Murasko, 2007). Academic self-efficacy was also predictive of educational expectations; hence, students with high academic self-efficacy were more likely to obtain higher degrees than those with low self-efficacy (Merritt & Buboltz, 2015).

In addition, research has shown that SES can be transmitted across generations of families (Chan & Boliver, 2013; Chan & Boliver, 2014; Erola & Moisio, 2007; Møllegaard & Jæger, 2015), which could perpetuate a cycle of further socioeconomic and health inequalities. The intergenerational transmission of SES puts those with low SES at a great disadvantage. Thus, it is important to explain the origin of these inequalities and understand the role of reserve capacity and other processes influencing both SES and health. Such knowledge will help in addressing relevant SES components and the pathways by which they influence health and education.

Acknowledging all these issues, we apply a life-course approach to examine how socioeconomic origins influence one's health and educational trajectories. Life-course studies posit that "socially-patterned" early life exposures operate via interrelated pathways to affect later life outcomes (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). We further enrich this approach by adding a multigenerational lens on socioeconomic background, potentially elucidating the source of inequalities and providing proof of cumulative effect. Our study aims to determine the effect of family SES on later mortality and education of adolescents as well as the pathways by which family SES influence these outcomes. Specifically, we assess the roles of adolescent factors, namely, reserve capacity, school achievement and puberty in socioeconomic inequalities in mortality and education. We also assess if grandparents' socioeconomic circumstances affect their grandchildren's educational attainment, an indicator of future SES.

2 LITERATURE REVIEW

2.1 Socioeconomic inequalities

According to Phelan and colleagues (2010), "money, knowledge, power, prestige and beneficial social connections" were resources inherently related to socioeconomic status (SES). When there are differences in the distribution of these resources among individuals or societies, socioeconomic inequalities are said to have occurred. Then, these affect multidimensional outcomes linked to one's sense of well-being (Perrons & Plomien, 2010). For example, large income disparities, even among high income countries, limited the educational opportunities and social mobility of its population (OECD, 2018). Research has also shown that unequal societies were characterised by lower levels of economic growth and greater poverty, higher crime rates, poorer health and social or political exclusion compared to egalitarian or more equal societies (Perrons & Plomien, 2010). Thus, ensuring equal opportunity and reducing inequalities of outcome as well as eradicating extreme poverty were included among the Sustainable Development Goals (SDGs) of the United Nations (UN) to be achieved by 2030 (United Nations Development Programme [UNDP], 2019).

Socioeconomic inequalities may be measured in absolute or relative terms. Absolute inequalities refer to the differences in rates or means between socioeconomic groups for a certain outcome while relative inequalities refer to disparities in the ratio of rates for a certain outcome across socioeconomic groups (van Zon, Bültmann, Mendes de Leon, & Reijneveld, 2015). Whereas socioeconomic inequalities have been reflected in various areas of well-being, health and educational inequalities merit special consideration because of their intersecting impact on the developmental pathways of children through adulthood (Lopez & Gadsden, 2016).

2.1.1 Measurement of socioeconomic status

Central to the issue of socioeconomic inequalities is the measurement of SES. There are individual measures which represent the social or economic component of SES or both (Matthews & Gallo, 2011). Social-based or status-based measures indicate

the individual's position within a social hierarchy such as occupational classification systems or "subjective social status" which captures an individual's perception of own status in relation to others (Cheng et al., 2015; Matthews & Gallo, 2011). On the other hand, economic-based or resource-based measures indicate access to material and social goods or assets such as educational attainment, home ownership, household income (Matthews & Gallo, 2011); or possession of certain household items as proxy for family wealth (Broer et al., 2019; OECD, 2018).

Commonly used individual measures which account for both the social and economic components of SES are income, education and occupation (Cheng et al., 2015; Conger, Conger, & Martin, 2010; Matthews & Gallo, 2011). Although these three measures were moderately correlated, each has its own strengths and weaknesses. Moreover, each measure contributes unique socioeconomic information (Cheng et al., 2015; Matthews & Gallo, 2011) and reveals various facets of family background (Broer et al., 2019).

For instance, education is regarded as the most stable SES marker because it addresses reverse causation issues, i.e., education precedes poor health outcomes among older ages (Mackenbach et al., 2015). Moreover, it can be used for those who are not in the labor force, e.g., mothers who opted to care for young children full-time or retired individuals (Matthews & Gallo, 2011). Additionally, it can influence future occupational opportunities and earning capacities (Adler & Newman, 2002) but this may vary across different population groups (Matthews & Gallo, 2011). On the other hand, it does not provide information on early educational experiences (Adler & Newman, 2002), as well as educational quality (Matthews & Gallo, 2011), which may also be important predictors of health and development.

Income provides information about purchasing power which can be used to access material and social goods such as health care, schooling, good housing and nutrition, among others (Adler & Newman, 2002). Data, however, on income may be inaccurately reported or unavailable and may not be applicable among those who are not in the labour force (Matthews & Gallo, 2011).

Occupation can be measured simply, using dichotomous categories of either being employed or unemployed, or hierarchically based on occupational classes (Adler & Newman, 2002). Among the employed, occupations vary considerably in terms of "prestige, qualifications, rewards, and job characteristics" which confer health risks differently (Adler & Newman, 2002). On the other hand, among the unemployed, no distinction is made between those who were not in the labour force by choice and those who lost their jobs and were unable to get employed. Objective rankings of occupation may pose a challenge as occupational classes differ across

sociodemographic groups (Matthews & Gallo, 2011). Also, occupation does not measure wealth or the abundance of economic resources (Cheng et al., 2015)

Neighbourhood or contextual SES measures have also been used as proxy indicators for individual SES (Cheng et al., 2015). These include SES characteristics at the neighbourhood or community level as well as aggregate SES of individuals living in the area (Matthews & Gallo, 2011). These measures provide additional information as clustering of sociodemographic characteristics such as income level and ethnicity were observed according to residential areas (Matthews & Gallo, 2011). Similarly, social networks and health behaviour (Moore & Littlecott, 2015) as well as policies and available resources (OECD, 2018) vary by contextual SES as shown by school level affluence (Moore & Littlecott, 2015). Also, poorer areas were found to have greater environmental health risks, security issues and lower social capital (Adler & Newman, 2002).

2.1.2 The SES-health gradient

Health inequalities or disparities in health outcomes due to SES were generally depicted by a socioeconomic gradient in health. The SES-health gradient occurs when population groups in lower SES levels have worse health outcomes compared to those placed above them in the socioeconomic hierarchy (Kawachi, Subramanian, & Almeida-Filho, 2002). Increasing rates of morbidity and mortality were observed for groups with decreasing SES levels (Adler et al., 1994; Matthews & Gallo, 2011).

Historically, remarkable evidence of the SES-health gradient was seen in the Whitehall study of British civil servants where relative risks (RR) of mortality over 10 years significantly increased with decreasing occupational ranks: RR of 1.6 for professional-executive grades, 2.2 for the clerical grades, and 2.7 for the lowest grades consisting of unskilled workers (Adler et al., 1994). Similarly, a Swedish study found absolute and relative inequalities in all-cause mortality based on family SES at birth indicated by parental occupation (Juárez, Goodman, & Koupil, 2016). The authors observed lower mortality rates with increasing hierarchy of parental occupation: higher and intermediate non-manual occupations, entrepreneurs and farmers (8.98 per 1000); lower non-manual and skilled manual occupations (10.00 per 1000); and unskilled manual occupations (10.29 per 1000) (Juárez et al., 2016). Hazard ratio (HR) estimates adjusted for sex and birth year also had a slight gradient increase (implying decreased survival) from lower non-manual and skilled manual occupations (HR 1.14, 95% CI 1.07 to 1.22) to unskilled manual occupations (HR

1.19, 95% CI 1.12 to 1.25) relative to higher occupational levels. In Finland, greater absolute differences in mortality rates and disparities in relative risks of mortality were observed among children and young adults based on parental education (Remes, Martikainen, & Valkonen, 2010). Among American adults aged 50 years and older, of different racial origins, absolute health inequalities were shown as first incident stroke rates increased with both lower childhood SES (based on parental education) and lower adulthood SES (own educational attainment) (Liu et al., 2013). The same study also demonstrated graded increases in relative risks of incident stroke with decreasing childhood and adulthood SES. This implies cumulative and combined effects of SES on health (Liu et al., 2013).

The same gradient also exists at a macro level. Greater national wealth was associated with better health outcomes in both children and adults (Viner et al., 2012). Krieger and colleagues (2010) showed declining age-standardized breast cancer incidence rates among older women who resided in high-income counties in America. In Europe, larger relative differences between mortality due to preventable and non-preventable causes were seen in Central and Eastern Europe, where countries had considerable resource inequalities, compared to those in the Nordic countries and continental Europe where resource inequalities were minimal (Mackenbach et al., 2015). Similarly, income inequality increased the association of health and SES measures of social origins such as parental education and occupation across Europe. This suggests that higher inequalities resulted in poorer public health (Chauvel & Leist, 2015). Nations with high completion rates of at least a secondary education also had reduced health and behavioural problems among young people (Freudenberg & Ruglis, 2007; Viner et al., 2012).

This SES-health gradient, however, is not constant during the life-course. It is evident during childhood (Bammann et al., 2016; Ip et al., 2016) and adulthood (Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002; Liu et al., 2013). Though, mixed results have been documented in adolescence and youth (Chen, Martin, & Matthews, 2006; Goodman, 1999; Piko & Fitzpatrick, 2007; Remes et al., 2010). For instance, expected SES-health gradients for acute conditions either appeared or disappeared only in adolescence (Chen, et al., 2006; Goodman, 1999). Studies have suggested that causal mechanisms must be related to adolescent development and have differed from childhood or adult processes which cause these conditions (Chen, et al., 2006; Goodman, 1999). In addition, these findings also supported the "equalisation" theory which proposed that SES differences in health might level out during adolescence and youth due to the decreasing effect of family background such as

social class and increasing influences from the community such as school and peer groups (Green, 2013; West, 1997).

Health inequalities within and between countries resulted from unequal distribution of resources due to "poor social policies and programmes, unfair economic arrangements, and bad politics". All these intersect with inherent differences in one's experiences of daily living conditions (Lopez & Gadsden, 2016). Since the burden is particularly great for those with low SES, addressing all SES components and the pathways by which they influence health should provide the greatest positive impact on health outcomes of this group (Adler & Newman, 2002).

2.1.3 Educational inequalities as outcomes

Socioeconomic inequalities in education exist when differences in educational outcomes are more strongly related to the students' socioeconomic background than their academic efforts, interests or study habits (OECD, 2018). Based on the Organisation for Economic Cooperation and Development's (OECD) Programme for International Student Assessment (PISA) and the Programme for the International Assessment of Adult Competencies (PIAAC), inequalities can occur in three types of educational outcomes. These were cognitive achievement, social and emotional well-being and educational attainment; outcomes covering educational inequalities during the life-course (from childhood to adulthood) which predict the student's status after secondary education and subsequent entry into the labour market (OECD, 2018).

Several forms of capital, inherent to family SES, were proposed to impact educational outcomes. Firstly, economic capital or availability of financial resources from parents can support children's extracurricular activities, enhancing their learning and expanding their social networks (Broer et al., 2019). On the other hand, lack of economic capital resulted in educational inequalities as seen among German students. Despite similar levels of previous school performance, children of middle and working classes, due to expected costs of university education, were more likely to discontinue tertiary schooling compared to children of upper classes (Becker and Hecken, 2009). Secondly, cultural capital as expressed in a student's physical appearance, language style, attitudes toward the school and teachers or social abilities (Broer et al., 2019) as well as involvement in intellectually and culturally stimulating activities (OECD, 2018) may perpetuate educational inequalities. Certain cultural resources may be favoured by school systems and place students at greater

advantages over their peers (Broer et al., 2019). PISA measured cultural capital through the students' participation in various cultural activities and cultural communication with parents such as discussion of social and political issues. The assessment showed that university completion rates varied by frequency of cultural activity. Children with tertiary-educated parents had more access to cultural activities and engaged in more cultural conversations with their parents than children from less-educated families (OECD, 2018). Lastly, social capital through supportive relations and strong social networks may explain higher educational achievements of high SES students (Broer et al., 2019). One example of this is parental involvement in school and parents' relations with teachers. Sociologists have noted that working class parents with less education and fewer academically- and professionally-oriented social resources compared to middle class parents tend to have less involvement with the academic activities of their children and relate differently with teachers. This creates differences in the educational experiences of their children (Thirutnurthy, Kirylo, & Ciabattari, 2010). These economic, cultural and social capital also vary with school-level SES such that more advantaged schools have better policies, resources, practices and characteristics than disadvantaged schools. These factors foster environments conducive to learning and lead to better academic outcomes (OECD, 2018).

Educational inequalities also result when educational systems create social stratification as seen in heterogenous systems. For instance, heterogenous schools exhibit variations in the socioeconomic composition of their students, teacher quality, school policies, resources, funding and fees (Broer et al., 2019). This is unlike in Nordic countries where schools tend to be homogenous and more inclusive, providing equal opportunities to students regardless of their geographical location, SES, gender and ethnicity (OECD, 2018). On the other hand, decentralised educational systems where local municipalities have autonomy in managing both privately- and publicly- funded schools contribute to school heterogeneity resulting in educational inequalities. Indeed, countries with decentralised educational systems such as Hungary and Lithuania were shown to have increased the achievement gaps in either mathematics or science between low and high SES students from 1995 to 2015 (Broer et al., 2019). Notably, countries with reduced investments in education also increased the SES-mathematics achievement gap (Broer et al., 2019). These findings suggest that macro level factors, along with individual or contextual SES indicators, contribute to the creation of educational inequalities within and between populations (Broer et al., 2019).

2.2 Reserve capacity framework

The "reserve capacity" framework, named after a concept in aging literature (Gallo & Matthews, 2003), integrated psychosocial, biological and behavioural pathways as an important cause of socioeconomic inequalities in health (Gallo, 2009; Gallo et al., 2009; Matthews & Gallo, 2011). It offered an empirical and novel approach to explain the SES-health gradient (Gallo & Matthews, 2003) and added another perspective on how it could potentially influence socioeconomic outcomes such as education and income (Matthews et al., 2010). Promising evidence on some aspects of the framework, particularly the SES, stress and psychosocial functioning links, were derived from studies on children and adolescent populations (Matthews et al., 2010).

2.2.1 Origins and theories

Research has attributed the socioeconomic disparities in health to varied environmental, behavioural and psychological factors although, none of these singly and fully explain the SES-health gradient (Adler & Newman, 2002; Gallo and Matthews, 2003). On the other hand, studies which examined the contribution of psychosocial factors showed promising results and consistently linked low SES and poor physical health (Matthews & Gallo, 2011). A broad, organising framework was developed to illustrate how SES and these psychosocial factors, namely: stress, resources and negative emotions, are related with intermediate bio-behavioural pathways and health (Gallo & Matthews, 2003; Gallo et al., 2009). This framework (Figure 1) was termed "reserve capacity," named after a concept in aging literature (Gallo & Matthews, 2003).

The reserve capacity model specified that individuals living in low SES environments have fewer psychosocial resources kept in reserve for dealing with stressful situations (arrow D). This bank of psychosocial resources termed "reserve capacity" may consist of interpersonal resources such as social support and integration as well as intrapersonal characteristics such as self-efficacy, mastery or a sense of perceived control (Matthews & Gallo, 2011). As low SES typically increased one's exposure to stress and adversity (arrow A), reserve capacity may not be replenished in time to buffer these stressors (arrow E). Thus, negative emotions and cognitions intensify (arrows B, F) and affect health outcomes (arrow L) via intermediate behavioural and physiological pathways (arrows C, G, K) (Gallo &

Matthews, 2003). The potential bidirectional links (arrows H, I, J) of emotions and cognitions with reserve capacity and with SES are also recognised in this framework (Gallo & Matthews, 2003).

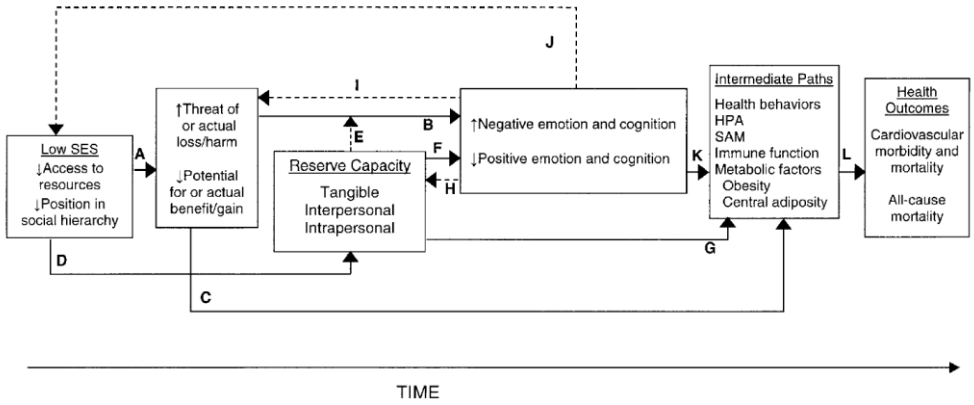

Figure 1. The reserve capacity model showing sequential associations from SES, psychosocial and other intermediate pathways to health outcomes. Solid lines indicate direct influences while dashed lines indicate possible reciprocal influences.

Reproduced from Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? by Gallo and Matthews. Copyright 2003 by the American Psychological Association, Inc.

Empirical evidence has shown that those with low SES reported lower levels of resilient intrapersonal resources such as self-efficacy, mastery, or a sense of perceived control as well as poorer social support or integration than high SES individuals (Gallo & Matthews, 2003). Low SES environments may negatively shape psychological and social functioning due to the interaction of different factors at multiple levels: individual, family and neighbourhood (Chen & Miller, 2013). For instance, low SES neighbourhoods were characterised by greater exposure to violence and threats to safety, altering social relationships within the neighbourhood leading to lower levels of social capital or poor collective trust and cohesion (Chen & Miller, 2013). Families living in such neighbourhoods exhibited less nurturing parenting approaches, had more stressful and conflictual relationships, greater material insecurities and more instability in their daily routines (Chen & Miller, 2013; Matthews & Gallo, 2011). Thus, at the individual level, a low SES person may experience more negative emotions such as depression and anxiety and cognitions such as hostility and pessimism compared to a high SES person. These individual factors then affect one's biological mechanisms and behavioural engagement (Chen & Miller, 2013), eventually influencing health outcomes.

Gallo and Matthews (2003) affirmed that although they focused on the psychosocial perspective, these were not the exclusive pathways through which morbidity and mortality occur. They acknowledged other mechanisms such as how low SES in early life poses accumulated health risks over the life-course; how environmental factors could directly affect biological and behavioural processes; and how some demographic variables alter the SES-health associations in populations (Matthews & Gallo, 2011). Tests of this framework, however, showed that psychosocial factors were important mediators of the association between SES and health. This is likely due to connections with biological and behavioural pathways predicting poor health; thus, these could potentially be targeted to reduce health inequalities (Gallo & Matthews, 2003; Matthews et al., 2010; Moor et al., 2014).

2.2.2 Links with biological pathways

These psychosocial resources demonstrated clear links with biological pathways (Chen & Miller, 2013). Various assessments of the underlying mechanisms through which SES affects physical health uncovered the role of psychosocial resources in altering physiological and biological markers involved in the manifestation and progression of disease (Matthews & Gallo, 2011). Evidence was based on the allostatic load model which suggests that as the body tries to restore stability in response to stress, physiological changes occur which cumulatively dysregulate multiple organ systems and increase health risks. Allostatic load was commonly measured with neuroendocrine and metabolic markers, cardiovascular reactivity and more recently, inflammatory markers (Matthews & Gallo, 2011).

For instance, low levels of family support predisposed individuals to greater inflammatory responses, found to be involved in pathogenic processes for many infectious and chronic diseases including clinical asthma and cardiovascular diseases (Chen & Miller, 2013). Indeed, increased risk to stroke and mortality were seen in those with reduced social resources (Gallo & Matthews, 2003; Holt-Lunstad et al., 2010). In addition, perimenopausal women with decreased levels of optimism, self-esteem and social support had increased depressive symptoms, anger and tension. These directly correlated with metabolic abnormalities such as impaired glucose and lipid metabolism, central adiposity and hypertension, increasing their risks for CVD morbidity and mortality (Matthews, Räikkönen, Gallo, & Kuller, 2008).

In contrast, high reserve capacities decreased morbidity and mortality risks by regulating stress response, promoting positive emotions and facilitating adaptive

coping which dampen pathogenic processes (Chen & Miller, 2012; Gallo et al., 2009). Persistence in terms of optimism and hopefulness was associated with lower levels of inflammatory markers, decreasing one's risk for heart disease and all-cause mortality (Chen & Miller, 2013). Women in low status jobs with perceptions of high job control had ambulatory blood pressure (ABP) levels, a strong predictor of CVD, comparable to those of women in high status jobs (Gallo, 2009). Thus, low SES individuals with strong control beliefs and social connectedness had health outcomes similar to those of higher SES individuals (Chen & Miller, 2012; Gallo & Matthews, 2003; Gallo et al., 2009; George, 2013).

The findings that socioeconomic and other early life conditions affect brain development also imply that differences in brain structures lead to differences in regulation of emotions and stress appraisals, signaling neural responses which may shape health risks of individuals (Matthews & Gallo, 2011; Matthews et al., 2010). As research on this field is relatively new, there is rich potential for explaining other psychobiological links which may further inform the SES-health relationship. Chen and Miller (2013) also recommended targeting a specific disease and discovering the underlying psychosocial and biological processes involved in its pathogenesis to clarify the stepwise fashion by which health is affected, from its social to its physical aspect.

2.2.3 Links with behavioural mechanisms

Research has shown that socioeconomic conditions directly influence the adoption of health behaviours such that those born in low SES families tend to have risky health-related behaviours. Conversely, those born in high SES families tend to engage in health-promoting behaviours (Bricard, Jusot, & Tubeuf, 2010; Chen & Miller, 2013; Moor et al., 2014; Moore & Littlecott, 2015; Mulder, de Bruin, Schreurs, van Ameijden, & van Woerkum, 2011; Tubeuf, Jusot, & Bricard, 2012). Health-related behaviours or lifestyle factors refer to individual actions and habits which maintain, restore or improve health (Schwarzer & Luszczynska, 2006). These can either be health-compromising such as lack of exercise, eating unhealthy diets, substance use, and non-compliance to treatment; or, health-promoting such as oral hygiene, regular exercise, fruit and vegetable consumption, and compliance to treatment, to name a few (Schwarzer & Luszczynska, 2006).

Apart from the social factors which determine health behaviour, the performance of specific behaviours was theorised to depend on psychosocial resources. There are

three main theories in psychology which explain how psychosocial factors can induce behavioural changes. Firstly, the Health Belief Model (HBM), developed in the 1950s, proposed several psychosocial constructs involved in an individual's engagement in health-promoting behaviours. These include perceptions of one's susceptibility to a disease or condition as well as its severity; perceptions about the benefits and barriers of a health action; and self-efficacy (Glanz, Rimer, & Viswanath, 2008). Self-efficacy refers to a person's confidence to perform the behaviour (Conner et al., 2013) and is also synonymous with behavioural control (Schwarzer & Luszczynska, 2006). Secondly, Bandura's Social Cognitive Theory (SCT) proposed that cognitions such as self-efficacy influences behavioural change by overcoming challenges and setting goals (Conner et al., 2013), exercising "control over one's environment and behaviour" (Schwarzer & Luszczynska, 2006). Thus, self-efficacy determines how much effort one exerts to perform and continue a behaviour despite associated challenges which may lower one's motivation (Schwarzer & Luszczynska, 2006). Lastly, the Theory of Planned Behaviour (TPB) posited that intention is the main determinant of behaviour and it depends on one's own attitude toward the behaviour, perceived approval of performance of the behaviour from important people (subjective norms) and the degree to which one perceives control (perceived behavioural control) over factors that may affect intention or behaviour (Glanz et al, 2008; Schwarzer & Luszczynska, 2006). Both SCT and TPB view intention and self-efficacy as direct determinants of behaviour (Conner et al., 2013).

Studies have shown that increased self-efficacy predicted performance in sports and engagement in physical activity (Feltz & Magyar, 2006; Robbins et al., 2004) as well as oral hygiene behaviour (Scheerman et al., 2016). General self-efficacy beliefs or broad optimism in one's competence to deal with stressful situations were associated with perceptions of good health and intentions to practice healthy lifestyle (Schwarzer & Luszczynska, 2006). Coping planning and perceived behavioural control predicted dental brushing behaviour (Pakpour & Sniehotta, 2012; Scheerman et al., 2016). On the other hand, lower levels of self-efficacy were associated with risky sexual behaviours and addictive behaviours such as smoking and drinking (Schwarzer & Luszczynska, 2006).

As adverse health and other development outcomes could likely be prevented by engagement in positive health behaviours, it is important to target the factors affecting behavioural development. Effective interventions should aim to enhance one's psychosocial resources, particularly, general self-efficacy and optimistic beliefs which provide a sense of personal competence to the individual. In turn, these are

likely to enable them to reduce risky behaviours and adopt healthier lifestyles (Schwarzer & Luszczynska, 2006).

2.2.4 Links with educational outcomes

Aside from health outcomes, reserve capacity may potentially influence socioeconomic outcomes such as education and income. It was proposed that individuals with high reserve capacity gain the coping skills necessary to attain higher education while those with low reserve capacity may lack these skills and attain lower education and income (Matthews et al., 2010).

Locus of control, a psychosocial resource which refers to the degree that an individual believes he has control over life events, significantly increased the probability of continued schooling after age 16 years and of obtaining a degree by age 29 years (Murasko, 2007). Although the exact explanatory mechanisms for these relationships were not determined, locus of control was deemed related to one's stress-coping abilities, probably increasing school productivity and ensuring academic success (Murasko, 2007).

Academic self-efficacy, indicating beliefs about ability to succeed in school, predicted educational expectations, hence students with high academic self-efficacy were more likely to obtain higher degrees than those with low self-efficacy (Merritt & Buboltz, 2015). The authors concluded that these students had developed dedicated studying skills and abilities for self-regulated learning and were more likely to pursue higher studies than students with low academic self-efficacy (Merritt & Buboltz, 2015).

Dispositional optimism, characterised by having favorable expectations for the future, and life satisfaction, defined as judgements about life in general, were another set of psychosocial resources found to be associated with education, income and social mobility (Boehm, Chen, Williams, Ryff, & Kubzansky, 2015). Those with higher education, occupational class and income had higher optimism and greater life satisfaction than those with less education, manual occupations and lower income (Boehm et al., 2015). In terms of social mobility, those with persistently high social status across generations were significantly more optimistic compared to the other groups while those persistently high and upwardly mobile were more satisfied compared to individuals whose social statuses were downwardly mobile and persistently low (Boehm et al., 2015). Boehm and colleagues (2015) surmised that

these psychological resources were associated with social structures and had cognitive links, thus, influencing educational outcomes and consequently, adult SES.

While Gallo and Matthews (2003) focused on physical health outcomes for testing their reserve capacity framework, they acknowledged that reserve capacity presented potential pathway effects for other life-course trajectories. Indeed, they emphasised that future research should use developmental and life-course approaches to capture the mechanisms involved in SES-health inequalities which may also persist across generations (Gallo & Matthews, 2003; Matthews & Gallo, 2011; Matthews et al., 2010). The findings of Murasko (2007) which suggested cumulative and pathway effects of psychosocial resources on both health and education using a life-course model provided novel contributions to this field. These also posed important implications for interventions designed to improve health and development outcomes.

2.2.5 Developmental and life-course perspective

The role of reserve capacity was initially assessed in studies of health inequalities among adults using cardiovascular morbidity outcomes and all-cause mortality (Gallo & Matthews, 2003; Matthews et al., 2010). The proponents, however, recommended that the life-course lens be applied because of varying degrees of SES-health relationships across time. A strong SES-health gradient was documented during childhood (Bammann et al., 2016; Ip et al., 2016; Gallo & Matthews, 2003). On the other hand, the inconsistent SES-health relationship observed in adolescence and youth (Chen et al., 2006; Goodman, 1999; Piko and Fitzpatrick, 2007; Remes et al., 2010) was probably influenced by significant life transitions occurring during this period (Johnson, Robert, & Elder, 2011).

A life-course perspective in research uses a combination of developmental, psychological, cognitive, biological and epidemiological concepts and processes in understanding human health and development (Kuh et al., 2003). It aims to "build and test theoretical models that postulate pathways" and interrelationships between exposures temporally preceding outcomes across the life-course (Kuh et al., 2003). Hendricks (2012) described three models (Figure 2) which explain how early life experiences shape later outcomes.

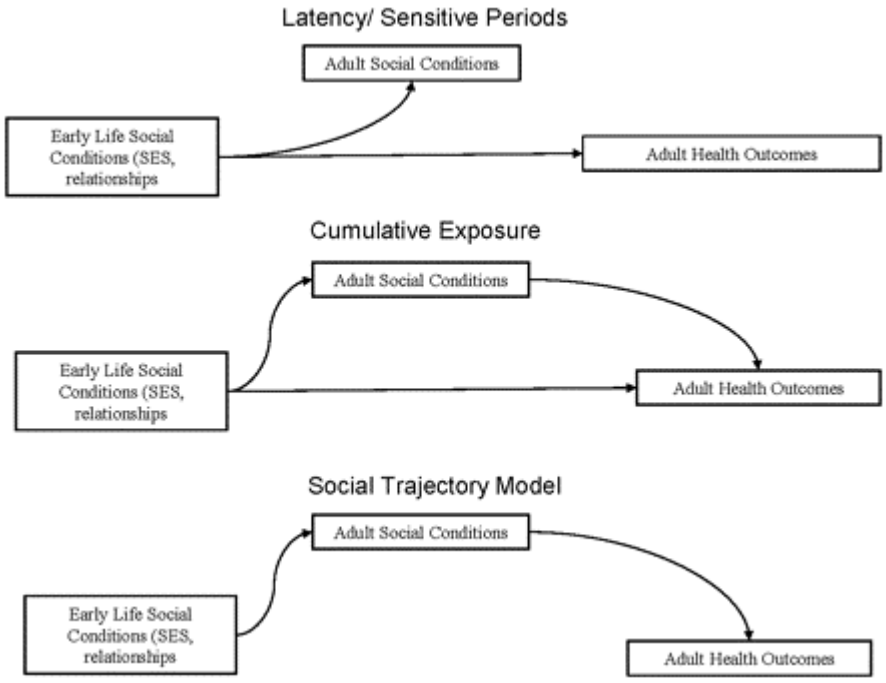

Figure 2. Three analytic models used in life-course approaches

Reproduced from Considering life course concepts by Hendricks. Copyright 2012 by Oxford University Press on behalf of The Gerontological Society of America.

In the top model, early life conditions are proposed to independently influence adult social conditions and health outcomes, regardless of the events occurring between these two life periods. Moreover, there is no hypothesised path between the two adult outcomes as this latency/sensitive period model emphasises that early life exposures strongly impact later life outcomes. The middle model deviates from the top model by viewing adult social conditions as an intermediate path between early life exposures and adult health outcomes. It also proposes that early life experiences may have both direct and indirect but cumulative effects on adult outcomes. In contrast, the last model disregards the direct effect of early life conditions on adult health but rather views adult health outcomes as resulting from the effect of early life experiences on adult social conditions (Hendricks, 2012).

Other related life-course concepts such as the presence of mediating and modifying factors describe the interrelationships and types of mechanisms among different exposure and outcome variables. For instance, a risk or protective factor may be a mediator if it is a variable that stands in the middle of the "causal chain" between the exposure and the outcome. It is also presumed to be a causal

consequence of the exposure and is associated with the outcome (Babyak, 2009). Based on Figure 2, adult social conditions as illustrated in the cumulative exposure and social trajectory models are potential mediators in the relationship between early life conditions and adult health outcomes (Hendricks, 2012).

In reserve capacity studies, a review which examined emotional factors as a mediating pathway presented mixed results. Emotional factors did not mediate the associations between SES and health outcomes such as coronary heart disease (CHD) and infectious diseases according to some studies (Matthews et al., 2010). On the other hand, some studies found that hostile personality traits strongly mediated the association between SES and mortality in men, but not in women (Matthews et al., 2010). The aggregate psychosocial resources yielded more promising evidence as mediators in the SES-health relationship. A multilevel analytical study among European and North American countries found that social support, particularly relationship with parents and friends mediated the association between family SES and adolescent health, largely explaining health inequalities (Moor et al., 2014). Social support, perceived control, mastery, optimism and self-esteem were also found to account for the association between SES and health outcomes such as stroke, metabolic syndromes and mortality (Gallo et al., 2009).

On the other hand, a third factor may be a modifier when it either enhances or diminishes the effect of the main exposure variable on the outcome, i.e., the main effect varies across different levels of the modifying factor which is empirically tested via statistical interaction (Kuh et al., 2003). For example, Chen and Miller (2013) demonstrated interaction between childhood SES, psychosocial resources and physiological risks. They showed that low SES individuals with high levels of reserve capacity had better health outcomes compared to their counterparts with poor cognitive reappraisal and emotion regulation as well as low optimism. On the other hand, there was no interaction between reserve capacity and physiological risks among high SES individuals (Chen & Miller, 2013).

As research initiatives further unravel the underlying causal mechanisms between early life factors and later outcomes and techniques and models become more sophisticated for empirically testing the relationships of variables, developmental and life-course approaches provide valuable insights for the understanding of how childhood and adolescent conditions contribute to psychosocial development and shape life-course trajectories (Gallo & Matthews, 2003).

2.2.6 Elements of reserve capacity in adolescence

Theoretically, reserve capacity represents one's reserve of tangible material goods, interpersonal factors related to social functioning and intrapersonal characteristics indicating resilience which are used for dealing with stressful life events (Gallo & Matthews, 2003; Gallo et al., 2009; Matthews & Gallo, 2011). Currently, there is no standard operationalisation of reserve capacity except for the implication that aggregate resources provide more support to the framework than a singular factor does (Gallo & Matthews, 2003; Gallo et al., 2009; Matthews & Gallo, 2011).

The studies included in Table 1 were mostly those cited by the proponents in their specification of reserve capacity which tested certain aspects of the framework in adolescent populations (Matthews & Gallo, 2011). Newer studies which assessed the role of psychosocial resources in the SES-health gradient as well as in educational trajectories, with or without formal use of the term "reserve capacity," were also included. As opposed to research on the psychosocial pathway among children or adults, the evidence base for adolescents is quite limited.

Table 1. List of studies assessing the role of reserve capacity on health and educational outcomes among adolescents, arranged in reverse chronological order

Author, year, study design	Study population	Indicator/s of reserve capacity	Outcome	Main findings
Brekke, 2015; longitudinal study	Grade 10 pupils in Oslo aged 15-16 years (n=5,335)	Social support based on relationship with family or friends	Enrolment in higher education	Social support from family predicted enrolment in higher education even after adjustment for parental SES. It also mediated the association between adolescent health and enrolment in higher education along with health behaviour.
Merritt & Buboltz, 2015; cross-sectional study	Undergraduate students at a US southern university with mean age of 19.7 years (n= 298)	Academic self-efficacy and perceptions of parental involvement	Desired level of educational degree	Family SES and parental involvement predicted academic self-efficacy. Academic self-efficacy, in turn, predicted academic expectations. Parental support mediated the relationship between family SES and self-efficacy.
Moor et al., 2014; Cross-sectional school-based survey	11, 13 and 15-year-old students in 41 countries in Europe, North America and Israel (n=117,460)	Social context of parents, school, peers such as relationships with parents, peer support, number of friends	Self-rated health (SRH)	Psychosocial factors had direct and indirect (through behavioural factors) impact on SRH. Relationship to father and academic achievement greatly mediated the association between family SES and SRH. All

				psychosocial factors had 30% contribution in explaining health inequalities.
Olsson, McGee, Nada-Raja, & Williams, 2013; longitudinal study	Birth cohort of 1037 people born in New Zealand assessed in childhood (5-9 years), adolescence (15-18 years) and adulthood (32 years)	Childhood and adolescent social connectedness, and academic achievement in adolescence	Adult psychosocial well-being	Childhood disadvantage directly affected childhood social connectedness and indirectly, adolescent social connectedness and academic achievement. Childhood indicators had direct paths to adolescent indicators. Both adolescent indicators had direct paths to adult well-being.
Räikkönen & Matthews, 2008; cross-sectional study	217 adolescents aged 14-16 years from two multi-ethnic high schools in Pennsylvania	Dispositional optimism and pessimism;	Ambulatory blood pressure (ABP) load	Optimism was not related with ABP. Those scoring on the lowest quartile of the distribution of pessimism scores had lowest systolic ABP levels than those with other scores. There was interaction between optimism and personal mood.
Chen, 2007; experimental study	115 American and African-American adolescents aged 16-19 years	Interventions Type 1: targeting perceptions of control by allowing adolescents to decide parameters of the task	Cardiovascular reactivity	The interventions were comparable in terms of adolescents' coping with the tasks. Significant interactions between SES and interventions were observed on cardiovascular reactivity where effects were seen on low SES adolescents. The resource manipulation intervention had

		Type 2: resource manipulation by providing adolescents with informational assistance during the task		greater impact compared to targeting control on low SES adolescents.
Evans, Kim, Ting, Tesher, & Shannis, 2007; longitudinal design for measure of allostatic load but correlational for another outcome	207 Grade 7 and 8 New York students	Maternal responsiveness	Allostatic load – index of chronic physiological stress; Cardiovascular reactivity and recovery	There was interaction of maternal responsiveness and cumulative risk exposures on allostatic load, but this had no effect on dynamic cardiovascular functioning. Results suggested that maternal responsiveness buffered effects of cumulative risks on allostatic load
Finkelstein, Kubzansky, Capitman, & Goodman, 2007; cross-sectional analyses from a school-based cohort	non-Hispanic black and white Ohio students aged 16 years on the average (n=1,167)	Optimism and engagement and disengagement coping styles	perceived stress	Coping styles and optimism were independent predictors of stress but only optimism mediated the relationship of parental education with stress.
Murasko, 2007; longitudinal study	1970 British Cohort Study with birth sample population of 16,135 followed through	Locus of control and self-esteem at age 10	Continued education and maternal report of health status (fair/poor) at age 16;	Locus of control predicted educational outcomes at age 16 and age 29, respectively. Similarly, self-esteem weakly predicted health status at age 16 and age 29. Adolescent outcomes were

	childhood (age 10), adolescence (age 16) and adulthood (age 29)		Obtained degree or its equivalent and self-report of health status (fair/poor) at age 29	associated with both outcomes in young adulthood. Including adolescent outcomes did not mediate the effects of childhood psychosocial factors on adult outcomes.
Piko and Fitzpatrick, 2007; cross-sectional study	1114 students in secondary schools of Hungary aged 14-21 years	Psychosocial health measured by self-perceived health, psychosomatic and depressive symptomatology	Psychosocial health and health behaviours	Lower SES was associated with poorer psychosocial health and low level of physical activity
Chen, Fisher, Bacharier, & Strunk, 2003; cross-sectional study	30 adolescents aged 13-18	Control beliefs on illness (asthma)	Immune and cortisol profiles - asthma biological markers	Control beliefs reduced the percentage of variance in immune markers that SES accounted for. Adjustment for control beliefs rendered the relationship between SES and immune markers statistically insignificant.
Due, Lynch, Holstein, & Modvig, 2003; cross-sectional study	Danish students aged 11, 13 and 15 years (n=5,205)	Social relations with parents, friends, teachers and school	Health status based on weekly physical and psychological symptoms	Almost no socioeconomic differences in health symptoms existed after simultaneous adjustment for all aspects of social relations. Parental and school relations indices were strongly associated with health status of adolescents.

2.3 Adolescence as an important stage in the life-course

The emphasis on adolescence as a crucial stage results from its unique position in the life-course and its strong potential to either complement or counteract the impact of early childhood experiences on life-course trajectories and adult outcomes (Johnson et al. 2011). As rapid biological and social changes take place during this period (Viner et al., 2012) which also coincide with cognitive, psychosocial and emotional development (Sanders, 2013), adolescents likely form new behaviours and competencies (Viner et al., 2012). These behaviours and competencies may affect how the adolescent experiences various life stage transitions, consequently influencing one's health and educational trajectories (Viner et al., 2012). Thus, life-course trajectories which may have been predetermined by early life social conditions may be redirected by intermediate pathways in adolescence (Johnson et al. 2011).

To provide a structure for the description of the possible pathways in adolescence which influence adult outcomes, we borrow the ecological approach used by Chen and Miller (2013) in understanding the mechanisms that contribute to health disparities. This approach recognises that social conditions produce differences at multiple levels – individual, family and neighbourhood – which shape the way individuals live (Chen & Miller, 2013). It also underscores the importance of the larger social structures and context where individuals are nested and supports the paradigm of Bronfenbrenner (1986) that human development is influenced by both intrafamilial processes and extrafamilial settings, particularly the environment. For this study, we focused on relevant individual, family and neighbourhood factors such as the school.

Exploring these adolescent mechanisms yields greater understanding of how socioeconomic and developmental processes in this life stage interact to influence biological and physical health over the life-course (Kroenke, 2008; Matthews & Gallo, 2011). Further, the understanding of these mechanisms provides implications for the nature, target and timing of interventions which could reduce health and socioeconomic inequalities in the future (Chen et al., 2006). Thus, the adolescent period presents opportunities for redirecting early life disadvantages into positive adolescent development and healthy transition into adulthood (Johnson et al., 2011; Sawyer et al., 2012).

2.3.1 Family SES and intergenerational transmission of SES

The associations between parents' socioeconomic circumstances and their children's health (Brekke, 2015; Chen et al., 2006; Juárez et al., 2016; Liu et al., 2013; Remes et al., 2010) and educational outcomes (Becker & Hecken, 2009; Bird, 2007; Brekke, 2015; Fergusson, Horwood, & Boden, 2008; Koivusilta, West, Saaristo, Nummi, & Rimpelä, 2013; Suhonen & Karhunen, 2019) have long been established. Research showed that children whose parents had low income and had little or no education faced greater barriers in achieving their human potential than children born to parents with higher income and education. They had less access to quality or higher education (Suhonen & Karhunen, 2019) and were less healthy. It was also likely that their parents had low priority for education compared to children of parents with high income and education (Bird, 2007). Low SES families were also characterised by high dependency ratios and complex family structures which affect the availability of material and social resources used to promote the well-being of children (Bird, 2007).

Previous research focusing on family SES as a predictor of child development has been based on two tenets namely, the family stress model (FSM) and the investment model (IM) (Conger et al., 2010). According to the FSM, economic hardship affects the relationships between parents. This leads to poor parenting practices which influence the cognitive, emotional and behavioural development of children (Conger et al., 2010). On the other hand, the IM proposes that families with more economic resources tend to invest in the health and education of their children than those with fewer resources who need to invest in their family's subsistence (Conger et al., 2010). Educational investments are also motivated by the parents' desire to maintain the status of their children and prevent their downward social mobility (Albertini & Radl, 2012).

Other causal hypotheses, commonly used to explain socioeconomic inequalities in health were: the social causation theory, the social selection theory and the indirect selection hypothesis (Foverskov & Holm, 2016). According to the social causation theory, social conditions, such as one's family SES, cause differences in the health and development of children (Conger et al., 2010; Martin et al., 2010). In contrast, the social selection theory, argues that one's health as well as individual characteristics predict future SES (Conger et al., 2010). The indirect selection hypothesis does not propose a causal relationship between health and SES, but

asserts that third factors, such as biological and psychological characteristics, account for the SES-health association (Foverskov and Holm, 2016).

Since neither social causation nor social selection exclusively explained the causal relationship between SES and child development, both theories were integrated into an "interactionist" model. Conger and colleagues (2010) proposed that like the social causation theory, the SES of parents, directly influenced their adult SES as well as their individual characteristics during childhood and adolescence. This, in turn, also independently shaped their adult SES, echoing the social selection theory. The interactionist model further proposed that SES indirectly influenced children's development through family dynamics, parenting practices and investments for children (Martin et al., 2010). This essentially provided logical support for the transmission of SES across generations.

An economic model on the intergenerational transmission of SES incorporated the human-capital approach to inequality (Becker & Tomes, 1979). It theorised that while family income and intergenerational mobility may depend on luck in market rewards (e.g., the rate of economic growth, taxes and subsidies, foresight about the incidence of "disturbances"), various family parameters due to the inheritability of "endowments" influence SES transmission across generations. These endowments include caste, religion, race, culture, genes, social networks and the propensity to invest in children (Becker & Tomes, 1979). Indeed, research findings have attributed the intergenerational transmission of SES from parents to children either to parental investments or endowments. While investments refer to active parental contributions of resources such as time and money, endowments encompass everything that parents possess, which are passed on to their children (Erola & Kilpi-Jakonen, 2017).

The increasing availability of longitudinal data spanning more than two generations in recent years made it possible to demonstrate that reproduction of social class across generations or intergenerational transmission of SES can occur across multiple generations. Evidence of grandparents' effect on intergenerational transmission of both health (Johnston, Schurer, & Shields, 2013; Modin and Fritzell, 2009; Osler, Andersen, Lund, & Holstein, 2005) and economic outcomes (Chan & Boliver, 2013; Chan & Boliver, 2014; Erola & Moisio, 2007; Møllegaard and Jæger, 2015) have been shown.

Family parameters influencing overall child development now extend beyond parental influences. Erola and Kilpi-Jakonen (2017) proposed that aside from

institutional mechanisms, there are other individual mechanisms operating within the family which affect resource transfers across generations. They highlighted the notions of compensation and multiplication which influence intergenerational transmission of SES. Multiplication occurs when additional resources from other family members are extended and increase or "multiply" effects, especially on child outcomes of those with high family resources. On the other hand, compensation results when other family members, such as older children, grandparents or parents' siblings compensate for or replace the loss of parental resources (e.g., through death or divorce) with other resource types; this is particularly beneficial for those with low resources (Erola & Kilpi-Jakonen, 2017). Bengtson (2011) stated that changing nuclear family structure characterised by multigenerational relationships due to greater longevity and higher rates of marital disruption and divorce probably increased the roles of grandparents in their grandchildren's lives. Grandparents now have more interactions with their grandchildren than before, particularly, when they are tapped as a source of informal childcare (Geurts, van Tilburg, Poortman, & Dykstra, 2014).

Grandparental investments were motivated by several factors and differed across types of grandparents. For example, Coall and Hertwig (2010), using an evolutionary framework, described genetic relatedness, paternity certainty and sex-specific reproductive strategies as reasons for grandparental investments. Likewise, these also explain why maternal grandparents, especially the maternal grandmother, tend to invest more in their grandchildren compared to paternal grandparents. Grandparents also transfer more resources to grandchildren with high reproductive value (tendency to reproduce and have descendants), and based on kin altruism and emotional connectedness.

In egalitarian societies with low-risk family contexts, the economic capital of grandparents may not matter much as grandparental investments in the form of socio-emotional support may have more impact in their grandchildren's physical and mental well-being (Coall & Hertwig, 2010). For instance, in Denmark, the grandparents' cultural capital (measured as their education and cultural participation) and not their economic nor social capitals, influenced the choice of secondary education of grandchildren which were either vocational or academic (Møllegaard& Jæger, 2015). Although, in Sweden, the grandparents' wealth, which included financial, net home and net real wealth, predicted the educational achievement of grandchildren, probably due to normative and insurance mechanisms (Hällsten &

Pfeffer, 2017). The normative mechanism accounts for the pro-education norms of families where they view education as a strategy to reproduce their wealth in subsequent generations. The insurance mechanism, on the other hand, refers to wealth's purpose as protection against potential risks or economic constraints which allow grandchildren to maximise their human capital (Hällsten & Pfeffer, 2017).

The intergenerational transmission of SES over three generations underscores the importance of grandparents in the origins of socioeconomic inequalities. Whether grandparents have direct or indirect effects on their grandchildren's development via parental mechanisms merit further testing and research. This is to confirm the impact of grandparents' SES and promote better understanding of the mechanisms involved in the transmission.

2.3.2 Family structure and rearing environment

It has long been recognized that the family, through its structure, functioning and processes as well as how it connects with its external environment, is fundamental to the development of children (Bronfenbrenner, 1986; Viner et al., 2012). Intergenerational transmission of lifestyle factors has also been documented (Aufseeser, Jekielek, & Brown, 2006; Bricard et al., 2010; El-Amin et al., 2015; Tubeuf et al., 2012), reiterating the large role of the family environment in health behavioural development. Indeed, a family unit can affect the views, actions and behaviours of its individual members (George, 2013) and can either protect against or confer risk of poor developmental outcomes (Viner et al., 2012).

Literature has shown that early life family environment, determined by the quality and type of parenting (Bird, 2007) as well as quantity of parent-child social interaction (Kroenke, 2008), has a large impact on children's socio-emotional or psychosocial development. Nurturing parenting provides intellectual stimulation and healthy social patterns of behaviour which positively affects the children's health and educational outcomes (Bird, 2007; Chen & Miller, 2013). Conversely, neglectful or abusive parenting predisposed children to poor developmental outcomes (Chen & Miller, 2013). Indeed, a disruptive family environment marked by parental conflict, chronic stress and neglectful parenting led to adverse biological and clinical outcomes in children and poor adult psychosocial functioning (Matthews & Gallo, 2011). An intervention using a family perspective approach which recommended changes in parenting strategies and included fathers to improve spousal

communication has resulted in improved cognitive and receptive language skills in young children (Black & Surkan, 2015).

During adolescence, the home environment can support one's well-being through parental monitoring, family communication, and parental modelling of positive behaviours (Aufseeser et al., 2006). Greater family involvement and satisfaction also provided the children with consistency and stability during early adolescence. This contributed to positive youth development and successful transitions into adulthood (Ward & Zabriskie 2011).

Adolescents who were not living with both parents had poor family functioning, indicated by more family conflict as well as less parental monitoring, cohesion and communication. This was predictive of substance use (Wagner et al., 2010). The absence of a father also predisposed children and adolescents to more emotional and behavioural problems, risky health behaviours and poor academic achievement compared to those with father involvement (East, Jackson & O'Brien, 2006). Similarly, Astone and McLanahan (1991) found that children raised in single-parent and step-parent families had less parental involvement and parent-child communication. This led to lower educational success of the children. On the other hand, Carlson and Corcoran (2001) found that children raised consistently in two-parent families had lower levels of behavioural problems and higher cognitive test scores compared to those raised in other family types such as single-parent households or changing family structures.

Apart from the household composition which varies depending on the involved nuclear family, the number of children and presence of extended family members, other related familial events such as fostering, adoption and orphanhood (Bird, 2007) as well as parental divorce, death and presence of new partners shape the family structure (Erola & Kilpi-Jakonen, 2017). Consequently, the family structure influences the family environment and predicts developmental outcomes of children (Bronfenbrenner, 1986). The effect of family structure likely operated through family income and parental socialisation indicative of the quality of family environment (Carlson & Corcoran, 2001). For instance, it implies a form of social capital such as social support since structural factors influence social capital formation (Weiss, 2012). Then, social support from relationships can be measured by both its structural and functional aspects (Holt-Lunstad et al., 2010).

While the family environment has a critical role in the healthy development of the children, intrafamilial processes are influenced within the context of a larger

environment (Bronfenbrenner, 1986). Social institutions such as the state, school and those within the community also affect the family through contributions of economic and social capitals (Bronfenbrenner, 1986; Erola & Kilpi-Jakonen, 2017). For instance, welfare states such as Finland and other Nordic countries, provide extensive social benefits and protection for their citizens (support for unemployment, childcare, education, etc.) to support families and ensure equality of opportunities for everyone (Erola & Kilpi-Jakonen, 2017). Indeed, policies or programs which recognise the interdependence of families and the communities where they belong to and strengthen both their capacities to support children's health and development reap greater and longer-term societal advantages (Hoagwood et al., 2018).

2.3.3 Peer Influence

Part of the adolescent's social and emotional development is having an increased desire for independence and autonomy from parents (Sawyer et al., 2012). Thus, during an individual's transition from childhood to youth, major influences are derived from one's peer group (Viner et al., 2012; West, 1997). Formation and adoption of new behaviours take place during this period (Viner et al., 2012) and peer norms are substituted for parental norms (Moore & Littlecott, 2015).

Unhealthy behaviours such as alcohol drinking, substance use and unsafe sex adopted in adolescence affect healthy transitions to adulthood and influence adult trajectories (Johnson et al., 2011; Sawyer et al., 2012). Bahr and Hoffmann (2010) showed that adolescents with close friends who used alcohol were more likely to have also used alcohol and participated in heavy drinking than those with friends who did not drink. Westling and colleagues (2008) similarly demonstrated that association with deviant or misbehaving peers independently predicted both cigarette and alcohol use in adolescents.

On the other hand, Padilla-Walker and Bean (2009) found that positive direct peer pressure based on having friends who "help you do what is right or encourage you to follow rules" increased positive behaviours such as social initiative, self-esteem and empathy, and decreased negative behaviours such as delinquency and depression.

The adolescents' approaches to learning and achievement motivation were likewise influenced by peer class-climate and academic valuing of one's best friend

(Nelson & DeBacker, 2008). Indeed, a study found that peer effects clearly existed in terms of academic achievement; academically strong students increased their peers' academic performances while weak students decreased those of their peers (Winston & Zimmerman, 2004). Peer expectations were also found to be related with university completion, even after controlling for school performance (OECD, 2018).

Peer influence works within social structures of neighbourhood and school (Viner et al., 2012) but generally exerts greater effects within school, especially during secondary education (West, 1997). Peer effects may function more commonly by directly attempting to change one's attitudes or behaviours or indirectly, through unintentional modelling (Padilla-Walker & Bean, 2009). Thus, peers may either protect against or confer salient health and developmental risks during adolescence (Viner et al., 2012).

Aside from peer effects on health behaviours and practices, social and emotional support derived or lacking from friends are linked to psychological processes such as stress, depression, isolation, loneliness and perceptions of not belonging (Holt-Lunstad et al., 2010). This may affect cognitive and biological pathways leading to health and education. Parental monitoring during this life period is very important to ensure that adolescents associate with good influencers (Bronfenbrenner, 1986; Tomé, Matos, Simões, Diniz, Camacho, 2012).

2.3.4 School

Education is one of the strongest pathways to good health and high SES (Freudenberg & Ruglis, 2007). In adolescence, educational success is largely determined by academic achievement as it leads to either schooling discontinuation or enrolment in higher education (Brekke, 2015; Johnson et al., 2011; Slominski, Sameroff, Rosenblum, & Kasser, 2011). Thus, the school, as an educational institution, is an influential social environment shaping the health and development of adolescents (Sawyer et al., 2012; Viner et al., 2012).

Several school characteristics found to be associated with higher dropout rates were having low SES student populations; high level of racial or ethnic segregation; poor school safety and disciplinary policies; high student-to-teacher ratios; academic tracking; and lack of programs and support for transition into high school (Freudenberg & Ruglis, 2007). On the other hand, safe and supportive schools which

promote peer connections and foster engagement between students and teachers decreased risky behaviours and other behavioural problems in adolescents (Moore & Littlecott, 2015; Sawyer et al., 2012). The students' positive perceptions of their classroom environment were related to increased academic efficacy, self-regulated learning and decreased disruptive behaviour. These perceptions were particularly centred on the teacher in terms of support, promotion of interaction and mutual respect (Ryan & Patrick, 2001). Additionally, good school leadership and strong student and parental connections with school positively influenced health outcomes in adolescents (Viner et al., 2012).

The schools' socioeconomic profile or affluence also operates through various factors to influence academic achievement of adolescents (Fergusson et al., 2008; OECD, 2018). It affected the availability of teaching resources and teaching strategies, disciplinary climate, class size and students' academic level (OECD, 2018). It also interacted with family SES in a way that further disadvantaged students from lower SES families (Moore & Littlecott, 2015). For instance, lower SES students attending less affluent schools were less likely to complete higher education than their higher SES peers attending the same schools (OECD, 2018). Also, while more affluent schools had environments characterised by healthier peer norms and behaviours, these behaviours were not seen among those students from poorer families attending such schools (Moore & Littlecott, 2015).

Several educational interventions recommended for improving student engagement and educational success required structural, institutional and organisational changes as well as changes in the curriculum, instruction and teacher support (Freudenberg & Ruglis, 2007). The experiences of countries which achieved educational success showed that major changes in national economic and political policies were needed (Little & Green, 2009) to support schools in their functioning roles in population development. For instance, the evolution of Finland's educational reform, which led to its current status as one of the world's outstanding educational systems, was not achieved overnight (OECD, 2011). In the 1950s and over the next decade, many private schools in the country started receiving state subsidies and coming under public control. This markedly increased the enrolment of students as most Finns discontinued schooling after six years of basic education during that time. After the war, the government created the comprehensive school (peruskoulu) reform which revolutionised Finnish education and made it more equitable for all young students. Through a new basic education system built in 1968,

based on the idea of a common or comprehensive school, compulsory basic education increased from six to nine years and was municipally run. This new system implied that students had longer years spent in education (Figure 3) and began tracking to upper secondary school at around 16 to 17 years of age (OECD, 2011). Subsequent reforms also included mandatorily requiring prospective teachers to complete postgraduate qualifications and creating entry pathways for vocational students to tertiary education. This eventually resulted to higher quality of employed teachers as well as better educated students in Finland (OECD, 2011).

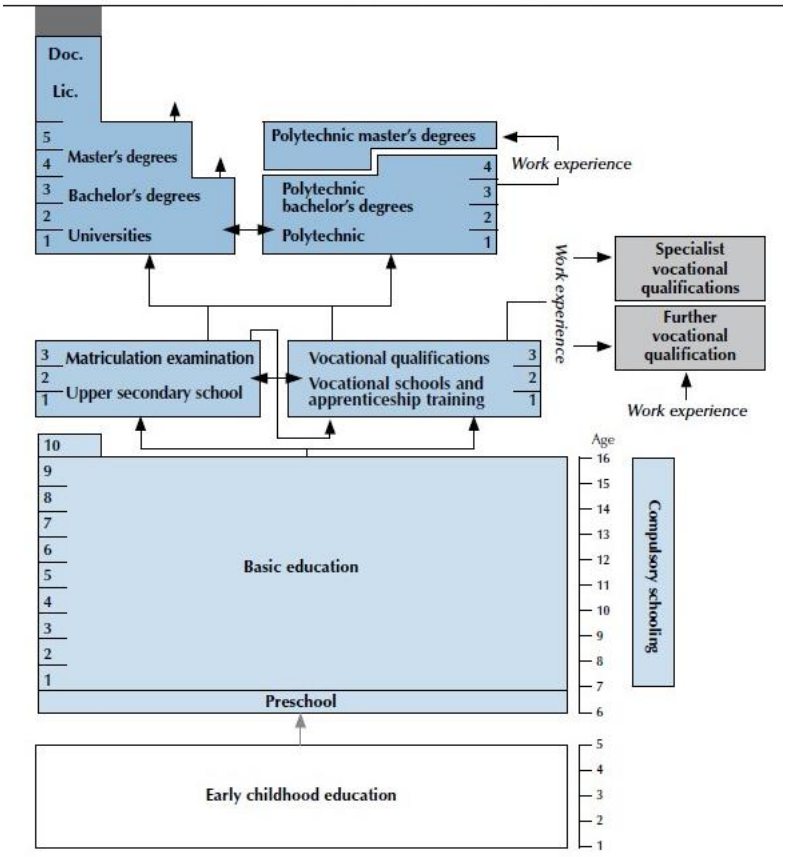

Figure 3. Finland's educational system

Reproduced from Lessons from PISA for the United States, Strong Performers and Successful Reformers in Education by OECD. Copyright 2011 by OECD Publishing.

2.3.5 Puberty

Apart from familial factors and those within the social structures where the adolescent lives, individual processes also serve as pathways influencing health and educational outcomes. During adolescence, puberty is one such salient pathway. It is a recognised marker of adolescence accompanied by biological maturation and physical development (Sawyer et al., 2012). Since puberty typically occurs at a time which coincides with the cognitive, social and emotional development of a person, it may lead to the formation of new behaviours and competencies (Viner et al., 2012). These behaviours and competencies affect how the adolescent experiences various life stage transitions in his/her relationships and environment, consequently shaping future trajectories (Viner et al., 2012).

Globally, secular changes towards earlier onset of puberty have been observed (Sawyer et al., 2012). In Europe, the mean age at pubertal onset occurred around 17-18 years in the 19th century (de Muinich Keizer & Mul, 2001) and declined to approximately 12-13 years in most of Western European and Scandinavian countries in the last century (Aksglaede, Olsen, Sørensen, & Juul, 2008; Parent et al., 2003). Similar trends were recorded in the United States, although, gender and racial differences existed (Euling et al., 2008). In some Asian, African and South American countries, such trends were observed among those living in privileged conditions (Parent et al., 2003).

Pubertal onset is influenced by a combination of genetic and environmental factors (de Muinich & Mul, 2001; Parent et al., 2003). These factors include certain gene regulators, gender, race and ethnicity (Euling et al., 2008; Obeidallah, Brennan, Brooks-Gunn, Kindlon, & Earls, 2000; Parent et al., 2003); exposure to endocrine disrupting chemicals (Aksglaede et al., 2008; Parent et al., 2003); urbanisation (de Muinich and Mul 2001; Parent et al., 2003); socioeconomic conditions (de Muinich and Mul 2001; Downing & Bellis, 2009; James-Todd, Tehranifar, Rich-Edwards, Titievsky, & Terry, 2010; Parent et al., 2003; Sun, Mensah, Azzopardi, Patton, & Wake, 2017); childhood health status (Bellis, Downing, & Ashton, 2006; de Muinich & Mul, 2001; Parent et al., 2003); and, family environment and stress (Bellis et al., 2006; Bleil et al., 2013; Golub et al., 2008).

The physical, behavioural, emotional and health effects of puberty are more pronounced when its timing occurs earlier or later than in one's age group (Sawyer et al., 2012). Altered pubertal timing results in psychological and adjustment issues

linked to elevated symptomatology and risks of psychopathology in adolescence and other disorders in adulthood (Golub et al. 2008; Graber, 2013). Early maturers had higher rates of depressive, substance and disruptive behaviour disorders (Bellis et al., 2006; Downing and Bellis, 2009; Golub et al., 2008; Graber, 2013; Koivusilta & Rimpelä, 2006). Early maturing girls had higher risks of developing cardiovascular disease (Bleil et al., 2013; Golub et al., 2008; Jacobsen, Oda, Knutsen, & Fraser, 2009; Lakshman et al., 2009), obesity, type 2 diabetes and breast cancer. Early maturing boys, on the other hand, had increased risks for testicular cancer (Golub et al., 2008). These boys and girls also had increased risks for accelerated skeletal maturation and short adult height, early sexual debut and potential sexual abuse (Golub et al., 2008). On the other hand, late maturation increased fracture risk (Zhu & Chan, 2017) and psychopathology in boys in terms of higher rates of depressive symptoms (Graber, 2013; Rudolf, Troop-Gordon, Lambert, & Natsuaki, 2014; Zhu & Chan, 2017) and disruptive behaviours and substance use disorders (Graber, 2013; Zhu & Chan, 2017), including suicidal tendencies (Golub et al., 2008). In girls, delayed puberty was associated with increased risk of cardiovascular disease and lower bone mineral density which may predict osteoporosis risk in adulthood (Zhu & Chan, 2017). Psychopathology, though, was not seen as a cause for concern in late maturing girls as this group had better psychosocial functioning outcomes compared to other maturers (Graber, 2013). Studies have shown lower levels of depressive symptoms among girls with delayed puberty in relation to those with on-time puberty (Rudolf et al., 2014; Zhu & Chan, 2017).

Hormonal changes during puberty were thought to influence brain development and result in enhanced synaptic connections, neuronal transmissions and heightened responses to stimuli, consequently affecting cognitive development (Sawyer et al., 2012). The effects of puberty on academic performance (Cavanagh, Riegle-Crumb & Crosnoe, 2007; Martin & Steinbeck, 2017) and educational outcomes (Koerselman & Pekkarinen, 2017; Koivusilta & Rimpelä, 2004) have been documented but results were inconsistent. Early maturing American girls with early puberty had poorer academic performance at the beginning and end of their high school years compared to those with on-time or later puberty (Cavanagh et al., 2007). Indeed, Gill and colleagues (2017) found that a one-year increase in age at menarche resulted in a longer time spent in education. In contrast, a British cohort study found that late puberty was associated with lower educational attainment and in boys, also with lower adult wages (Koerselman & Pekkarinen, 2017). When cognitive test scores at

age 16 were considered, however, these associations were reduced (Koerselman & Pekkarinen, 2017). Another study found that pubertal timing did not directly predict academic achievement but rather, affected academic motivation, such that those with later pubertal status had lower motivation than the others; academic motivation, then, influenced academic achievement (Martin & Steinbeck, 2017).

Commonly used markers of puberty in population studies are Tanner staging either through self-assessment or professional opinion based on the appearance of secondary sexual characteristics such as breast and pubic hair development; weight and height determination; age at menarche in girls (Parent et al., 2003); and ages at spermarche (de Muinich & Mul 2001; Euling et al., 2008) and first ejaculation in boys (Euling et al., 2008).

2.4 Issues on studies of socioeconomic inequalities and reserve capacity

As empirical evidence supporting the validity of the reserve capacity framework grew in recent years (Matthews et al., 2010), we learned more about the relationships among its certain components and found that these were moderated by demographic characteristics such as age, gender and ethnicity. Some methodological challenges were also noted.

In high income countries, gender inequalities existed in different health outcomes of adults (Viner et al., 2012). Generally, adult women were shown to be immune to the SES-health gradient effects in terms of mortality and morbidity, except for cardiovascular health outcomes (Phillips & Hamberg, 2015). The same pattern was observed in analyses of mortality of young adults (Remes et al., 2010). These were also noted in early adolescence, although, gender equalisation in some health behaviours have been observed in recent years (Viner et al., 2012).

The distribution of reserve capacity resources also varied by gender. Among adults, gender differences which cannot be explained by SES were found for decision latitude, availability of resources, coping and self-esteem (Sjögren & Kristenson, 2006). Among adolescents, levels of engagement coping were significantly higher in girls than in boys (Finkelstein et al., 2007). On the other hand, adolescent boys and girls did not vary in terms of optimism (Finkelstein et al., 2007; Räikkönen

&Matthews, 2008) and in most aspects of social relations except in terms of communication with their friends and perceived teacher support (Due et al., 2003).

Racial and ethnic disparities in health, apart from those due to underlying genetic influences, occur due to interactions with social mechanisms such as SES, culture, bias and health care access (Cheng et al., 2015). Race and ethnicity influenced outcomes of mental health, obesity, sexual health and risky health behaviour of adolescents in high income countries (Viner et al., 2012). Racial discrimination also acted as a social stress which manifested as higher stress-related biomarkers in black compared to white individuals. Such instance may shape health behaviours and also affect the psychological functioning of parents and children (Cheng et al., 2015). Indeed, a study found differences for optimism and engagement coping between non-Hispanic whites and non-Hispanic black adolescents (Finkelstein et al., 2007). In contrast, no significant differences were found for optimism of adolescents with African American or Caucasian origins (Räikkönen & Matthews, 2008). As populations in large regions become increasingly ethnically diverse such as in America (Cheng et al., 2015), and in Europe (Stronks et al., 2013), collection of race and ethnicity data is emphasized, particularly in child and adolescent research on health and socioeconomic inequalities.

Methodological challenges of the reserve capacity framework mainly include issues in study design and data measurement of key variables. Majority of research on reserve capacity in adolescents (Table 1), as well as in other population groups, were based on cross-sectional studies (Matthews & Gallo, 2011). These present temporal ambiguities and weak evidence for causality. Hence, life-course approaches and longitudinal studies are desired (Martikainen, Bartley & Lahelma, 2002). Ideally, experimental studies such as that done by Chen (2007) to test various facets of the framework should be conducted to obtain stronger and more informative evidence on the mechanisms influencing health and SES trajectories (Matthews & Gallo, 2011). But the feasibility of such approaches should be considered, and the generalisability of results should be evaluated.

The measurement and collection of key variables in the framework has inherent challenges. Evidence showed that effects of SES on health and development vary according to the type, frequency and timing of measurement used. Goodman (1999) found that indicators of SES such as income and education affected different health outcomes in adolescents. Individual and contextual measures of SES showed non-additive interaction effects on health behaviours of adolescents (Moore & Littlecott,

2015). Low SES measured at one timepoint did not show its hypothesised effect on adolescent health but when its duration was included, results showed the expected relationship (Evans & Kim, 2007).

Capturing the multidimensionality of psychosocial resources and the validity and reliability of the measures used also pose research difficulties. Psychosocial resources form at different levels of the social environment, develop or change along certain periods within the life-course and operate in different ways and in various combinations to influence health and development (Chen & Miller, 2013; Matthews et al., 2010). Thus, despite accumulating evidence on psychosocial resources, there is still no standard measurement of reserve capacity. Gallo (2009), however, recommended examining protective resources which are susceptible to intervention efforts.

Finally, to disentangle the role of reserve capacity in adolescence, age should not be treated as a confounder and controlled for in the analyses. Rather, disaggregated results with distinct age groupings should be presented when analysing health inequalities. The study of Juárez and colleagues (2016) could have been more informative if it had not mixed early life with adolescence as participants aged one to 19 years were grouped together. Likewise, distinct results could have been obtained had these studies treated adolescents separately from adults, instead of lumping participants from ages 15 to 74 years (van Oort, van Lenthe, & Mackenbach, 2005) and 18 to 69 years, respectively (Howarter & Bennett, 2013).

2.5 Synthesis

SES produces differences at multiple levels: individual, family and neigbourhood. These differences also intersect in various ways to shape a person's psychosocial characteristics and ultimately, influence health (Chen & Miller, 2013). Available evidence showed that SES-health links originated in childhood, through psychosocial processes formed within family environments (Matthews et al., 2010). Even with limited studies conducted among adolescents, the potential mediating role of reserve capacity on the relationship of parental SES with physical health was evident (Table 1). Results indicated that low SES children who developed adaptive resilience remained relatively healthy over the life-course (Chen & Miller, 2012). Literature on youth development also showed that children with psychosocial

resources, despite having low SES, had positive emotional and psychosocial development, enabling them to perform well in academics and be a productive member of society (Kroenke, 2008).

A life-course perspective where early life exposures either during childhood or adolescence (Kuh et al., 2003) and use of developmental models help explain the contribution of psychosocial and biobehavioural pathways in health and socioeconomic trajectories (Matthews & Gallo, 2011). Reducing inequalities in health and development necessitates national and global policies addressing social and economic conditions (Little & Green, 2009). These, however, require costly investments, lengthy processes and strong governance. On the other hand, understanding which pathways are influential can help identify the nature and timing of interventions which complement existing structural mechanisms (Gallo et al., 2009). Family- or school-based interventions especially directed at disadvantaged populations may be beneficial for improving health and SES in posterity (Hoagwood et al., 2018; Kroenke, 2008).

3 CONCEPTUAL FRAMEWORK

The conceptual framework for this study closely follows the life-course approach (see Figure 1) towards understanding the contribution of the psychobiological influences in the SES-health relationship (Matthews & Gallo, 2011). More formally known as the "reserve capacity model," it posits that individuals maintain a bank of psychosocial resources that are used in response to stress. Since low SES increases exposure to stress, one's reserve capacity may not be adequate when faced with such stressful situations. Thus, low SES individuals are more likely to experience negative emotions and psychological distress, which in turn, influence intermediate pathways leading to poor health (Matthews et al., 2010).

Similarly, we conceptualized that family SES directly affects one's health, using a terminal outcome (arrow A) and education (arrow B) in the life-course. Indirectly, mortality and education are linked to family SES via sequential psychosocial (arrows F and G), biological (arrows J and K) and educational (arrows H and I) pathways in adolescence. Family SES also directly influences the following: reserve capacity (arrow C), school achievement (arrow D) and puberty (arrow E) which are proposed to have interconnections with each other (arrows L, M and N). We have expanded the definition of family SES to reflect the influence of socioeconomic circumstances not only of parents but also of grandparents, implying intergenerational transmission of SES (Figure 4). In addition, reserve capacity included dimensions of perceived health and social support as well as health-promoting behaviors with underlying psychosocial resources. Assessment of social support included both structural (nuclear family) and functional forms (ease of communication at different levels) of social relationships (Holt-Lunstad et al., 2010).

Overall, the conceptual model adopts a life-course approach focusing on adolescent social and individual exposures and how these affect later life outcomes in health and education.

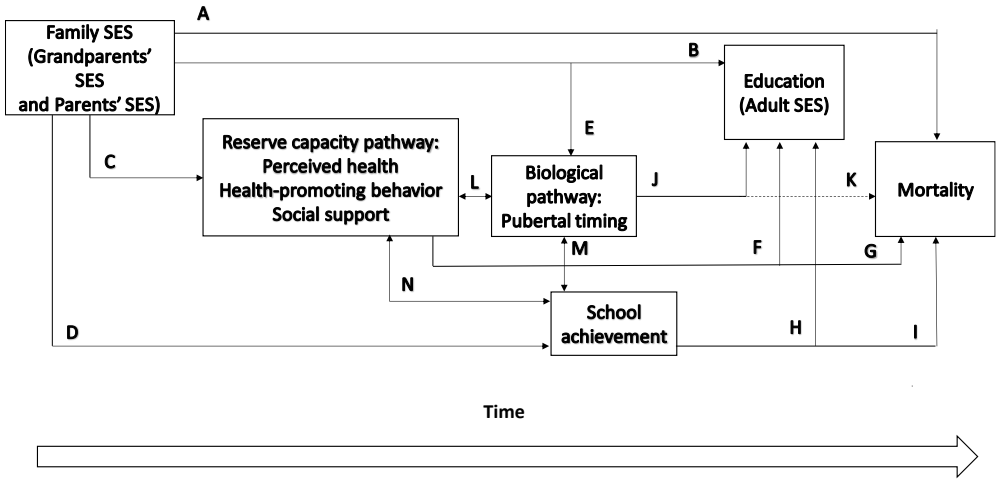

Figure 4. The effect of family socioeconomic status (SES) and the adolescent intermediate pathways of reserve capacity, puberty and school achievement on education and mortality in adulthood

Note: Path indicated by arrow K was not analyzed in this study.

4 AIMS OF THE STUDY

Our study aims to determine whether socioeconomic inequalities exist by assessing the effect of family SES on mortality and education of adolescents. We further determine the mechanisms by which family SES produce these inequalities by assessing the roles of reserve capacity and other pathways in adolescence such as pubertal timing and school achievement. We also assess if grandparents' socioeconomic circumstances affect their grandchildren's SES.

Specifically, we aim to answer the following questions:

1. What are the effects of reserve capacity and school achievement in adolescence on mortality and how do these mediate the relationship of family SES with later mortality? (Study I)
2. How do reserve capacity, school achievement and family SES in adolescence predict educational attainment? Do grandparents' socioeconomic circumstances also predict their grandchildren's education? (Study II)
3. Does a biological (pubertal timing) pathway influence educational attainment? What is the role of this pathway, along with reserve capacity and school achievement pathways, in the relationship of family SES with education? (Study III)

5 MATERIALS AND METHODS

This dissertation and the related original publications were based on two data sources linked to form a single, longitudinal dataset. The first source was the Adolescent Health and Lifestyle Surveys (AHLS) from 1979 to 1997. Conducted biennially since 1977, the surveys monitor the health and health-related lifestyle of adolescents in Finland using mailed questionnaires. The second source was registry data kept by Statistics Finland, specifically, the Finnish Official Cause-of-Death Register and the Register of Completed Education and Degrees, containing statistics on every resident in Finland. The data from Statistics Finland covered censuses every fifth year from 1970 to 1995, and yearly registry data from 2000 until 2009.

5.1 Study design and participants

A longitudinal study design was constructed by linking data from AHLS and the data from the registries by means of unique national personal identification numbers. Statistics Finland performed the data linkage according to a contract specifying the rights and duties of the data owners and administrators.

Baseline data were collected from AHLS of 1985, 1987, 1991, 1993 and 1995. Nationally representative samples of Finns aged 12, 14, 16, and 18 years born on certain days in June, July and August were drawn each study year from the Population Register Centre. A self-administered questionnaire, to be voluntarily answered, was sent by post in February, followed by two re-inquiries to non-respondents. Variables measured across all survey rounds were used.

Follow-up data containing outcomes of interest among AHLS participants as well as socioeconomic information of their parents and grandparents were obtained from the registries of Statistics Finland. The follow-up started on April 30 of each survey year and ended on December 31, 2009. Average follow-up time was 18.4 years. It ranged from 1 to 25 years with an estimated total of 770,161 person-years. At the end of the follow-up, the participants were aged 27 to 43 years.

Overall response rate was 79% (N=41,833), with 72% (N=19,509) for boys and 86% (N=22,324) for girls, respectively. In Study II, 11 cases without data on outcome were removed, yielding a study population of 41,822 adolescents. In Study III, those aged 12 years were further excluded as data on pubertal timing, one of the exposure variables, was not available for all of them. The last study used eligible data from 37,876 respondents. Table 2 shows the number of respondents according to age, sex and AHLS study year.

Table 2. Study population by age, sex and study year, Finland

Age	**Study year**					
Boys	**1985**	**1987**	**1991**	**1993**	**1995**	**Total**
12	353	405	425	400	393	1976
14	3	1341	1629	1861	1177	6011
16	453	1383	1562	1655	1232	6285
18	408	1012	1286	1460	1071	5237
Subtotal	1217	4141	4902	5376	3873	19509
Girls						
12	359	363	395	436	423	1976
14	4	1425	1837	2008	1301	6575
16	497	1479	1912	1943	1469	7300
18	469	1274	1626	1791	1313	6473
Subtotal	1329	4541	5770	6178	4506	22324
Total	2546	8682	10672	11554	8379	41833

In the earlier censuses of Statistics Finland, children (parents in this study) who were no longer living with their parents (grandparents in this study) during the time of the census could not be linked to their families. This explains the large number of grandchildren with unknown data for grandparents. Table 3 presents the extent of missing or unknown information on respective parents and grandparents of the respondents included in this study.

Table 3. Number of cases with missing/unknown information for parents and grandparents (N=41,833)

No data	No.	%
Father	693	1.7
Mother	43	0.1
Maternal grandfather	21507	51.4
Maternal grandmother	18538	44.3
Paternal grandfather	22677	54.2
Paternal grandmother	19620	46.9

5.1.1 Ethical considerations

The study protocol was approved by the Institutional Review Board and the Data Protection Ombudsman of Statistics Finland. The Joint Commission on Ethics of the University of Turku and the Turku University Hospital 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. The first review boards at the universities were established in Finland in the 1980s. AHLS was reviewed by the Ethical Review Board of the University of Helsinki, Department of Public Health in 1986. Parental consent was not considered by the ethics review board at that time. In later surveys, the latest in 2017, the relevant review boards have waived the parental consent. The participation of adolescents in the surveys was voluntary.

5.2 Outcomes: Mortality and education

The outcomes studied were obtained from the registries of Statistics Finland. Data on mortality of the index AHLS person was based on the recorded month and year of death in the Finnish Official Cause-of-Death Register. The adolescent's education which referred to the highest educational attainment, was based on the exact degree codes according to the Finnish Standard Classification of Education (Statistics Finland, 2018) recorded in the Register of Completed Education and Degrees. These degree codes corresponded to the level of education reached or completed e.g.,

primary, lower or upper secondary, tertiary or graduate levels of education. These codes were further grouped according to level/years of schooling: low (basic/9 years or less), middle (upper secondary/10–12 years), and high education (tertiary or higher/>12 years). In Study III, education was dichotomised and both low and middle categories were combined.

5.3 Exposure variables

5.3.1 Family socioeconomic circumstances

All variables indicating family socioeconomic circumstances were registry-derived data. Family SES was mainly based on the education of parents. In Study II, several indicators of family socioeconomic circumstances from Statistics Finland, including those of grandparents, were used. All parents' and grandparents' data were obtained nearest to the year when the adolescent was aged 15 years. Except for data on education, parental data obtained more than five years away from the child's 15th birthday and data from those whose parents died prior to the AHLS study year were considered missing to ensure that only parental influences within adolescence were measured.

Data on grandfather and grandmother from either maternal or paternal side were combined. If both grandparents from the same side had data but reflected different information, the one with the higher category was used. In case of missing data from one grandparent, the available information from the other grandparent was used. Those without data on both grandfathers and grandmothers were categorised as "unknown" in the variables relating to grandparents in order to preserve sample size for analyses. Table 4 shows the socioeconomic circumstances of the parents and grandparents of the study participants according to the variables defined below.

5.3.1.1 Education level

Data on the education levels of both parents and grandparents followed the same categorisation used for the adolescents. In Study I and III, a singular measure of parents' education was created combining information from one's mother and

father. If the parents belonged to different categories, the highest was selected. If one parent had missing data, the available parent's data was used.

5.3.1.2 Dwelling ownership

Information on dwelling ownership of the father, mother, maternal and paternal grandparents were obtained. Data were based on the grounds for ownership of dwelling and classified as either owner-occupied (owned or had shares in the housing unit), or rented (living in a rented apartment).

5.3.1.3 Employment status

Employment status was based on the indicated data (employed, unemployed, unknown) about one's main activity. The category 'unemployed' also included those who had at least one month of unemployment during the preceding 12 months of the census. Because most grandparents had retired, this variable was measured for parents only.

Table 4. Socioeconomic circumstances of the parents and grandparents of the adolescent boys and girls in the study (N=41,833)

Socioeconomic circumstances of parents and grandparents		**Boy (n=19,509)**		**Girl (n=22,324)**	
		No.	**%**	**No.**	**%**
Education					
Father	Low	7927	40.63	9292	41.62
	Middle	8653	44.35	9828	44.02
	High	2627	13.47	2875	12.88
	Missing	302	1.55	329	1.47
Mother	Low	7480	38.34	8713	39.03
	Middle	10351	53.06	11771	52.73
	High	1665	8.53	1820	8.15
	Missing	13	0.07	20	0.09
Parents (combined)	Low	4425	22.68	5212	23.35
	Middle	11818	60.58	13529	60.60
	High	3261	16.72	3573	16.01
	Missing	5	0.03	10	0.04
Paternal grandparents	Low	8691	44.55	9952	44.58

	Middle	1900	9.74	2069	9.27
	High	507	2.60	564	2.53
	Unknown	8411	43.11	9739	43.63
Maternal grandparents	Low	8983	46.05	10162	45.52
	Middle	2031	10.41	2294	10.28
	High	460	2.36	478	2.14
	Unknown	8035	41.19	9390	42.06
Dwelling ownership					
Father	Rented	2787	14.29	3186	14.27
	Owner-occupied	15301	78.43	17416	78.01
	Missing	1421	7.28	1722	7.71
Mother	Rented	3276	16.79	3778	16.92
	Owner-occupied	15700	80.48	17966	80.48
	Missing	533	2.73	580	2.60
Paternal grandparents	Rented	1537	7.88	1827	8.18
	Owner-occupied	9078	46.53	10225	45.80
	Unknown	8894	45.59	10272	46.01
Maternal grandparents	Rented	1649	8.45	1905	8.53
	Owner-occupied	9398	48.17	10578	47.38
	Unknown	8462	43.37	9841	44.08
Employment status					
Father	Unemployed	2029	10.4	2401	10.76
	Employed	16431	84.22	18653	83.56
	Missing	1049	5.38	1270	5.69
Mother	Unemployed	2315	11.87	2608	11.68
	Employed	16961	86.94	19464	87.19
	Missing	233	1.19	252	1.13

5.3.2 Reserve capacity in adolescence

In Study I and II, reserve capacity was obtained from the AHLS data and measured in three distinct dimensions of intra- and interpersonal factors, specifically: perceived health, health-promoting behaviour and social support (Table 5). In Study III, it referred to an unobserved latent construct represented by a set of nine observed variables whose variables were interrelated within each dimension (Figure 5). Overall reserve capacity is referred to as either good or weak.

5.3.2.1 Perceived health

This dimension was measured by three items: reported chronic disease, injury or disability that restricts daily activities (no/yes); a summary index of weekly perceived stress symptoms namely, stomachaches, tension or nervousness, irritability or outbursts of anger, trouble falling asleep or waking at night, headache, trembling of hands, feeling tired or weak, and feeling dizzy, categorised as having none, one symptom/week, 2–3/week and 4–8/week; and, self-rated health categorised as very good, average/good or poor.

5.3.2.2 Health-promoting behaviour

This 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, or very active efficient exerciser.

5.3.2.3 Social support

The social support dimension was indicated by four variables: having a nuclear family (living with both parents or not); ease of talking about troubling issues (easy, difficult, very difficult) to the following persons: father, mother or friends. Those who did not have a father (5%), mother (1%) or friends (0.5%) were set to "very difficult." In Study II, these variables were dichotomised and the category "very difficult" was combined with "difficult".

Table 5. Reserve capacity characteristics of the adolescent boys and girls in the study (N=41,833)

Reserve capacity	**Boy (n=19,509)**		**Girl (n=22,324)**	
	No.	**%**	**No.**	**%**
Perceived health				
Chronic disease				
No	17,791	91.19	20,134	90.19
Yes	1,718	8,81	2,190	9.81
Perceived stress symptoms				
None	9897	50.73	7144	32.00
1/week	4181	21.43	5129	22.98
2-3/week	3937	20.18	6442	28.86
4-8/week	1494	7.66	3609	16.17
Self-rated health				
Very good	7465	38.26	6233	27.92
Average or good	11637	59.65	15568	69.74
Poor	328	1.68	458	2.05
Missing	79	0.40	65	0.29
Health-promoting behaviour				
Physical activity				
Very active efficient exerciser	5114	26.21	3930	17.60
Active efficient exerciser	6017	30.84	6623	29.67
Occasional/low efficient exerciser	4645	23.81	7224	32.36
Does not exercise	3671	18.82	4503	20.17
Missing	62	0.32	44	0.20
Regular toothbrushing				
Several times/day	3982	20.41	10831	48.52
About once/day	9737	49.91	9689	43.40
About 1-5 times/week or less	5689	29.16	1754	7.86
Missing	101	0.52	50	0.22
Social support				
Nuclear family				
Yes	15366	78.76	17040	76.33
No	4022	20.62	5173	23.17
Missing	121	0.62	111	0.5
Talking about issues to father				
Easy	10421	53.42	8157	36.54
Difficult	6010	30.81	8470	37.94
Very difficult/No father	2571	13.18	5314	23.80
Missing	507	2.60	383	1.72
Talking about issues to mother				
Easy	13705	70.25	16235	72.72
Difficult	4429	22.70	4743	21.25

Very difficult/No father	1037	5.32	1175	5.26
Missing	338	1.73	171	0.77
Talking about issues to friends				
Easy	14764	75.68	20078	89.94
Difficult	3558	18.24	1772	7.94
Very difficult/No friends	762	3.91	288	1.29
Missing	425	2.18	186	0.83

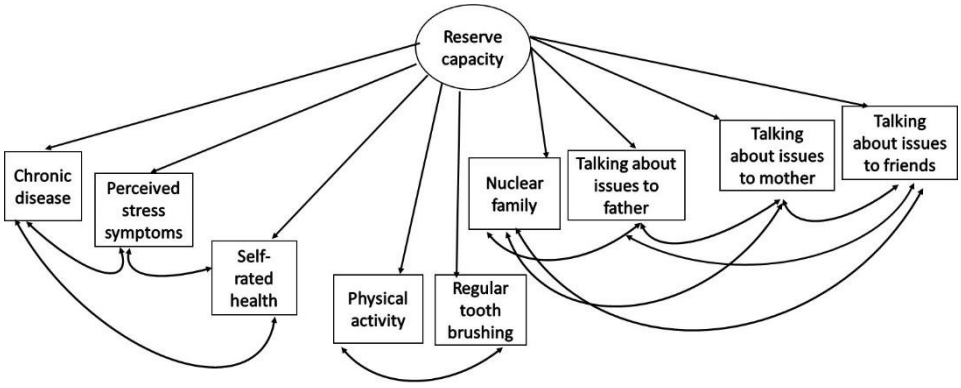

Figure 5. Measurement of reserve capacity as a latent variable in Study III

Note: The latent variable is enclosed in a circle with arrows indicating measurement from the actual variables (in boxes) collected in the study. Double-headed arrows under the boxes show covariances within variables in the same dimension.

5.3.3 Pubertal timing

To obtain an indicator of pubertal timing based on survey data, 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 previously 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 did not occur 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 did not occur by the time of enquiry (late). Close to 4000 (9.46%) adolescents aged 12 years at the time of surveys were excluded to minimise information bias since we cannot distinguish among them who

had average or late pubertal timing. Figure 6 presents the distribution of the adolescents according to pubertal timing categories.

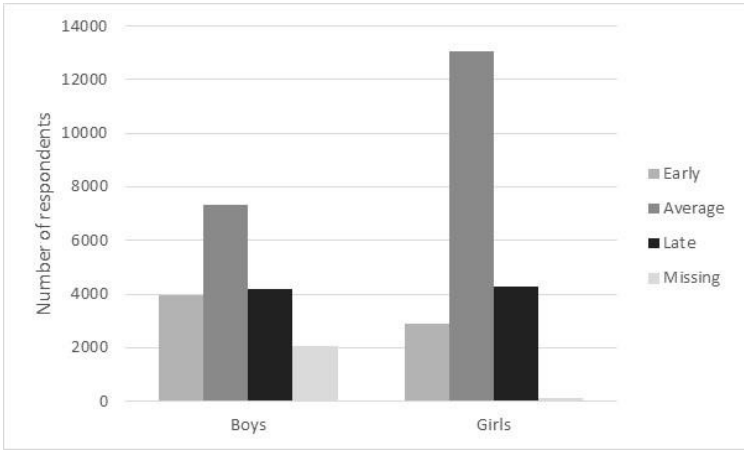

Figure 6. Distribution of adolescent boys (n=17,531) and girls (n=20345) according to pubertal timing

5.3.4 School achievement

Using survey data, adolescents were categorised as having: highest, 2nd highest, 2nd lowest, or lowest academic achievement. All respondents were asked to assess whether their end-of-term school report was much better (highest), slightly better (2nd highest), average (2nd lowest), slightly poorer or much poorer than the class average (lowest). For 12-14-year-olds (all in comprehensive schools), this self-assessment was the sole basis of their school achievement. For 16-18-year-olds, in addition to self-assessment of their school performance, school status (academic upper secondary school/vocational school/not attending school) was also considered. Their achievement was classified as follows: highest (in academic upper secondary school with better performance); 2nd highest (in vocational school with better performance or academic upper secondary school with average performance); 2nd lowest (in vocational school with poor to average performance or high school

with poor performance); and lowest (not in school). Figure 7 shows the distribution of adolescents according to school achievement.

In Study II, the number of categories used for school achievement was reduced to three and renamed as high, average or low. For all age groups, those previously classified in the highest and 2nd highest categories, comprised the new "high" and "average" groups, respectively. The two lowest categories were combined and reclassified as having "low" achievement.

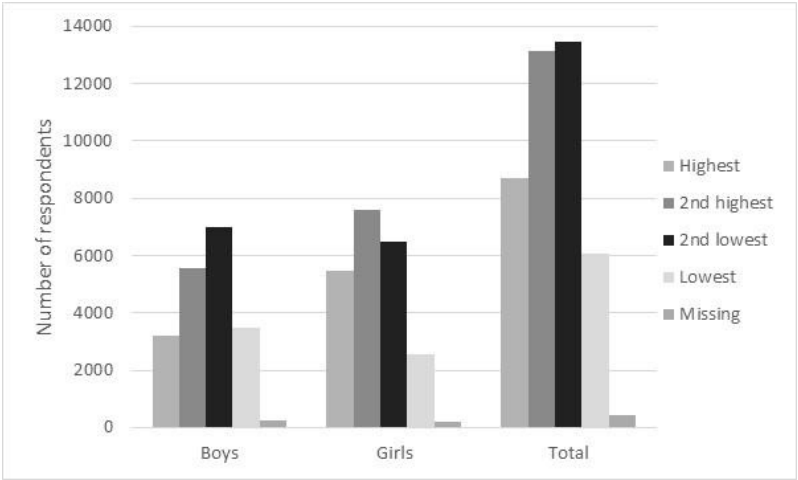

Figure 7. Distribution of adolescent boys (n=19,509) and girls (n=22,324) according to school achievement

5.4 Statistical analysis

5.4.1 Preliminary analysis

The relationships of the variables selected to comprise each dimension of reserve capacity were checked prior to multivariate analyses. Using Spearman's correlation, coefficients obtained indicated moderate positive correlations of variables per dimension. Cross-tabulations were also performed to check the associations of

variables in each dimension and Pearson chi-square results showed that they were significantly associated with each other.

The characteristics of the respondents included in the study were presented as frequencies and percentages for categorical variables and means for continuous variables. In Study II, the characteristics of AHLS respondents with unknown data for grandparents were compared with those of respondents with known data to assess whether selection bias occurred. Further analyses were also made to examine the effect of including this group in our study (See Section 5.4.2.2).

5.4.2 Multivariate methods

5.4.2.1 Cox regression

In Study I, we fitted a Cox proportional hazards models, separately for boys and girls, to determine the effect of family SES, reserve capacity variables and school achievement on mortality. Graphical assessments of proportional hazards were made using log-log survival curves for each independent variable. An example is illustrated in Figure 8 presenting survival curves of adolescents according to parents' education. Formally, adherence to the proportional hazards assumption was checked for each variable and globally, using a formal significance test based on the unscaled and scaled Schoenfeld residuals (UCLA: Statistical Consulting Group, 2015).

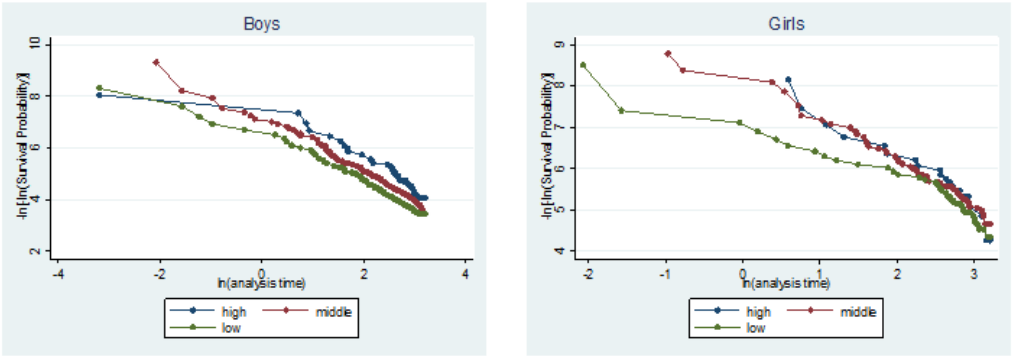

Figure 8. Example of survival curves of adolescents plotting survival probabilities (y-axis) versus categories of parental SES (x-axis)

First, a crude model, which considered family SES, each reserve capacity dimension and school achievement, was fitted to analyse each predictor's unadjusted effect on mortality risk (Model 1). Then, to study whether the reserve capacity variables modified the relationship between SES and mortality, all statistically significant ($p<0.05$) reserve capacity variables together with SES were included in a backward selection procedure until none could be deleted from the model (Model 2). Finally, school achievement was added (Model 3). An interaction term between parental education and school achievement was also tested.

Results were presented as hazard ratios (HRs) with 95% confidence interval (CI) estimates. Model fit was assessed using likelihood ratio tests and Akaike Information Criteria (AIC) (Bozdogan, 1987). Postestimation tests were done (checking of residuals and other plots) to ensure that the final models had the best fit. Respondents with missing data (5%) in one or more main variables studied were dropped from analysis. All analyses were performed using STATA version 12.1.

5.4.2.2 Multinomial logistic regression

In Study II, since educational attainment had three categories, we used multinomial logistic regression analyses to investigate its associations with family socioeconomic circumstances, reserve capacity variables and school achievement. Initially, bivariate analyses were done to determine the unadjusted effect of each predictor variable on education. Only those statistically significant ($p<0.05$) in bivariate analyses were included in the multivariate analyses. In both analyses, we adjusted for sex and age at the end of follow-up because of unequal follow-up times among the participants.

Three multivariate models were fitted through a backward elimination approach using low education as reference category for the outcome. The first model named Model 1 examined family SES variables; Model 2 included the Model 1 variables plus school achievement; and, Model 3 (final model) consisted of all statistically significant family SES variables, school achievement and reserve capacity variables. Another model, similar to the composition of the final model was fitted for a subset of the study population excluding data from those with unknown grandparents to check the consistency of our results.

Due to the numerous predictors considered in each model, statistical significance was set at $p<0.01$ for retaining variables in the models. Model fit was assessed using AIC values and likelihood ratio tests. The model parameters were presented as odds

ratios (ORs) with 95% CI estimates. All analyses were performed using STATA version 12.1.

5.4.2.3 Structural equation modelling

To analyse the mechanisms by which SES, puberty and reserve capacity influence education, we used structural equation models (SEM) in Study III. This enabled the inclusion of latent effects and testing of multiple pathways simultaneously (Grace & Bollen, 2005). SEM is composed of both a measurement model and a structural model. The measurement model is given by confirmatory factor analysis (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 & Muthén, 2012).

Our CFA model estimated the underlying construct of "reserve capacity" and created a single, general latent variable from the nine measured reserve capacity variables namely: 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 of perceived health, health-promoting behaviour and social support. We also fixed the variance of the latent variable at one to freely estimate the factor loadings for all the variables.

In Study III, the resulting estimates from the regression equations were probit coefficients interpreted as effects on a cumulative normal function of the probabilities that the response variable equals one (Muthén & Muthén, 2012). Unlike linear or logit regression coefficients, effect size from probit coefficients cannot be directly inferred as these coefficients give the change in the z-score or probit index for a one-unit change in the predictor. Interpretation is limited to the sign of the coefficient where a positive sign implies that an increase in the predictor leads to an increase in the predicted probability of the outcome. Conversely, a negative sign means that an increase in the predictor leads to a decrease in the predicted probability (UCLA: Statistical Consulting Group, 2019). We assigned a value of one to an outcome of low to middle education, thus, we predicted 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 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 & 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, Coughlan, & Mullen, 2008). Mplus 7.11 was used for both CFA and SEM analyses.

6 RESULTS

6.1 Mortality (Study I)

A total of 41,833 adolescent boys (46.64%) and girls (53.36%) were surveyed and followed-up until end of December 2009 or until their death, whichever occurred first. With 358,787 person-years of follow-up time among boys, the estimated mortality rate was 10.1 per 10,000 population. Majority (77.4%) of the 362 recorded deaths among boys were due to accidents. Among girls with 411,373 person-years of follow-up time, the estimated mortality rate was significantly lower ($p<0.001$) at 3.3 per 10,000 population. Out of 137 deaths recorded among girls, comparable proportions were caused by diseases (48.2%) and accidents (51.8%). Mean age at death among boys was 27.3 ± 6 years, with earliest death recorded at 15 years and oldest death at 41 years. Among girls, mean age of death was 27.8 ± 7 years, with ages at death ranging from 13 to 43 years.

6.1.1 Effect of family SES

In the crude model (Model 1), family SES significantly predicted the risk of mortality, with notable gradient effect in boys ($p<0.001$). When we adjusted for the effects of reserve capacity (Model 2) and school achievement (Model 3), mortality risks gradually decreased (Table 6), albeit a statistically significant gradient effect ($p<0.05$) of family SES still existed. Among boys whose parents had middle education, the unadjusted hazard ratio (HR) decreased by almost 19%: from 1.6 (95% CI 1.1-2.4) to an adjusted HR of 1.3 (95% CI 0.9-1.9), that was no longer statistically significant. A slightly greater reduction (27%) in effect of family SES was seen in boys whose parents had low education: from an unadjusted HR of 2.2 (95% CI 1.5-3.3) to an adjusted HR of 1.6 (95% CI 1.1-2.4). Both HR estimates were statistically significant, but the strength of association varied between the models. On the other hand, family SES did not predict the risk of mortality in girls (Table 6).

Table 6. Effect of family SES on mortality in boys and girls using Cox proportional hazards models presented as hazard ratios (HR) with 95% confidence interval (CI) estimates

Family SES	Model 1[a]	Model 2[b]	Model 3[c]
Boys			
High	1.0	1.0	1.0
Middle	1.6 (1.1-2.4)*	1.5 (1.0-2.1)*	1.3 (0.9-1.9)
Low	2.2 (1.5-3.3)**	1.9 (1.3-2.9)**	1.6 (1.1-2.4)*
Girls			
High	1.0	1.0	1.0
Middle	1.0 (0.6-1.8)	1.0 (0.6-1.7)	0.9 (0.5-1.5)
Low	1.4 (0.8-2.4)	1.2 (0.7-2.1)	1.0 (0.6-1.8)

*p < 0.05; **p < 0.001

[a]Model 1. Unadjusted effect of each predictor variable [b]Model 2. All significant reserve capacity variables from Model 1 and family SES. [c]Model 3. Model 2 variables and school achievement

6.1.2 Effects of reserve capacity and school achievement

In Model 3 described previously, both reserve capacity and school achievement in adolescence were found to be significant independent predictors of mortality. In boys, weak reserve capacity characterised by poor perceived health, poor health-promoting behavior and reduced social support increased their risk of death. Specific reserve capacity characteristics which predicted mortality in boys were having a chronic disease (HR 1.6, 95% 1.2-2.1); having more than four stress symptoms weekly (HR 1.7, 95% 1.2-2.3); not brushing one's teeth daily (HR 1.5, 95% 1.0-2.0); not having a nuclear family (HR 1.4, 95% 1.0-2.7); and not being able to talk to father easily (HR 1.6, 95% CI 1.2-2.1). Unlike in boys, poor health-promoting behaviour was not a predictor of mortality in girls. Only poor perceived health indicated by poor self-rated health (HR 4.5, 95% CI 2.2-9.4), and reduced social support measured by difficulty talking with one's father (HR 1.7, 95% 1.1-2.6) increased the girls' risk of death (Table 7).

School achievement had an inverse and graded relationship with mortality in boys. Those with low achievement had a double risk of death compared to boys with the highest school achievement. In girls, only those with the lowest achievement had a double risk of death (HR 2.4, 95% 1.4-4.1) relative to those in other categories. School achievement had no significant statistical interaction with family SES in both

boys and girls. Table 7 presents the detailed estimates for the effects of these adolescent factors on mortality based on Model 3.

Table 7. Effects of reserve capacity and school achievement on mortality in boys and girls using Cox proportional hazards models presented as hazard ratios (HR) with 95% confidence interval (CI) estimates

Predictors in adolescence	**Boys**	**Girls**
Perceived health		
Chronic disease		
No	1.0	n.s.
Yes	1.6 (1.2-2.1)**	
Perceived stress symptoms		
None	1.0	n.s.
1/week	1.1 (0.8-1.5)	
2-3/week	1.1 (0.8-1.5)	
4-8/week	1.7 (1.2-2.3)**	
Self-rated health		
Very good	n.s.	1.0
Average or good		1.4 (0.9-2.1)
Poor		4.5 (2.2-9.4)**
Health-promoting behaviour		
Regular tooth brushing		
Several times/day	1.0	n.s.
Once/day	1.1 (0.8-1.6)	
1-5 times/week or less	1.5 (1.0-2.0)*	
Social support		
Nuclear family		
Yes	1.0	n.s.
No	1.4 (1.0-1.7)*	
Talking about issues to father		
Easy	1.0	1.0
Difficult	1.2 (1.0-1.6)	1.3 (0.8-1.9)
Very difficult/No father	1.6 (1.2-2.1)**	1.7 (1.1-2.6)*
School achievement		
Highest	1.0	1.0
2nd highest	1.6 (1.0-2.4)*	1.0 (0.6-1.6)
2nd lowest	2.0 (1.3-3.1)**	1.0 (0.6-1.6)
Lowest	2.3 (1.4-3.5)**	2.4 (1.4-4.1)**

*$p < 0.05$; **$p < 0.001$; n.s. not significant

6.2 Educational level in adulthood (Study II and III)

Eleven (0.03%) respondents were excluded from the Study I population due to issues on measurement of highest education obtained over the follow-up period. In Study II, half of the total adolescent population obtained a middle education (55.2%); a third (35.7%) had high education; and less than a tenth (9.1%) had low education in adulthood.

In Study III, we used the same educational outcome but employed another statistical procedure to explore operating pathways in the relationship between family SES and education. Adolescents aged 12 years at the time of surveys, comprising about 10% of the Study II population were excluded due to measurement issues in one of the key variables for this age group. Thus, the final total sample population used in Study III was 37,876 adolescents. Additionally, we dichotomised the measurement of education because using three categories posed analytical challenges. Our data did not support the proportional odds assumption required in using SEM for ordinal outcomes. On the other hand, the categories cannot be treated as unordered or multinomial as done in Study II because SEM did not allow assessment of indirect effects for multinomial outcomes. One of the main hypotheses of Study III was to estimate the indirect effects of family SES on education. Thus, the low and middle categories of education were combined. Figure 9 presents the distribution of adolescents based on these two categories of education.

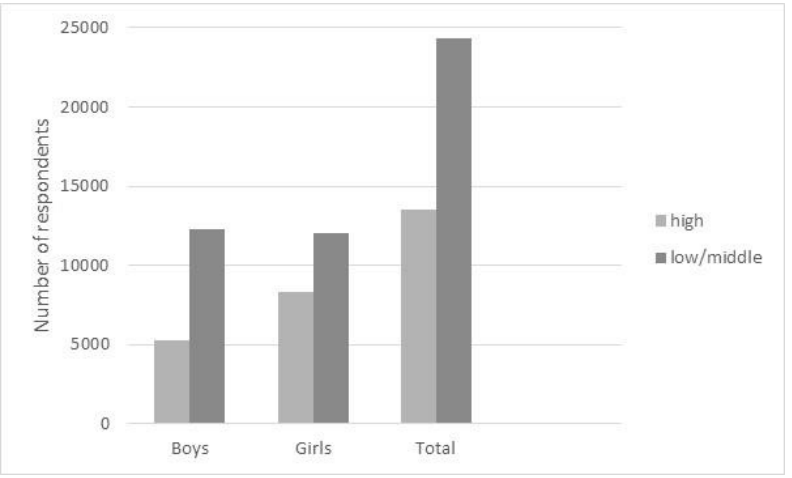

Figure 9. Distribution of adolescent boys (n=17,531) and girls (n=20,345) according to education in Study III

6.2.1 Effect of family socioeconomic circumstances

In Study II, results of bivariate analyses (Table 8) showed that all variables indicating family socioeconomic circumstances, after adjusting for sex and age at end of follow-up, were associated with education. The odds of attaining either middle or high education in adulthood were higher in adolescents whose parents and grandparents had the same education level compared to those born into families with low education. Similarly, the likelihood of obtaining either middle or high education compared to low, increased when parents and grandparents owned their dwellings and also when parents were employed. The family SES variables, particularly education, had graded and stronger associations with a high education than a middle education.

In multivariate analyses, when all variables including reserve capacity and school achievement were added into the model, most variables related to grandparents lost their effect and only maternal grandparents' dwelling ownership remained significantly associated with the grandchildren's education. The direction of associations observed for family SES variables were similar to those estimated in bivariate analyses, but the odds ratios were markedly reduced. Still, family

socioeconomic circumstances were more strongly associated with a high education than a middle education (Table 8).

Table 8. Bivariate and multivariate associations of each family SES variable with education level in adulthood (using low education as reference category), adjusting for sex and age at end of follow-up in Study II

Family socioeconomic circumstances	Bivariate analyses Odds Ratios (95% Confidence Intervals)		Multivariate analyses (final model) Odds Ratios (95% Confidence Intervals)	
	Middle	High	Middle	High
Education				
Father				
Low	1.0	1.0	1.0	1.0
Middle	1.3 (1.3-1.5)***	2.1 (1.9-2.3)***	1.2 (1.1-1.3)***	1.5 (1.4-1.7)***
High	1.8 (1.5-2.1)***	8.3 (7.0-9.8)***	1.1 (0.9-1.4)	2.6 (2.1-3.1)***
Mother				
Low	1.0	1.0	1.0	1.0
Middle	1.4 (1.3-1.5)***	2.6 (2.4-2.8)***	1.3 (1.2-1.4)***	1.8 (1.6-2.0)***
High	1.9 (1.5-2.4)***	9.4 (7.6-11.6)***	1.3 (1.0-1.6)	2.5 (2.0-3.2)***
Paternal Grandparents				
Low	1.0	1.0	n.s.	n.s.
Middle	1.1 (0.9-1.2)	1.5 (1.3-1.7)***		
High	1.2 (0.9-1.6)	2.9 (2.1-4.0)***		
Unknown	0.8 (0.8-0.9)***	0.8 (0.8-0.9)***		
Maternal Grandparents				
Low	1.0	1.0	n.s.	n.s.
Middle	1.0 (0.9-1.2)	1.4 (1.2-1.6)***		
High	1.2 (0.8-1.6)	3.1 (2.2-4.3)***		
Unknown	0.8 (0.8-0.9)***	0.9 (0.8-0.9)**		
Dwelling ownership				
Father				
Rented	1.0	1.0	1.0	1.0
Owner-occupied	2.1 (1.9-2.3)***	4.0 (3.6-4.4)***	1.4 (1.2-1.5)***	1.7 (1.5-2.0)***
Mother				
Rented	1.0	1.0	1.0	1.0
Owner-occupied	2.2 (2.0-2.4)***	4.3 (3.9-4.7)***	1.3 (1.1-1.5)***	1.5 (1.3-1.7)***
Paternal Grandparents				
Rented	1.0	1.0	n.s.	n.s.
Owner-occupied	1.3 (1.1-1.5)***	1.6 (1.4-1.9)***		

Unknown	1.0 (0.9-1.2)	1.2 (1.0-1.3)*		
Maternal Grandparents				
Rented	1.0	1.0	1.0	1.0
Owner-occupied	1.5 (1.3-1.8)***	2.1 (1.9-2.5)***	1.3 (1.1-1.5)**	1.5 (1.3-1.8)***
Unknown	1.2 (1.0-1.3)*	1.4 (1.3-1.7)***	1.0 (0.8-1.1)	1.0 (0.9-1.2)
Employment status				
Father				
Unemployed	1.0	1.0	1.0	1.0
Employed	1.4 (1.3-1.6)***	2.4 (2.2-2.8)***	1.0 (0.9-1.2)	1.2 (1.1-1.4)**
Mother				
Unemployed	1.0	1.0	1.0	1.0
Employed	1.6 (1.4-1.8)***	2.4 (2.1-2.6)***	1.2 (1.1-1.4)***	1.4 (1.2-1.5)***

Significance levels: * p<0.05, **p<0.01 ***p<0.001 n.s. not significant

In Study III, the direct pathways from family SES and education were statistically significant. Low family SES, based on parents' education significantly increased the probability of low education in both boys ($\beta = 0.16$, $p<0.001$) and girls ($\beta = 0.14$, $p<0.001$). Significant indirect effects of low family SES on education were also observed through weak reserve capacity (boys: $\beta = 0.01$; girls: $\beta = 0.02$; $p<0.001$) and poor school achievement pathways (boys: $\beta = 0.14$; girls: $\beta = 0.12$; $p<0.001$).

6.2.2 Effects of reserve capacity and school achievement

Bivariate analyses of the relationships between reserve capacity variables and education in Study II showed that all dimensions predicted education (Table 9). Graded associations of perceived stress symptoms, self-rated health, physical activity and frequency of tooth brushing with education were observed, where positive categories increased the likelihood of getting either middle or high education. In the social support dimension, however, talking about issues either to mother or friends was not significantly related to any category of the outcome. Adolescents with high achievement in school were more likely to attain a middle or high education compared to those with low achievement. School achievement and the statistically significant reserve capacity variables were more strongly associated with a high education than a middle education. During multivariate analyses, reserve capacity and school achievement showed independent associations with education; although, slightly reduced ORs were estimated and self-rated health and talking to father variables became statistically insignificant. As in bivariate analyses, school

achievement remained the strongest predictor of education even when the SES and reserve capacity variables were included in the final model (Table 9).

Table 9. Bivariate and multivariate associations of reserve capacity and school achievement with education level in adulthood (using low education as reference category), adjusting for sex and age at end of follow-up in Study II

Reserve capacity and school achievement	Bivariate analyses Odds Ratios (95% Confidence Intervals)		Multivariate analyses (final model) Odds Ratios (95% Confidence Intervals)	
	Middle	High	Middle	High
Perceived health				
Chronic disease				
Yes	1.0	1.0	1.0	1.0
No	1.2 (1.1-1.4)**	1.3 (1.1-1.5)***	1.3 (1.1-1.4)***	1.3 (1.2-1.5)***
Perceived stress symptoms				
4-8/week	1.0	1.0	1.0	1.0
2-3/week	1.3 (1.2-1.5)***	1.6 (1.4-1.8)***	1.2 (1.1-1.4)**	1.5 (1.3-.1.7)***
1/week	1.6 (1.4-1.8)***	1.8 (1.6-2.1)***	1.4 (1.3-1.7)***	1.6 (1.4-1.9)**
None	1.7 (1.5-2.0)***	2.0 (1.8-2.3)***	1.6 (1.4-1.8)***	1.8 (1.5-2.0)***
Self-rated health				
Poor	1.0	1.0	n.s.	n.s.
Average or good	1.4 (1.1-1.8)**	1.5 (1.2-2.0)**		
Very good	1.5 (1.2-1.9)**	1.9 (1.5-2.5)***		
Health-promoting behaviour				
Physical activity				
Does not exercise	1.0	1.0	1.0	1.0
Occasional/low efficient exerciser	1.3 (1.2-1.5)***	1.8 (1.6-2.0)***	1.2 (1.0-1.3)**	1.4 (1.2-1.6)***
Active efficient exerciser	1.4 (1.2-1.6)***	2.3 (2.1-2.6)***	1.2 (1.0-1.3)**	1.5 (1.4-1.8)***
Very active efficient exerciser	1.6 (1.4-1.8)***	2.9 (2.5-3.3)***	1.2 (1.1-1.4)**	1.6 (1.4-1.8)***
Regular tooth brushing				
<1-5 times/week	1.0	1.0	1.0	1.0
About once/day	1.7 (1.6-1.9)***	3.2 (2.8-3.5)***	1.5 (1.3-1.6)***	2.1 (1.9-2.4)***
Several times/day	1.9 (1.7-2.2)***	4.9 (4.4-5.5)***	1.5 (1.4-1.7)***	2.5 (2.2-2.9)***
Social support				
Nuclear family				
Yes	1.0	1.0	1.0	1.0
No	2.2 (2.0-2.4)***	3.8 (3.4-4.2)***	1.7 (1.5-1.8)***	2.3 (2.0-2.5)***

Talking about issues to father				
Difficult/No father	1.0	1.0	n.s.	n.s.
Easy	1.1 (1.0-1.2)	1.1 (1.0-1.2)*		
Talking about issues to mother				
Difficult/No mother	1.0	1.0	n.s.	n.s.
Easy	1.1 (1.0-1.2)	1.1 (1.0-1.2)		
Talking about issues to friends				
Difficult/No friends	1.0	1.0	n.s.	n.s.
Easy	1.0 (0.9-1.1)	0.9 (0.8-1.0)		
School achievement				
Low	1.0	1.0	1.0	1.0
Average	3.0 (2.7-3.3)***	10.7 (9.6-12.0)***	2.6 (2.3-2.9)***	7.9 (7.0-8.9)***
High	5.6 (4.5-7.0)***	53.6 (43.0-66.8)***	4.6 (3.7-5.8)***	32.4 (25.9-40.6)***

Significance levels: * p<0.05, **p<0.01 ***p<0.001 n.s. not significant

In Study III, reserve capacity was measured as a latent variable where variables within each dimension of perceived health, health-promoting behaviour and social support were hypothesised to have covariances (see Figure 5). The CFA model of reserve capacity fitted our data well in both boys and girls based on estimated RMSEA and CFI values. Also, all factor loadings of the nine variables were statistically significant. Larger factor loadings reflected a greater degree of relationship with the latent variable and the positive sign of coefficients implied direct relationship with latent reserve capacity (Table 10). The grouped variables had statistically significant covariances suggesting that the observed variables were related within each dimension.

Table 10. Results from confirmatory factor analyses (CFA) of reserve capacity model regressed on nine observed variables presented as standardized (β) coefficients in Study III

Observed variable	**Boys**		**Girls**	
	B	**p-value**	**B**	**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**	
	B	**p-value**	**B**	**p-value**
Perceived health				
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
Health-promoting behavior				
Physical activity with				
Regular tooth brushing	0.12	<0.001	0.10	<0.001
Social support				
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
Fit indices:				
RMSEA	0.04		0.03	
CFI	0.97		0.97	

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Direct pathways from reserve capacity to education were estimated. A weak reserve capacity in adolescence among boys ($\beta = 0.10$, $p<0.001$) and girls ($\beta = 0.12$, $p<0.001$), significantly increased the probability of attaining low to middle education. A low school achievement similarly increased the probability of attaining low to middle education level in both boys ($\beta = 0.52$; $p<0.001$) and girls ($\beta = 0.48$; $p<0.001$).

6.2.3 Effect of unknown grandparents

The proportion of adolescents with unknown grandparents' data by education level was comparable to those of adolescents whose grandparents had low education and rented dwellings. In terms of other variables, the pattern of distribution found in adolescents with unknown grandparents followed the distributions obtained in the total population.

As seen in Table 3, due to issues on data linkage, a large proportion of grandchildren in the surveys had unknown data for grandparents. We did not want to remove data from this group, thus, unknown categories were created for grandparents to include their data in the analyses. We then assessed how the associations were affected when only those with available data were used. Table 11 compares the results of multivariate analyses using the full sample and a subset of the population which excluded data from those with unknown grandparents. Slightly increased associations between education and some of the predictors (parents' education, school achievement and perceived stress symptoms in the perceived health dimension) were seen in the sample which excluded unknown grandparents. Also, the father's employment status and chronic disease in the perceived health dimension no longer predicted education in this group. Overall results, however, showed the same directions and magnitude of associations as the original analyses which included data on unknown grandparents (Table 11).

Table 11. Comparison of results of multivariate analyses on associations with education (using low education as reference category), adjusting for sex and age at end of follow-up, between population with and without unknown grandparents' data in Study II

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence	Multivariate analyses (N=36,517) Odds Ratios (95% Confidence Intervals)		Multivariate analyses (N=15,328)[a] Odds Ratios (95% Confidence Intervals)	
	Middle	Middle	Middle	High
Education				
Father				
Low	1.0	1.0	1.0	1.0
Middle	1.2 (1.1-1.3)**	1.5 (1.4-1.7)**	1.3 (1.1-1.5)**	1.7 (1.4-1.9)**
High	1.1 (0.9-1.4)	2.6 (2.1-3.1)**	1.2 (0.9-1.6)	3.1 (2.2-4.2)**
Mother				

Low	1.0	1.0	1.0	1.0
Middle	1.3 (1.2-1.4)**	1.8 (1.6-2.0)**	1.2 (1.1-1.4)*	1.8 (1.6-2.2)**
High	1.3 (1.0-1.6)	2.5 (2.0-3.2)**	1.8 (1.2-2.8)*	3.5 (2.3-5.4)**
Dwelling ownership				
Father				
Rented	1.0	1.0	1.0	1.0
Owner-occupied	1.4 (1.2-1.5)**	1.7 (1.5-2.0)**	1.4 (1.1-1.6)*	1.7 (1.4-2.2)**
Mother				
Rented	1.0	1.0	1.0	1.0
Owner-occupied	1.3 (1.1-1.5)**	1.5 (1.3-1.7)**	1.3 (1.1-1.6)*	1.6 (1.2-2.0)**
Maternal Grandparents				
Rented	1.0	1.0	1.0	1.0
Owner-occupied	1.3 (1.1-1.5)*	1.5 (1.3-1.8)**	1.2 (1.0-1.4)	1.4 (1.1-1.7)*
Unknown	1.0 (0.8-1.1)	1.0 (0.9-1.2)	-	-
Employment status				
Father				
Unemployed	1.0	1.0	n.s.	n.s.
Employed	1.0 (0.9-1.2)	1.2 (1.1-1.4)*		
Mother				
Unefemployed	1.0	1.0	1.0	1.0
Employed	1.2 (1.1-1.4)**	1.4 (1.2-1.5)**	1.2 (1.0-1.4)	1.3 (1.1-1.6)*
Perceived health				
Chronic disease				
Yes	1.0	1.0	n.s.	n.s.
No	1.3 (1.1-1.4)**	1.3 (1.2-1.5)**		
Perceived stress symptoms				
4-8/week	1.0	1.0	1.0	1.0
2-3/week	1.2 (1.1-1.4)*	1.5 (1.3-.1.7)**	1.5 (1.2-1.9)**	2.2 (1.7-2.8)**
1/week	1.4 (1.3-1.7)**	1.6 (1.4-1.9)**	1.5 (1.2-1.9)**	2.1 (1.7-2.7)**
None	1.6 (1.4-1.8)**	1.8 (1.5-2.0)**	1.8 (1.5-2.2)**	2.7 (2.2-3.4)**
Health-promoting behaviour				
Physical activity				
Does not exercise	1.0	1.0	1.0	1.0
Occasional/low efficient exerciser	1.2 (1.0-1.3)*	1.4 (1.2-1.6)**	1.1 (0.9-1.3)	1.2 (1.0-1.5)
Active efficient exerciser	1.2 (1.0-1.3)*	1.5 (1.4-1.8)**	1.0 (0.9-1.2)	1.3 (1.1-1.6)*
Very active efficient exerciser	1.2 (1.1-1.4)*	1.6 (1.4-1.8)**	1.1 (0.9-1.4)	1.5 (1.2-1.9)**
Regular tooth brushing				
<1-5 times/week	1.0	1.0	1.0	1.0
About once/day	1.5 (1.3-1.6)**	2.1 (1.9-2.4)**	1.4 (1.2-1.6)**	2.1 (1.7-2.5)**
Several times/day	1.5 (1.4-1.7)**	2.5 (2.2-2.9)**	1.5 (1.2-1.8)**	2.6 (2.1-3.2)**

Social support				
Nuclear family				
No	1.0	1.0	1.0	1.0
Yes	1.7 (1.5-1.8)**	2.3 (2.0-2.5)**	1.8 (1.6-2.1)**	2.6 (2.3-3.1)**
School achievement				
Low	1.0	1.0	1.0	1.0
Average	2.6 (2.3-2.9)**	7.9 (7.0-8.9)**	2.6 (2.1-3.1)**	7.6 (6.2-9.2)**
High	4.6 (3.7-5.8)**	32.4 (25.9-40.6)**	6.4 (4.1-10.0)**	42.0 (26.9-65.4)**

Significance levels: * p<0.01, **p<0.001, n.s. not significant

[a] unknown grandparents' data excluded in analysis

Multivariate analyses of final model: Family SES + school achievement + reserve capacity variables

6.2.4 Effect of puberty

In Study III, the effect of a biological pathway on education was assessed using pubertal timing. Table 12 presents how puberty effects varied with family SES in conjunction with other adolescent variables. In both boys and girls, delayed pubertal timing generally increased the probability of obtaining low to middle education. But, puberty ceased to have statistically significant associations with education when school achievement was added into both boys' and girls' models.

Table 12. Direct effects of family SES and biological pathway on education in a structural equation model adjusted for age at baseline and follow-up, presented as standardized (β) coefficients

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

Significance levels: * p<0.01, **p<0.001, n.s. not significant

[a] Model with family SES and puberty [b]Model 1 plus reserve capacity [c]Model 2 plus school achievement

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6.3 Family SES and the intermediate adolescent pathways (Study III)

Overall relationships of family SES and the adolescent pathways namely, reserve capacity, school achievement and puberty based on results of Study III are depicted in Figures 10 and 11. Both structural equation models, adjusted for the effects of ages at baseline and at follow-up, had a good fit for both population groups based on the calculated fit indices. The illustrations only presented estimates of direct effects as well as the factor loadings of measured variables on latent reserve capacity. Estimated coefficients relating to the age variables as well as the covariances among adolescent pathways were omitted for simplicity of presentations.

The hypothesis that family SES directly affected the intermediate adolescent pathways was fully supported by the model in boys (Figure 10). Low family SES significantly increased ($p<0.001$) the probability of delayed pubertal timing ($\beta = 0.03$); weak reserve capacity ($\beta = 0.10$); and low school achievement ($\beta = 0.26$). The model in girls (Figure 11) partially supported this hypothesis as family SES had significant direct effects only on reserve capacity ($\beta = 0.13$, $p<0.001$), and school achievement ($\beta = 0.25$, $p<0.001$). Additionally, in girls, a low family SES decreased the probability of delayed pubertal timing ($\beta = -0.02$, $p=0.05$) but this was not statistically significant.

The intermediate pathways also had statistically significant covariance estimates indicating the direction of relationship with each other. Pubertal timing had a negative relationship with reserve capacity in both boys ($\beta = -0.11$, $p<0.001$), and girls ($\beta = -.012$, $p<0.001$). But it had a positive relationship with school achievement in boys ($\beta = 0.05$, $p<0.001$). 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 in both boys ($\beta = 0.35$, $p<0.001$) and girls ($\beta = 0.37$, $p<0.001$).

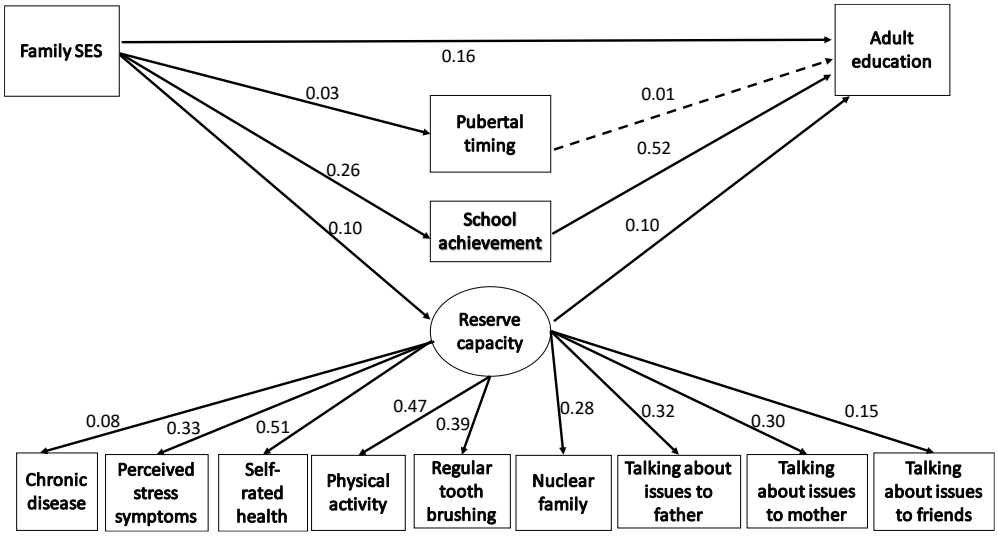

Figure 10. Boys: Structural equation model depicting relationships among family SES, pubertal timing, school achievement and reserve capacity in adolescence, and education level (RMSEA=0.05; CFI=0.90)

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

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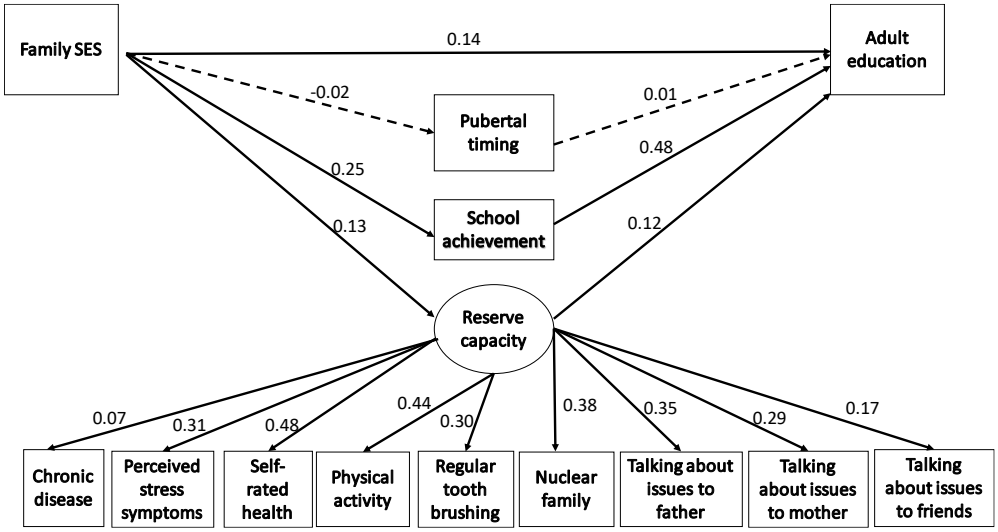

Figure 11. Girls: Structural equation model depicting relationships among family SES, pubertal timing, school achievement and reserve capacity in adolescence, and education level (RMSEA=0.04; CFI=0.91)

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

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7 DISCUSSION

7.1 Summary of main findings

Our longitudinal study determined the existence of socioeconomic inequalities in mortality and education. We also found that reserve capacity, pubertal timing and school achievement in adolescence influenced one's health and socioeconomic trajectories. Further, we have confirmed the effect of grandparents' SES on their grandchildren's education.

Low family SES significantly predicted mortality in boys but not in girls. Reserve capacity independently predicted mortality in adolescents but gender differences were observed in the relationships of the dimension-specific variables with mortality. For perceived health, the presence of chronic disease and higher number of perceived stress symptoms increased mortality risks in boys. On the other hand, poor self-rated health increased those in girls. Health-promoting behaviour, indicated by oral hygiene, as well as social support in terms of family structure were significant predictors of mortality only in boys. School achievement independently predicted mortality in adolescents. Moreover, it had gradient effects on boys' mortality, where the lowest achievers had more than double risk of death compared to the highest achievers. It had no statistical interaction with family SES. Both reserve capacity and school achievement reduced the effect of low family SES on mortality in boys.

Multiple measures of family SES, including maternal grandparents' SES, significantly predicted the education of the adolescents. But these had stronger effects on high education than middle education. The odds of getting either a middle or high education was higher when parents did not have low education, were employed, and owned their dwellings. A good reserve capacity, consisting of good perceived health based on the absence of chronic disease and fewer number of stress symptoms, health-promoting behaviours of efficient exercising and regular tooth brushing, and social support from a nuclear family structure, independently predicted both middle and high education. Similarly, those with a high achievement

in school more than quadrupled their probability of attaining middle education and markedly increased the likelihood of attaining a high education (OR: 32.4, 95% CI: 25.9-40.6) compared to low achievers. Both reserve capacity and school achievement also reduced the associations of family SES with education.

Using SEM, we demonstrated that a low family SES increased the probability of low education directly and indirectly, through weak reserve capacity and low school achievement pathways. Puberty was not a significant biological pathway in the association between family SES and education once school achievement in adolescence was accounted for. In addition, low family SES increased the probabilities of weak reserve capacity and low school achievement in adolescence and that of delayed pubertal timing in boys. Both reserve capacity and school achievement had direct effects on education. All adolescent intermediate pathways were significantly interrelated.

7.2 Role of family socioeconomic status including grandparents

7.2.1 Socioeconomic inequalities in mortality

We found socioeconomic inequalities in boy's mortality risks where those with low family SES had double risk of dying compared to those with high SES. Our results replicated those of other studies done within the region; although, only the study of Remes and colleagues (2010) used parental SES during adolescence while others used family SES at birth (Juárez et al., 2016) and adult SES (Mackenbach et al., 2015), respectively. Thus, we have also added evidence on the constancy of effect of low family SES on mortality, regardless of the measurement timing in the life-course.

Notably, the observed gender differences in mortality rates, including the attenuated SES effects on girls' mortality risks in our study were consistent with demonstrated global patterns using different SES indicators (Phillips & Hamberg, 2015). The gendered nature of health had been attributed to the underlying differences in experiences and behaviours associated with gender roles in particular social, cultural, political and economic settings, aside from inherent genetic and biological differences (Phillips & Hamberg, 2015).

Our findings also lend support to the social causation theory and family investment model which postulated that social conditions, such as family SES, caused differences in children's health and development (Conger et al., 2010; Martin et al., 2010), consequently predicting their mortality. Indeed, economic hardship in the family may translate to less investments made for children's health (Conger et al., 2010). Thus, societal approaches, such as government policies and programs addressing these social conditions, are needed to support low SES families and reduce health inequalities and ultimately, mortality inequalities. For instance, low SES families supported by the government through housing assistance, income supplementation and employment benefits were shown to have improved the health and behavioural outcomes of their children (Hoagwood et al., 2018).

7.2.2 Socioeconomic inequalities in education

Educational inequalities by family SES existed in our study as those from high SES families had increased likelihood of attaining higher education than their disadvantaged counterparts. Despite the expansion of educational opportunities over the years (OECD, 2018), socioeconomic inequalities in education still existed because low family SES was already a barrier towards enrolment in higher education (Brekke, 2015; Becker & Hecken, 2009). Our results were consistent with previous research which found strong associations between parents' SES and children's education (Becker & Hecken, 2009; Bird, 2007; Brekke, 2015; Fergusson et al., 2008; Koivusilta et al., 2013; Suhonen & Karhunen, 2019) as well as with evidence which showed several countries with population disparities in completion of tertiary education based on parents' education (OECD, 2018). We also reliably reproduced the direct pathway from family SES to educational attainment found by Fergusson and colleagues (2008) in a longitudinal study in New Zealand using the same modelling approach.

Family SES was similarly proposed to have operated via parental investments and values to influence educational attainment of children. For instance, high SES parents provided more investments on education through monetary transfers for education-related fees than low SES parents did (Albertini & Radl, 2012; Conger et al., 2010; Martin et al., 2010). Additionally, higher SES families, in contrast with lower SES families, placed greater value on education and had higher educational aspirations for their children (Albertini & Radl, 2012; Fergusson et al., 2008; Martin

et al., 2010). Interestingly, Albertini and Radl (2012), in their analysis of financial transfer behaviours of parents, concluded that aside from an altruistic desire to provide for the good of the children, status reproduction to prevent downward social mobility of their children was the main driving force for the financial transfers.

We found stronger effects of family SES on high education than middle education. This replicated the results of Becker and Hecken (2009) in Germany who also showed that the impact of social origin was greater for higher education than vocational training. They found that along with family SES, the type of post-secondary education to be pursued was dependent on individual decisions, heavily influenced by educational motivations and expected costs for university education (Becker & Hecken, 2009).

Furthermore, our results implicating intergenerational transmission of SES based on significant associations of grandparental SES with the education of grandchildren updated previous evidence in Finland which found weak intergenerational effects, particularly for economically disadvantaged grandparents (Erola & Moisio, 2007). Chan and Boliver (2013) stated that grandparental effects may occur when wealthy grandparents also make financial transfers for their grandchildren's education. The significant effect of maternal grandparents' dwelling ownership on their grandchildren's education may be a signal for transfer of wealth to grandchildren, parallel to the results of Hällsten and Pfeffer (2017) in Sweden. These also supported the theories of Coall and Hertwig (2010) about inherent tendencies of maternal grandparents to transfer more resources to grandchildren.

Apart from these familial processes, studies have also shown that family SES influenced children's educational attainment through children's own mediating characteristics, supporting the social selection theory (Conger et al., 2010). This confirmed our findings of indirect pathways of SES to educational attainment. In our study, family SES had direct paths to all adolescent pathways. Thus, underlying differences in how adolescents navigate through these pathways, along with other intra- or extra- familial and school factors unmeasured in our study, probably contributed to inequalities in educational trajectories. Extending our results further imply that since adolescents' future SES are shaped by these, we may foresee transmission effects on human capital development of their future children as well. This reflects the interactionist model of SES and development across generations (Martin et al., 2010).

7.3 Understanding the adolescent intermediate pathways

In recent years, the importance of adolescence for ensuring successful transitions into adulthood (Johnson et al., 2011), as well as meeting global health agendas, (Sawyer et al., 2012) were recognized. Exploring adolescent pathways linking SES with health and development yields crucial implications for interventions which could reduce health and socioeconomic inequalities in posterity.

7.3.1 Role of reserve capacity

The variables comprising reserve capacity in our study were proxy indicators of intra- and interpersonal psychosocial resources within dimensions of perceived health, health-promoting behaviour and social support. Page and colleagues (2009) showed that perceptions of health among adolescents were more related to their psychosocial functioning than aspects of their physical health. Additionally, several psychosocial attributes namely, coping planning, perceived control (Pakpour & Sniehotta, 2012), and self-efficacy (Scheerman et al., 2016), had been associated with dental brushing behaviour while self-efficacy had also been connected with physical activity (Feltz & Magyar, 2006; Robbins et al., 2004). Indeed, studies on socioeconomic inequalities of health showed that psychosocial factors partly operated via behavioural factors (Moor et al., 2014; van Oort et al., 2005), rationalising our expanded definition of reserve capacity. While social support had been commonly used in reserve capacity studies among adults (Matthews et al. 2010) and adolescents (Brekke, 2015; Due et al., 2003; Moor et al., 2014; Olsson et al., 2013), the other dimensions used in our study add new empirical evidence on the composition of reserve capacity in adolescence.

Good reserve capacity in adolescence mediated and reduced the effect of low family SES on mortality in boys. Moreover, it was an independent predictor of mortality in both boys and girls. Our findings suggested that good perceived health and having health-promoting behaviours and social support in adolescence may cumulatively protect one's health in later life, reducing mortality risks. These supported the developmental role of reserve capacity (Matthews & Gallo, 2011) and underscored the importance of the psychosocial pathway in the SES-health linkage (Gallo & Matthews, 2003; Matthews et al., 2010).

Mackenbach and colleagues (2002) determined that robust associations between perceived health and mortality were weakly explained by psychosocial factors. They also surmised that health perceptions probably included assessment of current health status as well as undiagnosed disease states, summatively capturing aspects related to survival (Mackenbach et al., 2002).

Our results showed that low frequency of tooth brushing in adolescence increased mortality risks in boys. Among adults, those who brushed their teeth less than once a day had higher levels of inflammation markers and increased risks for cardiovascular disease and fatality than those who had good dental brushing behaviour (de Oliveira, Watt & Hamer, 2010). A meta-analytic study found that several psychosocial resources such as coping and action planning (anticipation of barriers and ways to overcome these and perform the behaviour); intention (motivation to exert performance); social influences (pressure from others to perform); and self-efficacy (confidence in the ability to perform) were underlying tooth brushing behaviour during adolescence (Scheerman et al., 2016). These characteristics increased personal competence, facilitating engagement in other positive, protective health behaviours (Schwarzer & Luszczynska, 2006). Conversely, the boys' poor dental brushing behaviour during adolescence may have been carried over into adulthood, consequently resulting in other morbidities and mortality later in life.

Unlike other dimensions of reserve capacity, social support had been widely researched in terms of its effect on mortality. A meta-analytic review estimated that being in social relationships generally provided a 50% increase in odds of survival (Holt-Lunstad et al., 2010). Communication with parents, particularly one's father, was deemed a significant predictor of mortality risks in adolescents. Our results were congruent with other studies which showed that poorer relationships with parents in adolescence were associated with worse physical health (Due et al., 2003) and that relationship with a father largely mediated the association between SES and adolescent health (Moor et al., 2014). An extensive review found that adolescents with absentee fathers had more emotional and behavioural problems, risky health behaviours, and poor academic achievement than adolescents with involved fathers (East et al., 2006). We speculate that these outcomes associated with lack of paternal social support adversely impact health beyond adolescence. Interestingly, social support from friends did not influence one's risk of mortality in our study. We argue

that peer effects are probably more salient for health outcomes during adolescence than those occurring later in the life-course.

We observed slight gender differences in the relationship of reserve capacity with mortality. Whereas, all dimensions significantly and independently increased the boys' risk of death, health-promoting behaviour did not predict mortality risks in girls. Results from other studies yielded inconclusive patterns on gender variations for adolescent reserve capacity. Differences were found in the levels of engagement coping (Finkelstein et al., 2007), and most aspects of social relations (Due et al., 2003) between boys and girls. On the other hand, optimism (Finkelstein et al., 2007; Räikkönen & Matthews, 2008), and social connectedness (Olsson et al., 2013) in adolescence were similar between boys and girls. More research is needed to determine if reserve capacity in adolescence influence health differently in boys and girls.

We extended the theoretical underpinning of the reserve capacity framework by showing that all dimensions also influenced one's education. Constructing a latent reserve capacity construct for all the variables fitted our data well and showed a direct pathway to education. No distinct gender differences in latent reserve capacity were observed, though.

Generally, those with good perceived health, health-promoting behaviours and social support from family had higher likelihood of attaining middle or high education than those with weak reserve capacity. Our results were consistent with those of previous studies, although different psychosocial resources were associated with educational success, namely, locus of control (Murasko, 2007); academic self-efficacy (Merritt & Buboltz, 2015); optimism and life satisfaction (Boehm et al., 2015). It was hypothesised that individuals with a good reserve capacity had increased coping skills necessary for the attainment of higher degrees compared to those with a weak one (Matthews et al., 2010; Murasko, 2007). It was also implicated that reserve capacity in adolescence was related to cognitive development (Kroenke, 2008); thus, it may have logically predicted education. For instance, social connectedness and academic achievement in adolescence were more strongly related to each other in adolescence than in childhood (Olsson et al., 2013). Our results supported such evidence as we found a similar relationship between latent reserve capacity and school achievement in adolescence.

Although we did not find association of friends' social support on education, studies have indicated peer effects on education (Brekke, 2015; Winston &

Zimmerman, 2004). Brekke (2015) showed that relationship with friends decreased the odds of enroling in higher education. Such must be the case when peers had low expectations which resulted in lower university completion rates than those with high peer expectations (OECD, 2018). Indeed, friends were deemed to influence one's approaches to learning and achievement motivation (Nelson & DeBacker, 2008). Thus, having friends who excel academically, improved one's own school performance as opposed to having academically weak peers who pull down their peers' performance (Winston & Zimmerman, 2004).

We have demonstrated that low SES increased the probability of weak reserve capacity, consistent with the framework specifications that low SES deplete psychosocial resources due to cumulative stress (Gallo & Matthews, 2003; Matthews & Gallo, 2011). Indirectly, low SES influenced education through reserve capacity. Our results have also shown that a good reserve capacity mediated the associations of family SES with education, suggesting its potential to reduce socioeconomic inequalities in education. Indeed, in low SES families, significant psychosocial resources such as family support and academic success expectations improved development outcomes among the youth (Kroenke, 2008).

Unlike in health trajectories, though, the exact mechanisms through which reserve capacity operate to influence education have largely been unexplored. Thus, further research is needed to explain how reserve capacity directly affects one's education.

7.3.2 Role of school achievement

We have determined that low school achievement in adolescence was a strong, independent predictor of mortality. Moreover, its effect on mortality did not depend on the level of family SES as there was no statistical interaction between these two exposures. These results supported the findings of Martin and Kubzansky (2005), where increased risks of mortality with lower cognitive performance levels, independent of childhood SES, were seen among Americans. Other studies have shown links of cognitive achievement with health (Murasko, 2007; Lê-Scherban, Diez Roux, Li, & Morgenstern, 2014). For instance, cognitive ability in early adolescence predicted the probability of adult health status (Murasko, 2007). Lê-Scherban and colleagues (2014) also found consistent inverse associations of academic achievement from childhood to adolescence with later health using

different outcomes such as self-reported health status, body mass index (BMI) and psychological distress.

During adolescence, bidirectional links between health behaviours and school achievement were observed (Koivusilta et al., 2013), suggesting that school achievement operated through a behavioural pathway in influencing health and eventually, mortality. Likewise, academic achievement in adolescence had a significantly direct, though weak, pathway to adult well-being (Olsson et al., 2013). This indicates probable links to mortality via a psychosocial pathway.

Since school achievement also acted as a mediating factor between family SES and mortality in boys, its role in reducing mortality risks should be emphasised, especially in socioeconomically disadvantaged adolescents. Researchers have recognized that improving school achievement leads to good health because it is linked to good education. Consequently, this provides opportunities for adolescents to access material and psychosocial resources which protect their health (Freudenberg & Ruglis, 2007; Viner et al., 2012). Thus, an examination of other mechanisms by which adolescent school achievement affects mortality would enhance existing literature on adolescent health and development. It will also provide rich information, especially useful to health professionals and educators.

Our findings that school achievement was a strong predictor of and a significantly direct pathway to education, were congruent with previous evidence (Fergusson et al., 2008; Murasko, 2007; OECD, 2018; Slominski et al., 2011). These imply that adolescents who reported poorer academic achievement attained lower education compared to those with higher achievement in school. More importantly, school achievement is one important mechanism in adolescence that could set educational trajectories. We believe that school achievement, although measured differently in studies (e.g., indicated by academic grades, cognitive test scores or other school performance measures), reflects one's cognitive abilities and skills required for studying and learning. There may also be underlying psychosocial resources for school achievement as we found covariances between reserve capacity and school achievement. A large international assessment of students showed that high performing students had good social and emotional well-being (OECD, 2018). This further corroborated our findings.

We rationalise that these differences in the cognitive and psychosocial skill sets of adolescents, dependent on school achievement, affect other school-related determinants of education. For instance, previous school performance was proposed

to form educational motivations and expectations of academic success. This consequently influenced decisions to continue higher education (Becker & Hecken, 2009). Indeed, Brekke (2015) showed that those with good grades in secondary school and high educational expectations had higher odds of enroling in tertiary education than those with opposite characteristics.

In our study, school achievement was also an indirect pathway from family SES to education, similar to the results obtained in a longitudinal study in New Zealand (Fergusson et al., 2008). Moreover, it reduced the associations of family SES with education, suggesting that school achievement could buffer against adverse educational effects of family SES. Indeed, in a global assessment of adolescent students, those who were socioeconomically disadvantaged but high performers in school, tended to continue into higher education and gain skilled employment in later life (OECD, 2018).

7.3.3 Role of pubertal timing

In assessing socioeconomic inequalities in educational attainment, we found that delayed puberty increased the probability of low to middle education in both boys and girls, along with weak reserve capacities. Once we accounted for adolescent school achievement, however, puberty ceased to be a significant biological pathway leading to education. Similarly, Koerselman and Pekkarinen (2017) using British longitudinal data, found that the associations between late maturation and lower educational attainment in boys and girls were attenuated once they accounted for cognitive achievement in adolescence. In contrast, Gill and colleagues (2017) showed a small but statistically significant effect of later maturation in girls on longer time spent in education. Other studies have also demonstrated that at least in girls, instead of late maturation, early maturation was a risk factor for poor educational outcomes (Cavanagh et al., 2007; Copeland et al., 2010; Hendrick, Cohen, Deardorff, & Cance, 2016). Puberty was hypothesised to influence brain and cognitive development via hormonal changes (Sawyer et al., 2012). A novel study which had examined testosterone and estradiol hormones demonstrated that puberty sequentially influenced academic achievement by shaping academic motivation (Martin & Steinbeck, 2017). We also found that pubertal timing was significantly related to school achievement in boys. This implied that pubertal timing effects on education were probably mediated by a cognitive pathway. Indeed, previous studies have

shown links between puberty and school performance measures in adolescence such as grades and test scores (Cavanagh et al., 2007; Koerselman & Pekkarinen, 2017; Koivusilta & Rimpelä, 2004).

In addition, we found that puberty was related to reserve capacity in both boys and girls, as described in previous studies (Short & Rosenthal, 2008; Zhu & Chan 2017). Since we only measured interrelations, without consideration for any causal relationship between them, we can only infer that pubertal and psychosocial pathways in adolescence are connected. In our study, those with delayed puberty seemed to have better reserve capacity. This converged with the results of Martin and Steinbeck (2017) which showed decreased self-efficacy and lower valuing of school with advanced maturation in young adolescence. Thus, puberty may have exerted educational effects through a non-cognitive pathway such as reserve capacity.

Notably, low SES increased the probability of delayed pubertal timing among boys in our study. Socioeconomic inequalities in timing of puberty have been likewise documented, although evidence is mixed. There were studies which supported our results (de Muinich and Mul, 2001; Parent et al. 2003); studies which found that low SES accelerated, and did not delay puberty (Downing & Bellis 2009; Sun et al. 2017); and others which did not find any association at all (Xu, Norton, & Rahman, 2018). The inconsistent effects of SES on pubertal timing probably reflected inherent differences in population characteristics, including gender, ethnicity, genetic predisposition and health exposures (Parent et al., 2003), as well as methodological variations in studies dealing with puberty (Xu et al., 2018).

There is still limited and inconclusive evidence on the educational effects of puberty, particularly since boys' puberty is relatively understudied. Generally, results linking puberty with education, suggest that off-timing puberty, whether early or late, has possible long-term consequences in education, either through cognitive or psychosocial development in adolescence. Hence, research efforts in the future should consider the multidimensionality of puberty and its contexts to gain better understanding of how this experience shapes adolescent transitions from childhood to adulthood.

7.4 Strengths and limitations

Major strengths of our study included its large sample size, high response rate, long follow-up period and linkage with reliable, register-based, multigenerational data. The longitudinal design and life-course approach also enabled us to assess functional relationships among the variables using powerful, multivariate statistical techniques. Thus, our results lend strong evidence on the inequalities in health and socioeconomic trajectories of adolescents. The robustness and reliability of our results were also evident since we found multiple measures of family SES and reserve capacity consistently associated with education. We have also avoided possible selection bias by including data from those with unknown grandparents in the analyses. Moreover, we have obtained stable results even when we expanded previous works (Koivusilta & Rimpelä, 2004; Koivusilta et al., 2013; Mattila et al., 2008).

Although we needed to use proxy indicators since reserve capacity was conceptualised much later than when our data were collected, we measured a valid, underlying construct based on a good-fitting model of the latent variable. Our operationalisation of reserve capacity further elucidated other resources with psychosocial effects on health and educational trajectories. This merits inclusion in future reserve capacity studies.

We have identified some limitations of our study. Firstly, a small number of deaths was recorded which restricted the analysis on causes of deaths. Secondly, age at spermarche or first ejaculation may not be a sensitive measure of pubertal onset in boys due to a high number of false negative results (Euling et al., 2008). On the other hand, using ideal measures such as Tanner staging and assessment of other maturation characteristics was not feasible as our data were collected by mailed questionnaires. Lastly, other individual and structural or contextual factors which were known determinants of health and education in adolescents, e.g., adolescent psychopathology, educational aspirations and factors related to school and peers, were inherent limitations and not measurable in our study. This is primarily because our surveys were not school-based nor community-based. These may have contributed unobserved individual heterogeneity or frailty that could have biased the statistical inferences made in our study. Although the biases were probably small as we restricted our Type I errors from one to five percent in all three sub-studies and ensured best-fitting models, it is recommended that approaches for bias analysis such

as Monte Carlo sensitivity analysis or probabilistic bias analysis as well as estimation of marginal effects (Arah, 2017) be done for future research.

7.5 Future implications

Understanding how adolescent mechanisms relate with each other and contribute to socioeconomic inequalities in health and educational outcomes provides implications for interventions. These interventions should enable healthy transitions to adulthood, despite risk exposures in early life from disadvantaged social origins. In this light, future studies could design experimental or interventional approaches targeting reserve capacity and school achievement and tracking for health and educational outcomes among adolescents. When possible, it is emphasised that analyses be disaggregated by age, sex, and race or ethnicity.

Since we have demonstrated gradient effects of SES and the adolescent pathways on education, future research should try to assess other related factors influencing preference for vocational instead of university education. Risky health behaviours as well as school factors relating to teachers and peers could also be included to exhaust all possible avenues through which socioeconomic inequalities in education occur. Social immobility of disadvantaged groups, downward mobility, and other parental and grandparental characteristics are additional factors that may be explored within the pathways identified, given the availability of multigenerational data.

Further testing of the reserve capacity dimensions we have studied and their mediating roles in health and social mobility should also be conducted. In the long term, researchers could aim to develop a standardised tool for measuring reserve capacity, which is culturally sensitive and tailored to specific age groups. For a better understanding of the progression of socioeconomic inequalities in health, we also recommend including a mix of morbidity and mortality outcomes, possibly measured at several time points.

Acknowledging these links and studying the impacts of adolescent pathways, hopefully, point to new ways of supporting adolescents in maximising their health and learning potential in life. Health professionals, educators, program planners and policymakers could utilise the evidence to prepare more effective and targeted interventions, both in Finland and abroad, to justify prioritisation of adolescent

health and development programs. Ultimately, societies could achieve better health outcomes and improve socioeconomic status in posterity.

8 CONCLUSIONS

Our study has determined that although socioeconomic inequalities in health and education existed, there are intermediate pathways in adolescence which could buffer the adverse effects of low SES, because of their independent effects on mortality and education. Moreover, these pathways were interrelated in adolescence suggesting that biological, psychosocial and cognitive pathways operated together during important school and life transitions to influence educational outcomes. In addition, intergenerational transmission of SES from grandparents to grandchildren occurred, implying that social origins and possibly, family processes of multiple generations contributed to educational inequalities. Our findings emphasise the roles of reserve capacity and school achievement during adolescence as likely causal or mediating pathways to socioeconomic inequalities in mortality and education.

To address these inequalities, the obvious solution is to remove socioeconomic differences and provide populations with equitable access to resources represented by socioeconomic status to improve their health and education. Changing the social structures and systems which create these differences, however, is a lengthy process that may not be sustainable. It requires good governance, costly investments, multisectoral cooperation, and collective efficacy or social cohesion among families and communities. Results of our study showed that these inequalities could be reduced by targeting individual pathways which could protect against the effects of early socioeconomic disadvantages and improve future health and socioeconomic trajectories. Supporting multigenerational families and schools in building reserve capacities and improving school performance of adolescents, especially among those from low SES origins, could increase educational attainment and avert premature mortality.

9 REFERENCES

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health. The challenge of the gradient. *The American Psychologist*, 49(1), 15-24.

Adler, N. E., & Newman, K. (2002). Socioeconomic disparities in health: Pathways and policies. *Health Affairs* (Project Hope), 21(2), 60-76.

Albertini, M., & Radl, J. (2012). Intergenerational transfers and social class: Inter-vivos transfers as means of status reproduction?*. *Acta Sociologica*, 55(2), 107-123. doi:10.1177/0001699311431596

Aksglaede, L., Olsen, L. W., Sørensen, T. I. A., & Juul, A. (2008). Forty years trends in timing of pubertal growth spurt in 157,000 Danish school children. *Plos One*, 3(7), e2728.

Arah, O. A. (2017). Bias analysis for uncontrolled confounding in the health sciences. *Annual Review of Public Health*, 38, 23-38. doi:10.1146/annurev-publhealth-032315-021644 [doi]

Astone, N. M., & McLanahan, S. S. (1991). Family structure, parental practices and high school completion. *American Sociological Review*, 56(3), 309–320. doi:10.2307/2096106.

Aufseeser, D., Jekielek, S., & Brown, B. (2006). *The family environment and adolescent well-being: Exposure to positive and negative family influences*. Washington, D.C.: Child Trends; and San Francisco, CA: National Adolescent Health Information Center, University of California, San Francisco.

Babyak, M. A. (2009). Understanding confounding and mediation. *Evidence Based Mental Health*, 12(3), 68-71. doi:10.1136/ebmh.12.3.68

Bahr, S. J., & Hoffmann, J. P. (2010). Parenting style, religiosity, peers, and adolescent heavy drinking. *Journal of Studies on Alcohol and Drugs*, 71(4), 539-543.

Bammann, K., Gwozdz, W., Pischke, C., Eiben, G., Fernandez-Alvira, J., De Henauw, S., . . . Pigeot, I. (2016). The impact of familial, behavioural and psychosocial factors on the SES gradient for childhood overweight in europe. A longitudinal study. *International Journal of Obesity*, 41, 54.

Becker, G. S., & Tomes, N. (1979). An equilibrium theory of the distribution of income and intergenerational mobility. *Journal of Political Economy*, 87(6), 1153-1189.

Becker, R., & Hecken, A. E. (2009). Higher education or vocational training? An empirical test of the rational action model of educational choices suggested by Breen and Goldthorpe and Esser. *Acta Sociologica*, 52(1), 25-45.

Bellis, M. A., Downing, J., & Ashton, J. A. (2006). Adults at 12? trends in puberty and their public health consequences. *J Epidemiol Community Health*, 60, 910-911.

Bengtson, V. L. (2001). Beyond the nuclear family: The increasing importance of multigenerational bonds. *Journal of Marriage and Family*, 63(1), 1-16. doi:10.1111/j.1741-3737.2001.00001.x

Bird, K. (2007). The intergenerational transmission of poverty: An overview. (Working Paper No. 99). UK: Chronic Poverty Research Centre. Retrieved from http://ssrn.com/abstract=1629262 or http://dx.doi.org/10.2139/ssrn.162926

Black, M. M., & Surkan, P. J. (2015). Child development and maternal wellbeing: Family perspectives for low-income and middle-income countries. *The Lancet Global Health*, 3(8), e426-e427. doi:10.1016/S2214-109X(15)00084-4

Bleil, M. E., Adler, N. E., Appelhans, B. M., Gregorich, S. E., Sternfeld, B., & Cedars, M. I. (2013). Childhood adversity and pubertal timing: Understanding the origins of adulthood cardiovascular risk. *Biological Psychology*, 93(1), 213-219. doi:10.1016/j.biopsycho.2013.02.005

Boehm, J. K., Chen, Y., Williams, D. R., Ryff, C., & Kubzansky, L. D. (2015). Unequally distributed psychological assets: are there social disparities in optimism, life satisfaction, and positive affect?. *PloS one*, 10(2), e0118066. https://doi.org/10.1371/journal.pone.0118066

Bosma, H., Schrijvers, C., & Mackenbach, J. P. (1999). Socioeconomic inequalities in mortality and importance of perceived control: Cohort study. *BMJ : British Medical Journal*, 319(7223), 1469-1470.

Bozdogan, H. (1987). Model selection and Akaike's information criterion (AIC): The general theory and its analytical extensions. *Psychometrika*, 52(3), 345-370. doi:10.1007/BF02294361

Brekke, I. (2015). Health and educational success in adolescents: A longitudinal study. *BMC Public Health*, 15, 619. doi:10.1186/s12889-015-1966-0

Bricard, D., Jusot, F., & Tubeuf, S. (2010). Lifestyles: A channel of intergenerational transmission of health inequalities? (Issues in Health Economics No. 154). Paris: Institute for Research and Information in Health Economics. Retrieved from http://www.irdes.fr/EspaceAnglais/Publications/IrdesPublications/QES154.pdf

Broer, M., Bai, Y., & Fonseca, F. (2019). Socioeconomic inequality and educational outcomes: An introduction. In M. Broer, Y. Bai & F. Fonseca (Eds.), *Socioeconomic inequality and educational outcomes: Evidence from twenty years of TIMSS* (pp. 1-6). Cham: Springer International Publishing. doi:10.1007/978-3-030-11991-1_1 Retrieved from https://doi.org/10.1007/978-3-030-11991-1_1

Bronfenbrenner, U. (1986). Ecology of the family as a context for human development: research perspectives. *Developmental Psychology*, 22(6), 723-742. doi:10.1037/0012-1649.22.6.723

Carlson, M. J., & Corcoran, M. E. (2001). Family structure and children's behavioral and cognitive outcomes. *Journal of Marriage and Family*, 63(3), 779-792. doi:10.1111/j.1741-3737.2001.00779.x

Cavanagh, S. E., Riegle-Crumb, C., & Crosnoe, R. (2007). Puberty and the education of girls*. *Social Psychology Quarterly*, 70(2), 186-198.

Chan, T. W., & Boliver, V. (2013). The grandparents effect in social mobility: Evidence from British birth cohort studies. *American Sociological Review*, 78(4), 662-678. doi:10.1177/0003122413489130

Chan, T. W., & Boliver, V. (2014). Social mobility over three generations in Finland: A critique. *European Sociological Review*, 30(1), 13-17. doi:10.1093/esr/jct012

Chauvel, L., & Leist, A. K. (2015). Socioeconomic hierarchy and health gradient in Europe: The role of income inequality and of social origins. *International Journal for Equity in Health*, 14(1), 132. doi:10.1186/s12939-015-0263-y

Chen, E. (2007). Impact of socioeconomic status on physiological health in adolescents: An experimental manipulation of psychosocial factors. *Psychosomatic Medicine*, 69(4), 348-355. doi:PSY.0b013e3180592b20 [pii]

Chen, E., Fisher, E. B., Bacharier, L. B., & Strunk, R. C. (2003). Socioeconomic status, stress, and immune markers in adolescents with asthma. *Psychosomatic Medicine*, 65(6), 984-992.

Chen, E., Martin, A. D., & Matthews, K. A. (2006). Socioeconomic status and health: Do gradients differ within childhood and adolescence? *Social Science &*

Medicine, 62(9), 2161-2170. doi:https://doi.org/10.1016/j.socscimed.2005.08.054

Chen, E., & Miller, G. E. (2012). "Shift-and-persist" strategies: Why low socioeconomic status isn't always bad for health. *Perspectives on Psychological Science : A Journal of the Association for Psychological Science*, 7(2), 135-158. doi:10.1177/1745691612436694 [doi]

Chen, E., & Miller, G. E. (2013). Socioeconomic status and health: Mediating and moderating factors. *Annual Review of Clinical Psychology*, 9, 723-749. doi:10.1146/annurev-clinpsy-050212-185634 [doi]

Cheng, T. L., Goodman, E., & The Committee on Pediatric Research (2015). Race, ethnicity, and socioeconomic status in research on child health. *Pediatrics*, 135(1), e225-e237. doi:10.1542/peds.2014-3109

Coall, D. A., & Hertwig, R. (2010). Grandparental investment: Past, present, and future. *The Behavioral and Brain Sciences*, 33(1), 1-40. doi:10.1017/S0140525X09991105 [doi]

Conger, R. D., Conger, K. J., & Martin, M. J. (2010). Socioeconomic status, family processes,and individual development. *Journal of Marriage and Family*, 72(3), 685-704. doi:10.1111/j.1741-3737.2010.00725.x

Conner, M., McEachan, R., Jackson, C., McMillan, B., Woolridge, M., & Lawton, R. (2013). Moderating effect of socioeconomic status on the relationship between health cognitions and behaviors. *Annals of Behavioral Medicine*, 46(1), 19-30. doi:10.1007/s12160-013-9481-y

Copeland, W., Shanahan, L., Miller, S., Costello, E. J., Angold, A., & Maughan, B. (2010). Outcomes of early pubertal timing in young women: A prospective population-based study. *Am J Psychiatry*, 167(10), 1218-1225. doi:10.1176/appi.ajp.2010.09081190

de Muinich Keizer, S. M., & Mul, D. (2001). Trends in pubertal development in Europe. *Human Reproduction Update*, 7(3), 287-291.

de Oliveira, C., Watt, R., & Hamer, M. (2010). Toothbrushing, inflammation, and risk of cardiovascular disease: Results from scottish health survey. *BMJ (Clinical Research Ed.)*, 340, c2451; c2451-c2451. doi:10.1136/bmj.c2451

Downing, J., & Bellis, M. (2009). Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: A preliminary cross-sectional study. *BMC Public Health*, 9(1), 446.

Due, P., Lynch, J., Holstein, B., & Modvig, J. (2003). Socioeconomic health inequalities among a nationally representative sample of Danish adolescents: The role of different types of social relations. *Journal of Epidemiology and Community Health*, 57(9), 692-698.

East, L., Jackson, D., & O'Brien, L. (2006). Father absence and adolescent development: A review of the literature. *Journal of Child Health Care*, 10(4), 283-295. doi:10.1177/1367493506067869

El-Amin, S. E., Kinnunen, J. M., Ollila, H., Helminen, M., Alves, J., Lindfors, P., & Rimpela, A. H. (2015). Transmission of smoking across three generations in Finland. *International Journal of Environmental Research and Public Health*, 13(1), 74. doi:10.3390/ijerph13010074 [doi]

Erola, J., & Kilpi-Jakonen, E. (2017). *Compensation and other forms of accumulation in intergenerational social inequality. Social inequality across the generations* (pp. 3-24) Edward Elgar Publishing. doi:10.4337/9781786432568.00006 Retrieved from https://www.elgaronline.com/view/9781786432551.00006.xml

Erola, J., & Moisio, P. (2007). Social mobility over three generations in Finland, 1950–2000. *European Sociological Review*, 23(2), 169-183. doi:10.1093/esr/jcl027

Euling, S. Y., Herman-Giddens, M. E., Lee, P. A., Selevan, S. G., Juul, A., Sorensen, T. I., . . . Swan, S. H. (2008). Examination of US puberty-timing data from 1940 to 1994 for secular trends: Panel findings. *Pediatrics*, 121 Suppl 3, S172-91. doi:10.1542/peds.2007-1813D [doi]

Evans, G. W., Kim, P., Ting, A. H., Tesher, H. B., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology*, 43(2), 341-351. doi:2007-02739-006 [pii]

Evans, G. W., & Kim, P. (2007). Childhood poverty and health: Cumulative risk exposure and stress dysregulation. *Psychol Sci*, 18(11), 953-957. doi:10.1111/j.1467-9280.2007.02008.x

Feltz, D., & Magyar, M. (2006). Self-efficacy and adolescents in sport and physical activity. In F. Pajares, & T. Urdan (Eds.), *Self-efficacy beliefs of adolescents* (pp. 161-180). Greenwich, Connecticut: Information Age Publishing.

Fergusson, D. M., Horwood, L. J., & Boden, J. M. (2008). The transmission of social inequality: Examination of the linkages between family socioeconomic status in childhood and educational achievement in young adulthood. *Research in Social Stratification and Mobility*, 26(3), 277-295. doi:http://dx.doi.org/10.1016/j.rssm.2008.05.001

Finkelstein, D. M., Kubzansky, L. D., Capitman, J., & Goodman, E. (2007). Socioeconomic differences in adolescent stress: The role of psychological resources. *The Journal of Adolescent Health: Official Publication of the Society for Adolescent Medicine*, 40(2), 127-134. doi:10.1016/j.jadohealth.2006.10.006

Foverskov, E., & Holm, A. (2016). Socioeconomic inequality in health in the British household panel: Tests of the social causation, health selection and the indirect selection hypothesis using dynamic fixed effects panel models. *Social Science & Medicine, 150, 172-183*. doi://doi.org/10.1016/j.socscimed.2015.12.021

Freudenberg, N., & Ruglis, J. (2007). Reframing school dropout as a public health issue. *Preventing Chronic Disease*, 4(4)

Gallo, L. C. (2009). The reserve capacity model as a framework for understanding psychosocial factors in health disparities. *Applied Psychology: Health and Well-being*, 1(1), 62-72. doi:10.1111/j.1758-0854.2008.01000.x

Gallo, L. C., Espinosa de los Monteros, K. & Shivpuri, S. (2009). Socioeconomic status and health: What is the role of reserve capacity? *Current Directions in Psychological Science*, 18(5), 269-274.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin*, 129(1), 10-51.

George, M. P. (2013). The Mexican American health paradox: The collective influence of sociocultural factors on Hispanic health outcomes. *Discussions*, 9(2)

Geurts, T., van Tilburg, T., Poortman, A., & Dykstra, P. (2014). Child care by grandparents: Changes between 1992 and 2006. *Ageing and Society*, doi:10.1017/S0144686X14000270

Gill, D., Del, G. M., Rawson, T. M., Sivakumaran, P., Brown, A., Sheehan, N. A., & Minelli, C. (2017). Age at menarche and time spent in education: A Mendelian randomization study. *Behavior Genetics*, 47(5), 480-485. doi:10.1007/s10519-017-9862-2

Glanz, K., Rimer, B. K., & Viswanath, K. (2008). *Health behavior and health education: Theory, research, and practice* (4th ed.). San Francisco, CA: Jossey-Bass. Retrieved from http://www.loc.gov/catdir/toc/ecip0818/2008021038.html

Golub, M. S., Collman, G. W., Foster, P. M. D., Kimmel, C. A., Rajpert-De Meyts, E., Reiter, E. O., . . . Toppari, J. (2008). Public health implications of altered

puberty timing. *Pediatrics*, 121(Supplement 3), S218-S230. doi:10.1542/peds.2007-1813G

Goodman, E. (1999). The role of socioeconomic status gradients in explaining differences in US adolescents' health. *American Journal of Public Health*, 89(10), 1522-1528.

Graber, J. A. (2013). Pubertal timing and the development of psychopathology in adolescence and beyond. *Hormones and Behavior*, 64(2), 262-269. doi:10.1016/j.yhbeh.2013.04.003 [doi]

Grace, J. B., & Bollen, K. A. (2005). Interpreting the results from multiple regression and structural equation models. *Bulletin of the Ecological Society of America*, 86(4), 283-295. doi:10.1890/0012-9623(2005)86[283:ITRFMR]2.0.CO;2

Green, M. A. (2013). The equalisation hypothesis and changes in geographical inequalities of age based mortality in England, 2002–2004 to 2008–2010. *Social Science & Medicine*, 87, 93-98. doi:https://doi.org/10.1016/j.socscimed.2013.03.029

Hällsten, M., & Pfeffer, F. T. (2017). Grand advantage: Family wealth and grandchildren's educational achievement in sweden. *Am Sociol Rev*, 82(2), 328-360. doi:10.1177/0003122417695791

Hendrick, C. E., Cohen, A. K., Deardorff, J., & Cance, J. D. (2016). Biological and sociocultural factors during the school years predicting women's lifetime educational attainment. *Journal of School Health*, 86(3), 215-224. doi:10.1111/josh.12368

Hendricks, J. (2012). Considering life course concepts. *The Journals of Gerontology: Series B*, 67B(2), 226-231. doi:10.1093/geronb/gbr147

Hoagwood, K. E., Rotheram-Borus, M. J., McCabe, M. A., Counts, N., Belcher, H. M. E., Walker, D. K., & Johnson, K. A. (2018). *The interdependence of families, communities, and children's health: Public investments that strengthen families and communities, and promote children's healthy development and societal prosperity*. (NAM Perspectives. Discussion Paper.). Washington, DC.: National Academy of Medicine. doi:10.31478/201804a

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7(7), e1000316. doi:10.1371/journal.pmed.1000316

Hooper, D., Coughlan, J., & Mullen, M.,R. (2007). Structural equation modeling: Guidelines for determining model fit. *The Electronic Journal of Business Research Methods*, 6(1):53-60.

Howarter, A. D., & Bennett, K. K. (2013). Perceived discrimination and health-related quality of life: Testing the reserve capacity model in Hispanic Americans. *The Journal of Social Psychology*, 153(1), 62-79. doi:10.1080/00224545.2012.703973 [doi]

Ip, P., Ho, F. K. W., So, H., Chan, D. F., Ho, M., Tso, W., & Nelson, E. A. (2016). Socioeconomic gradient in childhood obesity and hypertension: A multilevel population-based study in a Chinese community. *PloS One*, 11(6), e0156945; e0156945-e0156945. doi:10.1371/journal.pone.0156945

Jacobsen, B. K., Oda, K., Knutsen, S. F., & Fraser, G. E. (2009). Age at menarche, total mortality and mortality from ischaemic heart disease and stroke: The Adventist health study, 1976-88. *International Journal of Epidemiology*, 38(1), 245-252. doi:10.1093/ije/dyn251 [doi]

James-Todd, T., Tehranifar, P., Rich-Edwards, J., Titievsky, L., & Terry, M. B. (2010). The impact of socioeconomic status across early life on age at menarche among a racially diverse population of girls. *Annals of Epidemiology*, 20(11), 836-842. doi:10.1016/j.annepidem.2010.08.006

Johnson, M. K., Robert, C., & Elder, G. H. (2011). Insights on adolescence from a life course perspective. *Journal of Research on Adolescence*, 21(1), 273-280. doi:10.1111/j.1532-7795.2010.00728.x

Johnston, D. W., Schurer, S., & Shields, M. A. (2013). Exploring the intergenerational persistence of mental health: Evidence from three generations. *Journal of Health Economics*, 32(6), 1077-1089. doi:http://dx.doi.org/10.1016/j.jhealeco.2013.09.001

Juárez, S. P., Goodman, A., & Koupil, I. (2016). From cradle to grave: Tracking socioeconomic inequalities in mortality in a cohort of 11 868 men and women born in Uppsala, Sweden, 1915–1929. *Journal of Epidemiology and Community Health*, 70(6), 569-575. doi:10.1136/jech-2015-206547

Kawachi, I., Subramanian, S. V., & Almeida-Filho, N. (2002). A glossary for health inequalities. *Journal of Epidemiology and Community Health*, 56(9), 647-652. doi:10.1136/jech.56.9.647

Koerselman, K., & Pekkarinen, T. (July 2017). *The timing of puberty and gender differences in educational achievement*. (No. IZA DP No. 10889). Bonn, Germany: IZA

Institute of Labor Economics initiated by Deutsche Post Foundation. doi:http://ftp.iza.org/dp10889.pdf

Koivusilta, L., & Rimpelä, A. (2004). Pubertal timing and educational careers: A longitudinal study. *Annals of Human Biology*, 31(4), 446-465. doi:10.1080/03014460412331281719

Koivusilta, L. K., & Rimpelä, A. H. (2006). Pubertal timing and health-related behaviours in adolescence - socio- economic outcomes in a follow-up study from Finland. *Italian Journal of Public Health*, 3(1), 41-52. doi:http://dx.doi.org/10.2427/5949

Koivusilta, L., Rimpelä A., & Vikat, A. (2003). Health behaviours and health in adolescence as predictors of educational level in adulthood: A follow-up study from Finland. *Social Science & Medicine*, 57(4), 577-593. doi://dx.doi.org/10.1016/S0277-9536(02)00405-7

Koivusilta, L., West, P., Saaristo, V. M. A., Nummi, T., & Rimpelä, A. (2013). From childhood socio-economic position to adult educational level - do health behaviours in adolescence matter? A longitudinal study. *BMC Public Health*, 13(1), 711.

Krieger, N., Chen, J. T., & Waterman, P. D. (2010). Decline in US breast cancer rates after the women's health initiative: Socioeconomic and Racial/Ethnic differentials. *Am J Public Health*, 100, S132-S139. doi:10.2105/AJPH.2009.181628

Kroenke, C. (2008). Socioeconomic status and health: Youth development and neomaterialist and psychosocial mechanisms. *Social Science & Medicine*, 66(1), 31-42. doi://dx.doi.org/10.1016/j.socscimed.2007.07.018

Kuh, D., Ben-Shlomo, Y., Lynch, J., Hallqvist, J., & Power, C. (2003). Life course epidemiology. *Journal of Epidemiology and Community Health*, 57(10), 778-783. doi:10.1136/jech.57.10.778

Kuh, D., Hardy, R., Langenberg, C., Richards, M., & Wadsworth, M. E. (2002). Mortality in adults aged 26-54 years related to socioeconomic conditions in childhood and adulthood: Post war birth cohort study. *BMJ (Clinical Research Ed.)*, 325(7372), 1076-1080.

Lê-Scherban, F., Diez Roux, A. V., Li, Y., & Morgenstern, H. Does academic achievement during childhood and adolescence benefit later health? *Annals of Epidemiology*, 24(5), 344-355. doi:10.1016/j.annepidem.2014.02.008

Lakshman, R., Forouhi, N. G., Sharp, S. J., Luben, R., Bingham, S. A., Khaw, K. T., . . . Ong, K. K. (2009). Early age at menarche associated with cardiovascular disease and mortality. *The Journal of Clinical Endocrinology and Metabolism*, 94(12), 4953-4960. doi:10.1210/jc.2009-1789 [doi]

Little, A. W., & Green, A. (2009). Successful globalisation, education and sustainable development. *International Journal of Educational Development*, 29(2), 166-174. doi:https://doi.org/10.1016/j.ijedudev.2008.09.011

Lopez, N., & Gadsden, V. L. (2016). Health inequities, social determinants, and intersectionality. *NAM Perspectives*, 6(12) doi:10.31478/201612a

Liu, L., Xue, F., Ma, J., Ma, M., Long, Y., & Newschaffer, C. J. (2013). Social position and chronic conditions across the life span and risk of stroke: A life course epidemiological analysis of 22 847 American adults in ages over 50. *International Journal of Stroke*, 8, 50-55. doi:10.1111/j.1747-4949.2012.00927.x

Mackenbach, J. P., Kulhanova, I., Bopp, M., Deboosere, P., Eikemo, T. A., Hoffmann, R., . . . EURO-GBD-SE Consortium. (2015). Variations in the relation between education and cause-specific mortality in 19 European populations: A test of the "fundamental causes" theory of social inequalities in health. *Social Science & Medicine* (1982), 127, 51-62. doi:10.1016/j.socscimed.2014.05.021 [doi]

Mackenbach, J. P., Simon, J. G., Looman, C. W. N., & Joung, I. M. A. (2002). Self-assessed health and mortality: Could psychosocial factors explain the association? *International Journal of Epidemiology*, 31(6), 1162-1168.

Martikainen, P., Bartley, M., & Lahelma, E. (2002). Psychosocial determinants of health in social epidemiology. *International Journal of Epidemiology*, 31(6), 1091-1093. doi:10.1093/ije/31.6.1091

Martin, A. J., & Steinbeck, K. (2017). The role of puberty in students' academic motivation and achievement. *Learning and Individual Differences*, 53(Complete), 37-46. doi:10.1016/j.lindif.2016.11.003

Martin, L. T., & Kubzansky, L. D. (2005). Childhood cognitive performance and risk of mortality: A prospective cohort study of gifted individuals. *American Journal of Epidemiology*, 162(9), 887-890. doi:kwi300 [pii]

Martin, M. J., Conger, R. D., Schofield, T. J., Dogan, S. J., Widaman, K. F., Donnellan, M. B., & Neppl, T. K. (2010). Evaluation of the interactionist model of socioeconomic status and problem behavior: A developmental

cascade across generations. *Development and Psychopathology*, 22(3), 695-713. doi:10.1017/S0954579410000374 [doi]

Matthews, K. A., & Gallo, L. C. (2011). Psychological perspectives on pathways linking socioeconomic status and physical health. *Annual Review of Psychology*, 62, 501-530. doi:10.1146/annurev.psych.031809.130711; 10.1146/annurev.psych.031809.130711

Matthews, K. A., Gallo, L. C., & Taylor, S. E. (2010). Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. *Annals of the New York Academy of Sciences*, 1186, 146-173. doi:10.1111/j.1749-6632.2009.05332.x [doi]

Matthews, K. A., Räikkönen, K., Gallo, L., & Kuller, L. H. (2008). Association between socioeconomic status and metabolic syndrome in women: Testing the reserve capacity model. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, 27(5), 576-583. doi:10.1037/0278-6133.27.5.576

Mattila, V. M., Parkkari, J., Koivusilta, L., Nummi, T., Kannus, P., & Rimpela, A. (2008). Adolescents' health and health behaviour as predictors of injury death. A prospective cohort follow-up of 652,530 person-years. *BMC Public Health*, 8, 90-2458-8-90. doi:10.1186/1471-2458-8-90 [doi]

Merritt, D. L., & Buboltz, W. (2015). Academic success in college: Socioeconomic status and parental influence as predictors of outcome. *Open Journal of Social Sciences*, 3(5), 127-135. doi:10.4236/jss.2015.35018

Modin, B., & Fritzell, J. (2009). The long arm of the family: Are parental and grandparental earnings related to young men's body mass index and cognitive ability?[miscellaneous article]. *International Journal of Epidemiology*, 38(3), 733-744.

Møllegaard, S., & Jæger, M. M. (2015). The effect of grandparents' economic, cultural, and social capital on grandchildren's educational success. *Research in Social Stratification and Mobility*, 42, 11-19. doi://dx.doi.org/10.1016/j.rssm.2015.06.004

Moor, I., Rathmann, K., Stronks, K., Levin, K., Spallek, J., & Richter, M. (2014). Psychosocial and behavioural factors in the explanation of socioeconomic inequalities in adolescent health: A multilevel analysis in 28 European and North American countries. *Journal of Epidemiology and Community Health*, 68(10), 912-921. doi:10.1136/jech-2014-203933 [doi]

Moore, G. F., & Littlecott, H. J. (2015). School- and family-level socioeconomic status and health behaviors: Multilevel analysis of a national survey in Wales, United Kingdom. *Journal of School Health*, 85(4), 267-275. doi:10.1111/josh.12242

Mulder, B. C., de Bruin, M., Schreurs, H., van Ameijden, E.,J.C., & van Woerkum, C.,M.J. (2011). Stressors and resources mediate the association of socioeconomic position with health behaviours. *BMC Public Health*, 11(1), 798. doi:10.1186/1471-2458-11-798

Murasko, J. E. (2007). A lifecourse study on education and health: The relationship between childhood psychosocial resources and outcomes in adolescence and young adulthood. *Social Science Research*, 36(4), 1348-1370. doi:https://doi.org/10.1016/j.ssresearch.2007.01.001

Muthén, L. K., & Muthén, B. O. (1998-2012). *Mplus User's guide* (Seventh Edition ed.). Los Angeles, Ca.: Muthén & Muthén.

Nelson, R. M., & DeBacker, T. K. (2008). Achievement motivation in adolescents: The role of peer climate and best friends. *The Journal of Experimental Education*, 76(2), 170-189.

Obeidallah, D. A., Brennan, R. T., Brooks-Gunn, J., Kindlon, D., & Earls, F. (2000). Socioeconomic status, race, and girls' pubertal maturation: Results from the project on human development in Chicago neighborhoods. *Journal of Research on Adolescence*, 10(4), 443-464. doi:10.1207/SJRA1004_04

OECD (2011), *Lessons from PISA for the United States*, Strong Performers and Successful Reformers in Education, OECD Publishing, Paris, https://doi.org/10.1787/9789264096660-en.

OECD (2018), *Equity in Education: Breaking Down Barriers to Social Mobility*, PISA, OECD Publishing, Paris, https://doi.org/10.1787/9789264073234-en.

Olsson, C. A., McGee, R., Nada-Raja, S., & Williams, S. M. (2013). A 32-year longitudinal study of child and adolescent pathways to well-being in adulthood. *Journal of Happiness Studies*, 14(3), 1069-1083. doi:10.1007/s10902-012-9369-8

Osler, M., Andersen, A. M., Lund, R., & Holstein, B. (2005). Effect of grandparent's and parent's socioeconomic position on mortality among Danish men born in 1953. *European Journal of Public Health*, 15(6), 647-651. doi:10.1093/eurpub/cki066

Padilla-Walker, L. M., & Bean, R. A. (2009). Negative and positive peer influence: Relations to positive and negative behaviors for African American, European American, and Hispanic adolescents. *Journal of Adolescence*, 32(2), 323-337. doi:https://doi.org/10.1016/j.adolescence.2008.02.003

Page, R. M., Simonek, J., Ihász, F., Hantiu, I., Uvacsek, M., Kalabiska, I., & Klarova, R. (2009). Self-rated health, psychosocial functioning, and other dimensions of adolescent health in Central and Eastern European adolescents. *The European Journal of Psychiatry*, 23(2), 101-114.

Pakpour, A. H., & Sniehotta, F. F. (2012). Perceived behavioural control and coping planning predict dental brushing behaviour among Iranian adolescents. *Journal of Clinical Periodontology*, 39(2), 132-137. doi:10.1111/j.1600-051X.2011.01826.x

Parent, A., Teilmann, G., Juul, A., Skakkebaek, N. E., Toppari, J., & Bourguignon, J. (2003). The timing of normal puberty and the age limits of sexual precocity: Variations around the world, secular trends, and changes after migration. *Endocrine Reviews*, 24(5), 668-693.

Perrons, D., & Plomien, A. (2010). *Why socio-economic inequalities increase? Facts and Policy Responses in Europe*. EUR 24471 EN. European Union, Brussels, Belgium. Retrieved from http://dx.publications.europa.eu/10.2777/26112

Phelan, J. C., Link, B. G., & Tehranifar, P. (2010). Social conditions as fundamental causes of health inequalities: Theory, evidence, and policy implications. *Journal of Health and Social Behavior*, 51(1), S28-S40. doi:10.1177/0022146510383498

Phillips, S. P., & Hamberg, K. (2015). Women's relative immunity to the socio-economic health gradient: Artifact or real? *Global Health Action*, 8, 27259. doi:10.3402/gha.v8.27259 [doi]

Piko, B. F., & Fitzpatrick, K. M. (2007). Socioeconomic status, psychosocial health and health behaviours among Hungarian adolescents. *The European Journal of Public Health*, 17(4), 353-360. doi:10.1093/eurpub/ckl257

Räikkönen, K., & Matthews, K. A. (2008). Do dispositional pessimism and optimism predict ambulatory blood pressure during school days and nights in adolescents? *Journal of Personality*, 76(3), 605-630. doi:10.1111/j.1467-6494.2008.00498.x

Remes, H., Martikainen, P., & Valkonen, T. (2010). Mortality inequalities by parental education among children and young adults in Finland 1990-2004. *Journal of Epidemiology and Community Health*, 64(2), 136-141. doi:10.1136/jech.2008.082388 [doi]

Robbins, L. B., Pender, N. J., Ronis, D. L., Kazanis, A. S., & Pis, M. B. (2004). Physical activity, self-efficacy, and perceived exertion among adolescents. *Research in Nursing & Health*, 27(6), 435-446. doi:10.1002/nur.20042 [doi]

Rudolph, K. D., Troop-Gordon, W., Lambert, S. F., & Natsuaki, M. N. (2014). Long-term consequences of pubertal timing for youth depression: Identifying personal and contextual pathways of risk. *Development and Psychopathology*, 26(4 Pt 2), 1423-1444. doi:10.1017/S0954579414001126 [doi]

Ryan, A. M., & Patrick, H. (2001). The classroom social environment and changes in adolescents' motivation and engagement during middle school. *American Educational Research Journal*, 38(2), 437-460. doi:10.3102/00028312038002437

Sanders, R. A. (2013). Adolescent psychosocial, social, and cognitive development. *Pediatrics in Review*, 34(8), 354-359. doi:10.1542/pir.34-8-354

Sawyer, S. M., Afifi, R. A., Bearinger, L. H., Blakemore, S., Dick, B., Ezeh, A. C., & Patton, G. C. (2012). Adolescence: A foundation for future health. *The Lancet*, 379(9826), 1630-1640. doi:10.1016/S0140-6736(12)60072-5

Scheerman, J. F., van Loveren, C., van Meijel, B., Dusseldorp, E., Wartewig, E., Verrips, G. H., . . . van Empelen, P. (2016). Psychosocial correlates of oral hygiene behaviour in people aged 9 to 19 - a systematic review with meta-analysis. *Community Dentistry and Oral Epidemiology*, 44(4), 331-341. doi:10.1111/cdoe.12224 [doi]

Schwarzer, R., & Luszczynska, A. (2006). Self-efficacy, adolescents' risk-taking behaviors, and health. In T. Urdan, & F. Pajares (Eds.), *Self-efficacy beliefs of adolescents* (pp. 139-159). Greenwich, Connecticut: Information Age Publishing.

Short, M. B., & Rosenthal, S. L. (2008). Psychosocial development and puberty. *Annals of the New York Academy of Sciences*, 1135, 36-42. doi:10.1196/annals.1429.011 [doi]

Sjögren E, & Kristenson M. (2006). Can gender differences in psychosocial factors be explained by socioeconomic status. *Scandinavian Journal of Public Health*, 34(1), 59–68.

Slominski, L., Sameroff, A., Rosenblum, K., & Kasser, T. (2011). Longitudinal predictors of adult socioeconomic attainment: The roles of socioeconomic status, academic competence, and mental health. *Development and Psychopathology*, 23(1), 315-324. doi:10.1017/S0954579410000829 [doi]

Spittel, M. L., Riley, W. T., & Kaplan, R. M. (2015). Educational attainment and life expectancy: A perspective from the NIH office of behavioral and social sciences research. *Social Science & Medicine* (1982), 127, 203-205. doi:10.1016/j.socscimed.2014.11.017 [doi]

Statistics Finland. (2018). *Finnish standard classification of education 2011*. Retrieved from http://www.stat.fi/meta/luokitukset/koulutus/001-2011/index_en.html

Stronks, K., Snijder, M. B., Peters, R. J. G., Prins, M., Schene, A. H., & Zwinderman, A. H. (2013). Unravelling the impact of ethnicity on health in Europe: The HELIUS study. *BMC Public Health*, 13, 402-402. doi:10.1186/1471-2458-13-402

Suhonen, T., & Karhunen, H. (2019). The intergenerational effects of parental higher education: Evidence from changes in university accessibility. *Journal of Public Economics*, 176, 195-217 doi:https://doi.org/10.1016/j.jpubeco.2019.07.001

Sun, Y., Mensah, F. K., Azzopardi, P., Patton, G. C., & Wake, M. (2017). Childhood social disadvantage and pubertal timing: A national birth cohort from Australia. *Pediatrics*, 139(6), 10.1542/peds.2016-4099. doi:e20164099 [pii]

Thirutnurthy, V., Kirylo, J. D., & Ciabattari, T. (2010). Issue in education: Cultural capital, social capital, and educational inequality. *Childhood Education*, 87(2), 119-121. doi:10.1080/00094056.2011.10521456

Tomé, G., Matos, M., Simões, C., Diniz, J. A., & Camacho, I. (2012). How can peer group influence the behavior of adolescents: Explanatory model. *Global Journal of Health Science,* 4(2), 26-35. doi:10.5539/gjhs.v4n2p26

Tubeuf S., Jusot, F., & Bricard, D. (2012). Mediating role of education and lifestyles in the relationship between early-life conditions and health: Evidence from the 1958 British cohort. *Health Economics*, 21, 129-150. doi:10.1002/hec.2815

UCLA: Statistical Consulting Group. (2015). *Statistica computing seminars: Survival analysis with Stata.* Retrieved from http://www.ats.ucla.edu/stat/stata/seminars/stata_survival/

UCLA: Statistical Consulting Group. (2019). *Probit regression | STATA annotated output*. Retrieved from https://stats.idre.ucla.edu/stata/output/probit-regression/

United Nations Development Programme. (2019). Sustainable development goals. Retrieved from http://www.undp.org/content/undp/en/home/sustainable-development-goals.html

van Oort, F. V., van Lenthe, F. J., & Mackenbach, J. P. (2005). Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in the Netherlands. *Journal of Epidemiology and Community Health*, 59(3), 214-220. doi:59/3/214 [pii]

van Zon, S.,K.R., Bültmann, U., Mendes de Leon, C.,F., & Reijneveld, S. A. (2015). Absolute and relative socioeconomic health inequalities across age groups. *PloS One*, 10(12), e0145947. doi:10.1371/journal.pone.0145947

Viner, R. M., Ozer, E. M., Denny, S., Marmot, M., Resnick, M., Fatusi, A., & Currie, C. (2012). Adolescence and the social determinants of health. *The Lancet*, 379(9826), 1641-52. doi:https://doi.org/10.1016/S0140-6736(12)60149-4

Wagner, K. D., Ritt-Olson, A., Chou, C., Pokhrel, P., Duan, L., Baezconde-Garbanati, L., . . . Unger, J. B. (2010). Associations between family structure, family functioning, and substance use among Hispanic/Latino adolescents. *Psychology of Addictive Behaviors : Journal of the Society of Psychologists in Addictive Behaviors*, 24(1), 98-108. doi:10.1037/a0018497

Ward, P. J., & Zabriskie, R. B. (2011). Positive youth development within a family leisure context: Youth perspectives of family outcomes. *New Directions for Youth Development*, 2011(130), 29-42. doi:10.1002/yd.395

Weiss, H. E. (2012). The intergenerational transmission of social capital: A developmental approach to adolescent social capital formation*. *Sociological Inquiry*, 82(2), 212-235. doi:10.1111/j.1475-682X.2012.00414.x

West, P. (1997). Health inequalities in the early years: Is there equalisation in youth? *Social Science & Medicine* 44(6), 833-858. doi:https://doi.org/10.1016/S0277-9536(96)00188-8

Westling, E., Andrews, J. A., Hampson, S. E., & Peterson, M. (2008). Pubertal timing and substance use: The effects of gender, parental monitoring and deviant peers. *The Journal of Adolescent Health : Official Publication of the Society for Adolescent Medicine*, 42(6), 555-563. doi:10.1016/j.jadohealth.2007.11.002 [doi]

Winston, G., & Zimmerman, D. (2004). *Peer effects in higher education. College choices: The economics of where to go, when to go, and how to pay for it* (pp. 395-424) University of Chicago Press. Retrieved from http://www.nber.org/chapters/c10105,

Xu, Y., Norton, S., & Rahman, Q. (2018). Early life conditions, reproductive and sexuality-related life history outcomes among human males: A systematic review and meta-analysis. *Evolution and Human Behavior*, doi:10.1016/j.evolhumbehav.2017.08.005

Zhu, J., & Chan, Y. (2017). Adult consequences of self-limited delayed puberty. *Pediatrics*, 139(6). doi:10.1542/peds.2016-3177

PUBLICATION I

Adolescent reserve capacity, socioeconomic status and school achievement as predictors of mortality in Finland - A longitudinal study

Paulyn Jean Acacio-Claro, Leena Kristiina Koivusilta, Judith Rafaelita Borja, and Arja Hannele Rimpelä

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RESEARCH ARTICLE Open Access

Adolescent reserve capacity, socioeconomic status and school achievement as predictors of mortality in Finland - a longitudinal study

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Abstract

Background: Despite robust evidence on the inverse relationship between socioeconomic status (SES) and mortality, deviations from expected results have been observed likely due to school achievement and psychosocial resources, termed as "reserve capacity." Since adolescence is a critical period in developing sound psychological and behavioural patterns and adolescent markers of SES were seldom used, we determine if family SES in adolescence predicts later mortality. We also study how reserve capacity (perceived health, health-promoting behaviour and social support) and school achievement modify this relationship and reduce the negative effects of low SES.

Methods: A longitudinal study was designed by linking baseline data on 12 to 18 year-old Finns in 1985–95 ($N = 41,833$) from the Adolescent Health and Lifestyle Surveys with register data on mortality and SES from Statistics Finland. Average follow-up time was 18.4 years with a total of 770,161 person-years. Cox regression models, stratified by sex, were fitted to determine the effects of variables measured during adolescence: family SES, reserve capacity and school achievement on mortality risk.

Results: All reserve capacity dimensions significantly predicted mortality in boys. Perceived health and social support predicted that in girls. Adolescents with the lowest school achievement were more than twice at risk of dying compared to those with better school performance. Low SES increased the risk of death in boys (Hazard ratios: 1.6, 95% CI 1.1–2.4) but not in girls. Reserve capacity and school achievement weakened the effects of low SES on boys' risk of death.

Conclusions: High reserve capacity and good school achievement in adolescence significantly reduce the risk of mortality. In boys, these also mitigate the negative effect of low SES on mortality. These findings underscore the roles of reserve capacity and school achievement during adolescence as likely causal or modifying factors in SES-health inequalities.

Keywords: Mortality, Socioeconomic status, Psychosocial resources, Reserve capacity, Life course epidemiology

Background

Research has extensively demonstrated the relationship between health and socioeconomic status (SES), often measured through income, education or occupation. Many studies have proven that low SES has adverse effects on health, acting cumulatively on morbidity and mortality [1–6]. A number of studies found high risks of premature death in both men and women with limited education, manual occupations and poor housing conditions [3, 5]. Also, regardless of adult socioeconomic status, poor socioeconomic conditions in early life were confirmed to be associated with mortality later in life [3, 5, 6]. Hence, SES has been proposed as a "fundamental cause" of health inequalities because it represents several resources like money, knowledge, prestige, power and beneficial social connections which can be used to improve health regardless of the disease mechanisms working at a given time [7]. Thus, even with improvements in medicine and other

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advances in health technologies, those without access to these resources lack the means to protect their health. This theory was empirically proven through a large study with a multi-country setting [8].

Despite the robust evidence on health disadvantages of low SES [1–6, 8], deviations have been observed [9–11]. Developmental studies have shown that early adverse exposures to poor environments could activate adaptive responses or mechanisms that provide long-term health advantages [12]. For example, early microbial exposure has been shown to boost immunity and increase resistance to diseases [13–15]. However, this field still warrants further testing and research.

Another perspective which likely explains said "epidemiological paradox," initially described in distinct racial groups [16] is the psychosocial mechanism. Matthews and Gallo [9] proposed that individuals draw upon a bank of psychosocial resources called "reserve capacity" in response to acute and chronic stressors. Reserve capacity is a multidimensional concept which includes interpersonal resources such as social support and integration and intrapersonal characteristics such as self-efficacy, mastery or a sense of perceived control [9, 17–19]. We further extend the reserve capacity framework to include health behaviour since psychosocial resources underlie these factors and operate through them [20]. For instance, dental brushing behaviour and physical activity have been shown to improve with high self-efficacy [21–23]. Our study focuses on three dimensions: perceived health, health-promoting behaviour and social support.

Low reserve capacities trigger negative emotional and physiological responses and exacerbate the effect of low SES on cardiovascular morbidity and all-cause mortality via biological and behavioural intermediate pathways [9, 18, 19]. High reserve capacities decrease morbidity and mortality risks by regulating stress response, promoting positive emotions and facilitating adaptive coping which dampen pathogenic processes [11, 19]. For instance, some studies have attributed excess cardiovascular disease risk in low SES individuals to perceptions of weak job control [18]. On the other hand, accounting for self-efficacy reduced the risk of cardiovascular disease among those with low SES [17]. Low SES individuals with strong control beliefs and social connectedness had health outcomes similar to those of higher SES individuals [11, 19]. Conversely, increased risk to mortality were seen in those with reduced social resources [24].

There is a complex interplay of processes by which SES affects health throughout one's lifetime. While family conditions determine early life SES and affect health outcomes in adulthood [3, 4], academic achievement in adolescence influences health, as well as current and future SES [25–27]. High achievement is associated with better health status and high SES [2, 25–27]. Decisions regarding school career leading to future adult education are affected by achievements in school [26, 27]. In addition, reserve capacity is shaped during adolescence [28]. Acknowledging these links, our study adopts a life-course approach [9, 29], where exposures during young adolescence are examined for their effects on the health trajectory, more specifically mortality.

Our aim is to study the relationship of family SES with mortality in adolescence and early adulthood. Moreover, we determine whether adolescent reserve capacity and school achievement contribute to mortality risk and modify the relationship between SES and mortality.

Methods

Study design

A longitudinal study was designed linking two data sources by means of unique national personal identification numbers. Baseline data were obtained from the Adolescent Health and Lifestyle Surveys (AHLS) of 1985, 1987, 1991, 1993 and 1995. Nationally representative samples of 12-, 14-, 16-, and 18-year-old Finns born on certain days in June, July and August were drawn each study year from the Population Register Centre. Overall response rate was 79% (N = 41,833), with 72% (N = 19,509) for boys and 86% (N = 22,324) for girls, respectively. A self-administered questionnaire was sent in February, followed by two re-inquiries to non-respondents. The variables used in our study were comparable across all survey rounds.

Follow-up data and information on family SES were respectively obtained from the Finnish Official Cause-of-Death Register and from the Register of Completed Education and Degrees, containing statistics on every resident in Finland. The follow-up started on 30 April, each survey year, and ended 31 December, 2009, or when the participant died. Average follow-up time was 18.4 years. It ranged from 1 to 25 years and had a total of 770,161 person-years. At the end of the follow-up, the participants were aged 27 to 43 years.

Statistics Finland performed the data linkage of the national registries and the AHLS data according to a contract specifying the rights and duties of both parties. The study protocol was approved by its Institutional Review Board and the Data Protection Ombudsman. The Joint Commission on Ethics of the University of Turku and the Turku University Hospital 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. The first review boards at the universities were established in Finland in the 1980s. AHLS was reviewed by the Ethical Review Board of the University of Helsinki, Department of Public Health in 1986. Parental consent was not considered by the ethics review board at that

time. In later surveys, the latest in 2017, the relevant review boards have waived the parental consent.

Outcome and predictor variables

Table 1 shows the distribution of outcome and predictor variables. The outcome variable was death, defined by month and year. The predictor variables described family SES, reserve capacity and school achievement.

Family SES was based on parents' education from Statistics Finland categorized into basic, secondary and high. Data was obtained nearest to the year when the adolescent was aged 15 years. If parents belonged to different categories, the highest was selected. If one parent was missing (2%), the available parent's data was used.

Within each dimension of reserve capacity (survey data), correlations and associations of the variables were calculated. Moderate positive correlations (Spearman's) and statistically significant associations (Pearson chi-square tests) ensured that they measured the same dimension.

a. *Perceived health* included three items: has a chronic disease, injury or disability that restricts daily activities (no/yes); a summary index of weekly perceived stress symptoms (stomachaches, 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 having none, 1 symptom/week, 2–3/week and 4–8/week; and, self-rated health categorized as very good, good to average, poor.
b. *Health-promoting behaviour* 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.
c. *Social support* 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, very difficult). Those who did not have a father (5%), mother (1%) or friends (0.5%) were set to "very difficult."

For school achievement, adolescents were categorized as having: highest, 2nd highest, 2nd lowest or lowest academic achievement. The respondents were asked to assess whether their end-of-term school report was much better, slightly better, average, slightly poorer or much poorer than the class average. For 12–14-year-olds (all in comprehensive schools), the last two were combined. For 16–18-year-olds, the first two were further combined and school status (high school/vocational school/not attending school) was additionally used. Respective categories included: high school, better than class average; vocational school, better or high school, average; vocational school, poor to average or high school, poor; and, not at school.

Statistical analysis

Cox proportional hazards models, stratified by sex, were fitted to determine the relationship of predictor variables with mortality and calculate hazard ratios (HR) with 95% confidence intervals (CIs). Adherence to the proportional hazards assumption was checked using log-log survival curves and a formal significance test based on the unscaled and scaled Schoenfeld residuals [30]. First, a crude model, which considered family SES, each reserve capacity dimension and school achievement, was fitted to analyse each predictor's unadjusted effect on mortality risk (Model 1). Then, to study whether the reserve capacity variables modified the relationship between SES and mortality, all statistically significant ($p < 0.05$) reserve capacity variables together with SES were included in a backward selection procedure until none could be deleted from the model (Model 2). Finally, school achievement was added (Model 3). An interaction term between family SES and school achievement was also tested. Model fit was assessed using likelihood ratio tests and Akaike Information Criteria (AIC) [31]. Post-estimation tests were done (checking of residuals and other plots) to ensure that the final model had the best fit. Respondents with missing data (5%) in one or more main variables studied were dropped from analysis. All analyses were performed using STATA version 12.1.

Results

Characteristics of the study population

Table 1 lists the detailed characteristics of the total population according to family SES, dimensions of reserve capacity, school achievement and outcome status. Less than one-fourth of boys and girls had low family SES. Generally, most adolescents had positive reserve capacity characteristics but boys and girls differed in terms of perceived stress symptoms and tooth brushing frequency, which were more common among the girls, and ease of talking about issues to father, which was more common among the boys. High achievement in school was also more common among the girls compared to boys.

Among boys, with 358,787 person-years of follow-up time (mean 18.4 years), mortality rate was 10.1 per 10,000 population. In contrast, mortality rate among the

Table 1 Distribution of participants according to age at baseline, predictor variables and outcome status, Finland

Age at baseline and predictor variables in adolescence	Total population ($n = 41,833$)				Number of Deaths ($n = 499$)			
	Boys ($n = 19,509$)		Girls ($n = 22,324$)		Boys ($n = 362$)		Girls ($n = 137$)	
	No.	%	No.	%	No.	%	No.	%
Age at baseline (years)								
12	1976	10.1	1976	8.9	39	10.8	12	8.8
14	6011	30.8	6575	29.4	97	26.8	34	24.8
16	6285	32.2	7300	32.7	135	37.3	43	31.4
18	5237	26.9	6473	29.0	91	25.1	48	35.0
Family SES (parents' education)								
Higher education	3261	16.7	3573	16.0	35	9.7	19	13.9
Secondary education	11,818	60.6	13,530	60.6	211	58.3	77	56.2
Basic or lower	4425	22.7	5210	23.3	116	32.0	41	29.9
No data	5	0.0	11	0.1	0	0.0	0	0.0
Perceived health								
Chronic disease								
No	17,791	91.2	20,134	90.2	312	86.2	119	86.9
Yes	1718	8.8	2190	9.8	50	13.8	18	13.1
Perceived stress symptoms								
None	9897	50.7	7144	32.0	156	43.1	35	25.5
1/week	4181	21.4	5129	23.0	77	21.3	22	16.1
2–3/week	3937	20.2	6442	28.9	77	21.3	47	34.3
4–8/week	1494	7.7	3609	16.1	52	14.3	33	24.1
Self-rated health								
Very good	7465	38.3	6233	27.9	117	32.3	27	19.7
Average or good	11,637	59.6	15,568	69.7	229	63.3	99	72.3
Poor	328	1.7	458	2.1	13	3.6	11	8.0
No data	79	0.4	65	0.3	3	0.8	0	0.0
Health-promoting behaviour								
Physical activity								
Very active efficient exerciser	5114	26.2	3930	17.6	84	23.2	13	9.5
Active efficient exerciser	6017	30.9	6623	29.7	105	29.0	44	32.1
Occasional/low efficient exerciser	4645	23.8	7224	32.3	78	21.5	44	32.1
Does not exercise	3671	18.8	4503	20.2	93	25.7	36	26.3
No data	62	0.3	44	0.2	2	0.6	0	0.0
Regular tooth brushing								
Several times/day	3982	20.4	10,831	48.5	53	14.6	69	50.4
About once/day	9737	49.9	9689	43.4	161	44.5	54	39.4
About 1–5 times/week or less	5689	29.2	1754	7.9	145	40.1	14	10.2
No data	101	0.5	50	0.2	3	0.8	0	0.0
Social support								
Nuclear family (with both parents)								
Yes	15,366	78.8	17,040	76.3	250	69.1	96	70.1
No	4022	20.6	5173	23.2	106	29.3	40	19.2
No data	121	0.6	111	0.5	6	1.6	1	0.7

Table 1 Distribution of participants according to age at baseline, predictor variables and outcome status, Finland *(Continued)*

Age at baseline and predictor variables in adolescence	Total population ($n = 41,833$)				Number of Deaths ($n = 499$)			
	Boys ($n = 19,509$)		Girls ($n = 22,324$)		Boys ($n = 362$)		Girls ($n = 137$)	
	No.	%	No.	%	No.	%	No.	%
Talking about issues to father								
Easy	10,421	53.4	8157	36.6	156	43.1	38	27.7
Difficult	6010	30.8	8470	37.9	115	31.8	49	35.8
Very difficult/No father	2571	13.2	5314	23.8	80	22.1	47	34.3
No data	507	2.6	383	1.7	11	3.0	3	2.2
Talking about issues to mother								
Easy	13,705	70.3	16,235	72.7	226	62.4	87	63.5
Difficult	4429	22.7	4743	21.2	100	27.6	32	23.4
Very difficult/No mother	1037	5.3	1175	5.3	31	8.6	16	11.6
No data	338	1.7	171	0.8	5	1.4	2	1.5
Talking about issues to friends								
Easy	14,764	75.7	20,078	89.9	258	71.3	120	87.6
Difficult	3558	18.2	1772	7.9	69	19.0	14	10.2
Very difficult/No friends	762	3.9	288	1.3	26	7.2	1	0.7
No data	425	2.2	186	0.9	9	2.5	2	1.5
School achievement								
Highest	3217	16.5	5481	24.6	29	8.0	27	19.7
2nd highest	5563	28.5	7590	34.0	82	22.6	37	27.0
2nd lowest	6993	35.8	6482	29.0	148	40.9	34	24.8
Lowest	3400	17.9	2577	11.5	101	27.9	37	27.0
No data	246	1.3	194	0.9	2	0.6	2	1.5

girls with 411,373 person-years of follow-up time (mean 18.4 years), was lower ($p < 0.001$) at 3.3 per 10,000 population.

Predictors of mortality in adolescent boys

Table 2 shows that family SES was significantly and inversely associated with risk of mortality in boys (Model 1), even when the effects of reserve capacity (Model 2) and school achievement (Model 3) were taken into account. Adjusted estimates showed that all reserve capacity dimensions were significant predictors of mortality. Increased risks of death were particularly observed among those with a chronic disease (HR 1.6, 95% 1.2–2.1) and many (4–8) stress symptoms (HR 1.7, 95% 1.2–2.3), those not brushing their teeth daily (HR 1.5, 95% 1.0–2.0), those without a nuclear family (HR 1.4, 95% 1.0–2.7) and those who cannot talk to father easily (HR 1.6, 95% CI 1.2–2.1). All categories below the highest school achievement strongly predicted the risk of mortality, even in the presence of other predictors. The interaction term between family SES and school achievement was not statistically significant.

Accounting for reserve capacity significantly reduced the effect of low SES on the risk of death, more so when school achievement was controlled for. Among boys whose parents had secondary education, HR estimates decreased by almost 19%, from 1.6 (95% CI 1.1–2.4) to 1.3 (95% CI 0.9–1.9). Total reduction in HR estimates was greater (27%) among those whose parents had basic/lower education, from 2.2 (95% CI 1.5–3.3) to 1.6 (95% CI 1.1–2.4). Interestingly, HR estimates for reserve capacity did not change markedly even with adjustment for the effect of school achievement.

Predictors of mortality in adolescent girls

There were fewer predictor variables significantly related to risk of mortality in girls (Table 3). Family SES was not associated with girls' risk of death. Accounting for the effects of family SES and school achievement (Model 3), increased mortality risks were observed among girls with poor perceived health indicated by poor self-rated health (HR 4.5, 95% CI 2.2–9.4) and lack of social support due to difficulty talking with one's father (HR 1.7, 95% 1.1–2.6). Only the lowest category of school achievement significantly increased their risk of death (HR 2.4, 95% CI 1.4–4.1). As observed in boys, the interaction term between family SES and school achievement was also not statistically significant.

Table 2 Cox proportional hazards models for the effect of socioeconomic status, reserve capacity variables and school achievement on mortality in Finland, Boys, Hazard ratios with 95% confidence interval (CI) estimates

Predictor variables in adolescence	Model 1[a]	Model 2[b]	Model 3[c]
Family SES (parents' education)			
Higher	1.0	1.0	1.0
Secondary	1.6 (1.1–2.4)*	1.5 (1.0–2.1)*	1.3 (0.9–1.9)
Basic or lower	2.2 (1.5–3.3)**	1.9 (1.3–2.9)**	1.6 (1.1–2.4)*
Perceived health			
Chronic disease			
No	1.0	1.0	1.0
Yes	1.5 (1.1–2.1)**	1.6 (1.2–2.1)*	1.6 (1.2–2.1)**
Perceived stress symptoms			
None	1.0	1.0	1.0
1/week	1.2 (0.9–1.5)	1.1 (0.8–1.5)	1.1 (0.8–1.5)
2–3/week	1.2 (0.9–1.6)	1.1 (0.8–1.5)	1.1 (0.8–1.5)
4–8/week	1.8 (1.3–2.6)**	1.7 (1.2–2.4)*	1.7 (1.2–2.3)**
Self-rated health			
Very good	1.0		
Average or good	1.2 (0.9–1.5)	n.s.	n.s.
Poor	1.8 (0.9–3.3)		
Health-promoting behaviour			
Physical activity			
Very active efficient exerciser	1.0		
Active efficient exerciser	1.0 (0.7–1.3)	n.s.	n.s.
Occasional/low efficient exerciser	1.0 (0.7–1.3)		
Does not exercise	1.3 (1.0–1.8)		
Regular tooth brushing			
Several times/day	1.0	1.0	1.0
Once/day	1.2 (0.9–1.7)	1.2 (0.9–1.7)	1.1 (0.8–1.6)
1–5 times/week or less	1.9 (1.3–2.6)**	1.7 (1.2–2.3)*	1.5 (1.0–2.0)*
Social support			
Nuclear family (with both parents)			
Yes	1.0	1.0	1.0
No	1.5 (1.2–1.9)*	1.4 (1.1–1.8)*	1.4 (1.0–1.7)*
Talking about issues to father			
Easy	1.0	1.0	1.0
Difficult	1.2 (0.9–1.6)	1.2 (1.0–1.6)	1.2 (1.0–1.6)
Very difficult/No father	1.6 (1.1–2.2)**	1.6 (1.2–2.1)*	1.6 (1.2–2.1)**
Talking about issues to mother			
Easy	1.0		
Difficult	1.2 (0.9–1.6)	n.s.	n.s.
Very difficult/No mother	1.2 (0.7–1.8)		
Talking about issues to friends			
Easy	1.0		
Difficult	1.0 (0.8–1.3)	n.s.	n.s
Very difficult/No friends	1.4 (0.9–2.1)		

Table 2 Cox proportional hazards models for the effect of socioeconomic status, reserve capacity variables and school achievement on mortality in Finland, Boys, Hazard ratios with 95% confidence interval (CI) estimates *(Continued)*

Predictor variables in adolescence	Model 1[a]	Model 2[b]	Model 3[c]
School achievement			
Highest	1.0		1.0
2nd highest	1.7 (1.1–2.6)*		1.6 (1.0–2.4)*
2nd lowest	2.4 (1.6–3.6)**	–	2.0 (1.3–3.1)**
Lowest	3.1 (2.0–4.7)**		2.3 (1.4–3.5)**

*$p < 0.05$; **$p < 0.001$; n.s. not significant
[a]Model 1. All predictor variables [b]Model 2. All significant reserve capacity variables from Model 1 and family SES. [c]Model 3. Model 2 variables and school achievement

In the crude model (Model 1), the lowest SES category, although not statistically significant, showed an inverse relationship with mortality (HR 1.4, 95% 0.8–2.4). However, this effect was diluted and HR estimates became null when reserve capacity and school achievement were taken into account. Similar to results seen in boys, HR estimates for reserve capacity did not change markedly even when school achievement was added into the model.

Discussion

Summary and interpretation of results

Our study found that family SES in adolescence significantly predicted risk of death only in boys. Among reserve capacity dimensions, poor perceived health (presence of chronic disease and weekly stress symptoms in boys; poor self-rated health in girls) as well as reduced social support (difficulty in talking to father in both groups; not living in a nuclear family in boys) generally increased the mortality risk of adolescents. Poor health-promoting behaviour (poor oral hygiene) increased the risk only in boys. Adolescents with low school achievement had 1.6–2.3 times higher risk of dying compared to the highest achievers. Reserve capacity and school achievement independently mitigated the effects of low SES on mortality risk among boys.

Family SES was related with boys' mortality risk in adolescence and early adulthood in our study. In Finland, previous research also revealed that health inequalities in adolescence and early adulthood persisted in boys from low SES environments possibly due to risky living standards and lifestyle-related factors [32, 33]. Likewise, studies on adult SES measures and outcomes presented stronger effects of SES on mortality for men relative to women because of underlying gender roles and other social characteristics [6, 10]. Typically, though, socioeconomic differentials in morbidity and mortality were recognised as less salient in the adolescent population compared to adults due to a certain level of "equalisation" of risk exposures [32, 34].

Our findings showed that all reserve capacity dimensions significantly predicted mortality risk in boys. Among girls, similar results were observed, except for health-promoting behaviour. A particular study which found difference in psychosocial resources between teenage boys and girls used a different dimension from those analysed in our study [28]. Thus, we cannot conclusively say that there are gender differentials in reserve capacity. Moreover, most epidemiological studies which dealt with reserve capacity's role in SES-health inequalities controlled for the effect of sex and combined results for both groups [17, 34, 35].

Since poor health perceptions are usually influenced by the presence of co-morbid conditions and symptoms [36], we included these along with self-rated health in the perceived health dimension. Studies have shown that perceived health was strongly and independently associated with mortality, even after controlling for known risk factors [36, 37], and objective physician ratings [38]. Researchers have explained that this indicator may have a summative property of capturing health aspects relevant to survival which are not measured by other health indicators [37]. In adolescence, health perceptions also reflect one's overall sense of psychosocial functioning aside from physical health [39]. Based on our results, changing self-perceptions of health and alleviating stress symptoms might improve both psychosocial and physical functioning in adolescence.

In our study, physical activity was not associated with the risk of death. Perhaps, this was because among those who died and regardless of their SES, both boys and girls were physically active in their adolescent years. Such health-promoting behaviour is usually adopted early in life [20] and further reinforced by school environments [40]. However, lack of health-promoting behaviour in terms of poor tooth brushing habits, was associated with boys' mortality risk. The girls in our study generally had good dental behaviour, hence, there was little variation in the distribution of exposure, unlike in boys. Tooth brushing behaviour, also formed during childhood, probably reflected family conditions, such as how well parents provide care and monitor their children's health behaviour, to some extent [22].

Table 3 Cox proportional hazards models for the effect of socioeconomic status, reserve capacity variables and school achievement on mortality in Finland, Girls, Hazard ratios with 95% confidence interval (CI) estimates

Predictor variables in adolescence	Model 1[a]	Model 2[b]	Model 3[c]
Family SES (parents' education)			
Higher	1.0	1.0	1.0
Secondary	1.0 (0.6–1.8)	1.0 (0.6–1.7)	0.9 (0.5–1.5)
Basic or lower	1.4 (0.8–2.4)	1.2 (0.7–2.1)	1.0 (0.6–1.8)
Perceived health			
Chronic disease			
No	1.0	n.s.	n.s.
Yes	1.2 (0.7–2.0)		
Perceived stress symptoms			
None	1.0		
1/week	0.8 (0.5–1.4)	n.s.	n.s.
2–3/week	1.3 (0.8–2.1)		
4–8/week	1.5 (0.9–2.5)		
Self-rated health			
Very good	1.0	1.0	1.0
Average or good	1.4 (0.9–2.2)	1.4 (0.9–2.2)	1.4 (0.9–2.1)
Poor	4.5 (2.1–9.6)**	5.2 (2.5–10.6)**	4.5 (2.2–9.4)**
Health-promoting behaviour			
Physical activity			
Very active efficient exerciser	1.0		
Active efficient exerciser	1.9 (1.0–3.5)*	n.s.	n.s.
Occasional or low efficient exerciser	1.7 (0.9–3.2)		
Does not exercise	2.0 (1.0–3.8)*		
Regular tooth brushing			
Several times/day	1.0		
Once/day	0.9 (0.6–1.3)	n.s.	n.s.
1–5 times/week or less	1.3 (0.7–2.3)		
Social support			
Nuclear family (with both parents)			
Yes	1.0	n.s	n.s.
No	1.2 (0.8–1.7)		
Talking about issues to father			
Easy	1.0	1.0	1.0
Difficult	1.3 (0.8–2.0)	1.2 (0.8–1.9)	1.3 (0.8–1.9)
Very difficult/No father	1.7 (1.0–2.8)*	1.8 (1.1–2.7)*	1.7 (1.1–2.6)*
Talking about issues to mother			
Easy	1.0		
Difficult	1.0 (0.6–1.6)	n.s	n.s.
Very difficult/No mother	1.9 (1.1–3.5)*		
Talking about issues to friends			
Easy	1.0		
Difficult	1.1 (0.6–1.9)	n.s	n.s.
Very difficult/No friends	0.4 (0.6–3.1)		

Table 3 Cox proportional hazards models for the effect of socioeconomic status, reserve capacity variables and school achievement on mortality in Finland, Girls, Hazard ratios with 95% confidence interval (CI) estimates *(Continued)*

Predictor variables in adolescence	Model 1[a]	Model 2[b]	Model 3[c]
School achievement			
Highest	1.0		1.0
2nd highest	1.0 (0.6–1.6)		1.0 (0.6–1.6)
2nd lowest	1.0 (0.6–1.7)	–	1.0 (0.6–1.6)
Lowest	2.8 (1.7–4.7)**		2.4 (1.4–4.1)**

*$p < 0.05$; **$p < 0.001$; n.s. not significant
[a]Model 1. All predictor variables [b]Model 2. All significant reserve capacity variables from Model 1 and family SES. [c]Model 3. Model 2 variables and school achievement

Research on the effect of social support on mortality was extensive. A meta-analytic review showed that overall effect size of being in social relationships provided up to a 50% increase in odds of survival [24]. In our study, important aspects of social support were related to family structure and communication with father. Researchers have recognized that a "risky" family environment early in life predisposed children to various emotional and physical disorders [9, 17, 34]. In a study among Hungarian adolescents, a non-intact family structure was a significant determinant of risky health behaviours such as use of cigarettes, alcohol and marijuana [34]. Our results showed that poor communication with one's father increased the mortality risk of adolescents. However, the mechanisms by which communication with one's father influences health during adolescence is beyond the scope of our study. Nonetheless, our results, comparable to earlier findings [41], underscore the importance of paternal relationship as a form of social support. This is congruent with evidence that showed children had less emotional and behavioral problems with father's involvement during childhood and adolescence [42].

School achievement also significantly predicted the risk of death in both genders in our study. Previous studies showed that school achievement in adolescence empowered a person to make healthy choices and adopt healthy habits [25, 26]. It also ensured completion of high school education, often leading to a college degree, greatly improving one's future SES [8, 26]. In our study, increasing mortality risk in boys was estimated with each category below the highest achievement. In girls, only the lowest category was significantly related to risk of death. The lack of interaction between family SES and school achievement implies that both education-related variables exhibit a similar and expected gradient with mortality.

As shown in literature [11, 17, 19], our results demonstrated that reserve capacity reduced the effect of low SES on mortality risk among boys. Interestingly, the addition of school achievement into the model further weakened the effect of low SES on boys' risk of death. Yet, it did not modify the risk estimates obtained from the reserve capacity dimensions, suggesting that these factors are important predictors which independently affect mortality risks in adolescents. The results of our study lend further support for the life-course approach to the SES-health relationship.

Strengths and weaknesses

Most studies have utilized either childhood or adult markers of SES. Adolescent indicators are seldom used, even though adolescence is a critical period in developing sound psychological and behavioural patterns, which are carried forward into adulthood [28]. Our prospective study addressed this research gap using large, nationwide samples with a long follow-up period and reliable register-based data. Our study added support to the importance of the life-course approach in epidemiologic research on SES-health inequalities.

Studies which dealt with a reserve capacity framework among adolescents were limited. The opportunity to combine survey data with register-based data on death made it possible to build a longitudinal dataset and study potential psychosocial factors mediating the SES-health gradient. Since the survey data was collected in the 1980s and 1990s, it was not designed to measure dimensions of reserve capacity. Due to this, we needed to use proxy measures for each reserve capacity dimension. The selection of variables was based on a cluster of single-item indicators which correlated with each other. However, proxy measures may give unreliable results and further research is needed to validate these.

Despite issues in measurements, we tried to analyse a wide range of reserve capacity dimensions. This follows the methodological framework of the proponents of reserve capacity who emphasised that it is "a bank of resilient resources that contributes to the SES and health relationship" [9, 17–19] instead of a single psychosocial factor or dimension. Moreover, we presented results disaggregated by sex, providing evidence to the interconnections of SES, gender and health inequalities.

Conclusions

We found that reserve capacity, measuring psychosocial resources plus health-promoting behaviour, and good school achievement in adolescence reduce the risk of mortality in adolescence and early adulthood. In boys, these also mitigate the negative effect of low SES on mortality. These findings underscore the role of reserve capacity and school achievement during adolescence as likely causal or mediating mechanisms in SES-health inequalities.

Abbreviations

AHLS: Adolescent Health and Lifestyle Surveys; AIC: Akaike Information Criteria; CI: confidence interval; HR: hazard ratio; OR: odds ratio; SES: socioeconomic status

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Availability of data and materials

The dataset analysed during the current study is not publicly available due to contract specifications with Statistics Finland. Data are however available from the authors upon reasonable request and with permission of the Adolescent Health and Lifestyle Research Group at the University of Tampere, Finland.

Authors' contributions

PC, LK and AR designed and conceptualised the study. PC analysed the data and wrote the first draft of the manuscript. LK, JB and AR contributed to the interpretation of data and critically reviewed and revised the manuscript. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Ethics approval and consent to participate

Statistics Finland performed the data linkage of the national registries and the data of the Adolescent Health and Lifestyle Surveys (AHLS) from the years 1979–1997 according to a contract specifying the rights and duties of both parties. The study protocol was approved by its Institutional Review Board and the Data Protection Ombudsman. The Joint Commission on Ethics of the University of Turku and the Turku University Hospital 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. The survey data used in the study were gathered in 1979–1997. The first review boards at the universities were established in Finland in the 1980s. AHLS was reviewed by the Ethical Review Board of the University of Helsinki, Department of Public Health in 1986. Parental consent was not considered by the ethics review board at that time. In later surveys, the latest in 2017, the relevant review boards have waived the parental consent for the now adult participants and the consent of the participants was assumed upon return of the completed questionnaire.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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References

1. Adler NE, Newman K. Socioeconomic disparities in health: pathways and policies. Health Aff (Millwood). 2002;21(2):60–76.
2. Gottfredson LS. Intelligence: is it the Epidemiologists' elusive "fundamental cause" of social class inequalities in health? Journal of Personality & Social Psychology. 2004;86(1):174–99.
3. Smith GD, Hart C, Blane D, Gillis C, Hawthorne V. Lifetime socioeconomic position and mortality: prospective observational study. BMJ. 1997; 314(7080):547.
4. Liu L, Xue F, Ma J, Ma M, Long Y, Newschaffer CJ. Social position and chronic conditions across the life span and risk of stroke: a life course epidemiological analysis of 22 847 American adults in ages over 50. Int J Stroke. 2013;8:50–5.
5. Kuh D, Hardy R, Langenberg C, Richards M, Wadsworth ME. Mortality in adults aged 26-54 years related to socioeconomic conditions in childhood and adulthood: post war birth cohort study. BMJ. 2002;325(7372):1076–80.
6. Juárez SP, Goodman A, Koupil I. From cradle to grave: tracking socioeconomic inequalities in mortality in a cohort of 11 868 men and women born in Uppsala, Sweden, 1915–1929. J Epidemiol Community Health. 2016;70(6):569–75.
7. Phelan JC, Link BG, Tehranifar P. Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. J Health Soc Behav. 2010;51(1):Suppl 28–40. doi:10.1177/0022146510383498.
8. Mackenbach JP, Kulhanova I, Bopp M, Deboosere P, Eikemo TA, Hoffmann R, et al. Variations in the relation between education and cause-specific mortality in 19 European populations: a test of the "fundamental causes" theory of social inequalities in health. Soc Sci Med. 2015;127:51–62.
9. Matthews KA, Gallo LC. Psychological perspectives on pathways linking socioeconomic status and physical health. Annu Rev Psychol. 2011;62:501–30.
10. Phillips SP, Hamberg K. Women's relative immunity to the socio-economic health gradient: artifact or real? Glob Health Action. 2015;8:27259.
11. Chen E, Miller GE. "Shift-and-persist" strategies: why low socioeconomic status Isn't always bad for health. Perspect Psychol Sci. 2012;7(2):135–58.
12. Gluckman PD, Hanson MA, Spencer HG, Bateson P. Environmental influences during development and their later consequences for health and disease: implications for the interpretation of empirical studies. Proc R Soc B Biol Sci. 2004;272(1564):671–7.
13. McDade TW, Rutherford J, Adair L, Kuzawa CW. Early origins of inflammation: microbial exposures in infancy predict lower levels of C-reactive protein in adulthood. Proc R Soc B Biol Sci. 2009;277(1684):1129–37.
14. Cozen W, Hamilton AS, Zhao P, Salam MT, Deapen DM, Nathwani BN, et al. A protective role for early oral exposures in the etiology of young adult Hodgkin lymphoma. Blood. 2009;114(19):4014–20.
15. Lynch SV, Wood RA, Boushey H, Bacharier LB, Bloomberg GR, Kattan M, et al. Effects of Early Life Exposure to Allergens and Bacteria on Recurrent Wheeze and Atopy in Urban Children. J Allergy Clin Immunol 2014;134(3): 593–601.e12.
16. George MP. The Mexican American Health Paradox: The Collective Influence of Sociocultural Factors on Hispanic Health Outcomes. Discussions [Internet]. 2013;9(2). http://www.inquiriesjournal.com/a?id=663. Accessed 1 Sep 2017.
17. Matthews KA, Gallo LC, Taylor SE. Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. Ann N Y Acad Sci. 2010;1186:146–73.

18. Gallo LC. The reserve capacity model as a framework for understanding psychosocial factors in health disparities. Applied Psychology: Health and Well-Being. 2009;1(1):62–72.
19. Gallo LC, Espinosa de los Monteros K, Shivpuri S. Socioeconomic status and health: what is the role of reserve capacity? Curr Dir Psychol Sci. 2009;18(5):269–74.
20. Moor I, Rathmann K, Stronks K, Levin K, Spallek J, Richter M. Psychosocial and behavioural factors in the explanation of socioeconomic inequalities in adolescent health: a multilevel analysis in 28 European and north American countries. J Epidemiol Community Health. 2014;68(10):912–21. doi:10.1136/jech-2014-203933.
21. Pakpour AH, Sniehotta FF. Perceived behavioural control and coping planning predict dental brushing behaviour among Iranian adolescents. J Clin Periodontol. 2012;39(2):132–7.
22. Cinar AB, Tseveenjav B, Murtomaa H. Oral health-related self-efficacy beliefs and toothbrushing: Finnish and Turkish pre-adolescents' and their mothers' responses. Oral Health Prev Dent. 2009;7(2):173–81.
23. Robbins LB, Pender NJ, Ronis DL, Kazanis AS, Pis MB. Physical activity, self-efficacy, and perceived exertion among adolescents. Res Nurs Health. 2004;27(6):435–46.
24. Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. PLoS Med. 2010;7(7):e1000316.
25. Koivusilta L, West P, Saaristo VMA, Nummi T, Rimpela A. From childhood socio-economic position to adult educational level - do health behaviours in adolescence matter? A longitudinal study. BMC Public Health. 2013;13(1):711.
26. Freudenberg N, Ruglis J. Reframing school dropout as a public health issue. Prev Chronic Dis. 2007; https://www.cdc.gov/pcd/issues/2007/oct/07_0063.htm. Accessed 9 June 2016.
27. Lê-Scherban F, Diez Roux AV, Li Y, Morgenstern H. Does academic achievement during childhood and adolescence benefit later health? Ann Epidemiol. 2016;24(5):344–55.
28. Finkelstein DM, Kubzansky LD, Capitman J, Goodman E. Socioeconomic differences in adolescent stress: the role of psychological resources. J Adolesc Health. 2007;40(2):127–34.
29. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. J Epidemiol Community Health. 2003;57(10):778–83.
30. UCLA. Statistical consulting group. Survival Analysis with Stata: Statistica Computing Seminars; 2015. https://stats.idre.ucla.edu/stata/seminars/stata-survival/. Accessed 16 Dec 2015.
31. Bozdogan H. Model selection and Akaike's information criterion (AIC): the general theory and its analytical extensions. Psychometrika. 1987;52(3):345–70.
32. Remes H, Martikainen P, Valkonen T. Mortality inequalities by parental education among children and young adults in Finland 1990-2004. J Epidemiol Community Health. 2010;64(2):136–41.
33. Torikka A, Kaltiala-Heino R, Rimpelä A, Marttunen M, Luukkaala T, Rimpelä M. Self-reported depression is increasing among socio-economically disadvantaged adolescents-repeated cross-sectional surveys from Finland from 2000 to 2011. BMC Public Health. 2014;14(1):1–10.
34. Piko BF, Fitzpatrick KM. Socioeconomic status, psychosocial health and health Behaviours among Hungarian adolescents. Eur J Pub Health. 2007;17(4):353–60.
35. Sjogren E, Kristenson M. Linquest group. Can gender differences in psychosocial factors be explained by socioeconomic status? Scand J Public Health. 2006;34(1):59–68.
36. DeSalvo KB, Bloser N, Reynolds K, He J, Muntner P. Mortality prediction with a single general self-rated health question: a meta-analysis. J Gen Intern Med. 2005;21(3):267–75.
37. Mackenbach JP, Simon JG, Looman CWN, Joung IMA. Self-assessed health and mortality: could psychosocial factors explain the association? Int J Epidemiol. 2002;31(6):1162–8.
38. Todd MA, Goldman N. Do interviewer and physician health ratings predict mortality?: a comparison with self-rated health. Epidemiology. 2013;24(6)
39. Page RM, Simonek J, Ihász F, Hantiu I, Uvacsek M, Kalabiska I, et al. Self-rated health, psychosocial functioning, and other dimensions of adolescent health in central and eastern European adolescents. The European Journal of Psychiatry. 2009;23(2):101–14.
40. Moore GF, Littlecott HJ. School- and family-level socioeconomic status and health behaviors: multilevel analysis of a National Survey in Wales, United Kingdom. J Sch Health. 2015;85(4):267–75.
41. Salonna F, Geckova AM, Zezula I, Sleskova M, Groothoff JW, Reijneveld SA, et al. Does social support mediate or moderate socioeconomic differences in self-rated health among adolescents? Int J Public Health. 2012;57(3):609–17.
42. East L, Jackson D, O'Brien L. Father absence and adolescent development: a review of the literature. Journal of Child Health Care. 2006;10(4):283–95.

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PUBLICATION II

How socioeconomic circumstances, school achievement and reserve capacity in adolescence predict adult education level: a three-generation study in Finland

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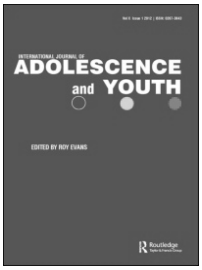

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How socioeconomic circumstances, school achievement and reserve capacity in adolescence predict adult education level: a three-generation study in Finland

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ABSTRACT

Family socioeconomic circumstances directly influence adult education level. Adolescent psychosocial resources and health-promoting behaviour collectively termed as 'reserve capacity' and school achievement may likely mediate the effect of family socioeconomic circumstances on adult education level. We tested these relationships using 1985–1995 survey data on 12–18-year-old Finns ($N = 41,822$) linked with three-generation registry data of Statistics Finland until 2009. Results of the multinomial logistic regression models, adjusted for sex and age at end of follow-up, showed that socioeconomic circumstances of parents and grandparents predicted adult education level. School achievement and reserve capacity dimensions of perceived health, health-promoting behaviour and social support in adolescence also positively predicted adult education. Moreover, these tended to decrease the effect of family socioeconomic circumstances on educational level. Our findings suggest that formulating interventions which build reserve capacity and improve school performance, especially among adolescents from disadvantaged socioeconomic backgrounds, could likely reduce educational inequalities.

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Education; socioeconomic; psychosocial; reserve capacity; school achievement

Introduction

Education is a strong predictor of health (Freudenberg & Ruglis, 2007; Liu & Hummer, 2008). Studies have robustly shown that a low educational attainment is associated with poorer health outcomes (Fergusson, Horwood, & Boden, 2008; Matthews & Gallo, 2011) and shorter life expectancies (Mackenbach et al., 2015; Spittel, Riley, & Kaplan, 2015). Additionally, education predicts an individual's future occupational prospects and earning capacities (Adler & Newman, 2002; Matthews & Gallo, 2011) and influences one's life-course opportunities, including those of the offspring (Fergusson et al., 2008). It is commonly used as an indicator of socioeconomic status (SES) and recognized as a key marker of success in adulthood (Slominski, Sameroff, Rosenblum, & Kasser, 2011). Thus, one of the goals included in the 2030 Agenda for Sustainable Development by multilateral groups in partnership with the United Nations, is universal access to education at all levels (United Nations, n.d.).

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Evidence points to socioeconomic circumstances of the family as largely shaping the mechanisms and processes of an individual's educational attainment (Conger, Conger, & Martin, 2010; Fergusson et al., 2008; Koivusilta, West, Saaristo, Nummi, & Rimpelä, 2013; Merritt & Buboltz, 2015; Slominski et al., 2011). The socioeconomic circumstances of the family determine available resources for investments in the human capital formation of children, such as health and education (Bird, 2007), and also the transfer of these resources from one generation to another (Albertini & Radl, 2012). Hence, even in high income countries, children born in low SES families have higher risk of educational failure and underachievement (Fergusson et al., 2008). They also have increased tendencies to acquire low SES in adulthood (Matthews, Gallo, & Taylor, 2010).

Aside from family SES, cognitive ability, usually measured through academic competence or school achievement, strongly determines educational attainment in adulthood. Good grades obtained in secondary school were strong predictors of enrolment in higher education (Brekke, 2015). Even grades obtained early in elementary school had predicted adult educational attainment (Entwisle, Alexander, & Olson, 2005). Academic competence incites higher academic aspirations and enables one to meet the rigors of post-secondary education (Merritt & Buboltz, 2015).

A low SES family background is the earliest exposure and risk factor for having less education and low adult SES in the life-course perspective (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). Adolescence follows this early life environment and further shapes psychosocial development, (Kroenke, 2008) which is a potential pathway for adult educational outcomes (Murasko, 2007). Researchers found that low SES families who provided psychosocial resources through cognitive and emotional support raised resilient children who succeeded academically (Merritt & Buboltz, 2015) and functioned well in life compared to their low SES counterparts without such resources (Kroenke, 2008). These psychosocial resources were integrated as the concept of reserve capacity and include interpersonal resources such as social support and integration and intrapersonal characteristics such as self-efficacy, mastery or a sense of perceived control (Gallo, Espinosa de los Monteros, & Shivpuri, 2009; Gallo & Matthews, 2003; Matthews & Gallo, 2011; Matthews et al., 2010). It was proposed that individuals with high reserve capacity gain the coping skills necessary to attain higher education while those with low reserve capacity may lack these skills and attain lower education (Matthews et al., 2010). Such a mechanism raises the question of how reserve capacity can mediate the effect of family SES on future educational attainment. We further extend the reserve capacity framework to include dental brushing behaviour and physical activity as these have been shown to improve with high self-efficacy (Cinar, Tseveenjav, & Murtomaa, 2009; Pakpour & Sniehotta, 2012; Robbins, Pender, Ronis, Kazanis, & Pis, 2004). Our study, therefore, focuses on three dimensions of reserve capacity: perceived health, health-promoting behaviour and social support.

While most empirical data dealt with transmission of SES from parents to offspring, recent findings have demonstrated that grandparents' occupational class could be transmitted to grandchildren (Chan & Boliver, 2013; Erola & Moisio, 2007) and that other capital of grandparents could influence their grandchildren's educational success (Møllegaard & Jæger, 2015). This implies that transmission of low education across generations of families could perpetuate a cycle of socioeconomic disadvantage. In order to break this, it is important to elucidate the origin of inequalities in education and understand the processes which create these. It is in this perspective that we aim to investigate if the effect of family SES on adult education level persists across three generations, implying that educational inequalities may have originated from socioeconomic circumstances of grandparents. Moreover, we want to determine how reserve capacity and school achievement in adolescence modify the associations between family socioeconomic circumstances and adult education level.

Methods

Study design

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, conducted biennially since 1977, monitors the health and health-related lifestyle of adolescents in Finland. Nationally representative samples of 12-, 14-, 16-, and 18-year-old Finns born on certain days in June, July and August were drawn each study year from the Population Register Centre. Variables measured across all survey rounds were used. A self-administered questionnaire was sent in February, followed by two re-inquiries to non-respondents. Eligible data from 41,822 adolescents (79.2% response rate) were included. Response rates by sex and age groups were as follows: 72.4% in boys (n = 19,504), 86.3% in girls (n = 22,318), at least 80% in adolescents aged 12 years (n = 3,948), 14 years (12,583) and 16 years (n = 13,582), respectively and 75.4% in those aged 18 years (n = 11,709).

Follow-up data were obtained from registries of Statistics Finland, which contained socioeconomic information for the AHLS participants, their parents and grandparents. 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. Follow-up started on 30 April, each survey year, and ended on 31 December 2009. At the end of the follow-up, the participants' ages ranged from 27 to 43 years.

Statistics Finland had constructed family formation data to link generations. In the earlier censuses, children (parents in this study) who were no longer living with their parents (grandparents in this study) during the time of the census could not be linked to their families, which explains the large number of grandchildren with unknown data for grandparents (Table 1). Part of the missing information is due to the late digitalization of the censuses (from 1970 onwards). The proportion of adolescents with unknown grandparents' data by adult education level was similar to those of adolescents whose grandparents had low education and rented dwellings. In terms of other variables, the pattern of distribution found in adolescents with unknown grandparents followed the distributions obtained in the total population. Further analyses were made to assess the effect of including this group in our study (Appendix 1).

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. Identification of the study participants was withheld from the investigators.

Outcome variable

Adult education level

The adolescent's highest educational level was used and grouped according to years of schooling: low (9 years or less), middle (10–12 years), and high education (>12 years).

Predictor variables

Several indicators of family socioeconomic circumstances were used. All parents' and grandparents' data were obtained nearest to the year when the adolescent was aged 15 years. Parental data obtained more than five years away from the child's 15th birthday and data from those whose parents died before the AHLS year were considered missing to ensure that only parental influences within adolescence were measured.

Education level of father, mother, maternal and paternal grandparents

Education levels of parents and grandparents were categorized in the same way as that of the adolescents'. Data on grandfather and grandmother from either maternal or paternal side were combined. Where both grandparents existed and information was different, the one with the higher category was used. In case of missing data from one grandparent, the available information from the other grandparent was used.

Table 1. Distribution of family socioeconomic circumstances, school achievement and reserve capacity variables in adolescence according to education level in adulthood.

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence			Education level in adulthood				
			Total population $N = 41,822$		Low $n = 3801$	Middle $n = 23,073$	High $n = 14,948$
			No.	%	Row %	Row %	Row %
Family variables							
Education	Father	Low	17,212	41.2	12.0	62.2	25.8
		Middle	18,481	44.2	7.7	55.2	37.1
		High	5500	13.1	3.3	32.4	64.3
		Missing	629	1.5	18.4	63.0	18.6
	Mother	Low	16,186	38.7	12.5	63.0	24.5
		Middle	22,121	52.9	7.5	53.1	39.4
		High	3483	8.3	3.4	31.5	65.1
		Missing	32	.1	31.3	53.1	15.6
	Paternal grandparents	Low	18,643	44.6	8.4	55.8	35.8
		Middle	3969	9.5	7.1	48.1	44.8
		High	1070	2.5	4.6	37.2	58.2
		Unknown	18,140	43.4	10.5	57.1	32.4
	Maternal grandparents	Low	19,144	45.8	8.4	56.1	35.5
		Middle	4324	10.3	7.6	48.4	44.0
		High	938	2.3	4.5	36.0	59.5
		Unknown	17,416	41.6	10.4	56.9	32.7
Dwelling ownership	Father	Rented	5972	14.3	16.9	60.1	23.0
		Owner-occupied	32,711	78.2	7.2	53.7	39.1
		Missing	3139	7.5	14.1	60.7	25.2
	Mother	Rented	7052	16.9	17.6	60.4	22.0
		Owner-occupied	33,659	80.4	7.1	53.9	39.0
		Missing	1111	2.7	14.1	60.7	25.2
	Paternal grandparents	Rented	3364	8.0	10.5	56.4	33.1
		Owner-occupied	19,302	46.2	7.5	53.2	39.3
		Unknown	19,156	45.8	10.4	67.0	32.6
	Maternal grandparents	Rented	3554	8.5	11.6	58.2	30.2
		Owner-occupied	19,975	47.8	7.5	53.2	39.3
		Unknown	18,293	43.7	10.4	56.7	32.9
Employment status	Father	Unemployed	4430	10.6	13.1	60.8	26.1
		Employed	35,076	83.9	8.2	54.1	37.7
		Missing	2316	5.5	14.9	60.4	24.7
	Mother	Unemployed	4923	11.8	13.9	58.3	27.8
		Employed	36,415	87.0	8.4	54.6	37.0
		Missing	484	1.2	14.5	62.0	23.5
Adolescence variables							
School achievement		Low	19,533	46.7	15.8	68.2	16.0
		Average	13,152	31.4	3.9	51.8	44.3
		High	8697	20.8	1.3	30.5	68.2
		Missing	440	1.1	24.1	62.0	13.9
Reserve capacity							
Perceived health	Chronic disease	Yes	3905	9.3	11.8	54.8	33.4
		No	37,917	90.7	8.8	55.2	36.0
	Perceived stress symptoms	4–8/week	5100	12.2	12.3	55.4	32.3
		2–3/week	10,376	24.8	9.4	53.7	36.9
		1/week	9308	22.3	8.5	54.8	36.7
		None	17,038	40.7	8.2	56.2	35.6
	Self-rated health	Poor	785	1.9	16.3	54.9	28.8
		Average or good	27,198	65.0	9.3	55.8	34.9
		Very good	13,695	32.8	8.3	53.8	37.9
		Missing	144	.3	13.9	55.5	30.6

(*Continued*)

Table 1. (*Continued*).

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence			Education level in adulthood				
			Total population $N = 41,822$		Low $n = 3801$	Middle $n = 23,073$	High $n = 14,948$
			No.	%	Row %	Row %	Row %
Health-promoting behaviour	Physical activity	Does not exercise	8169	19.5	13.6	60.8	25.6
		Occasional/low efficient exerciser	11,868	28.4	8.7	57.0	34.3
		Active efficient exerciser	12,639	30.2	7.9	52.8	39.3
		Very active efficient exerciser	9040	21.6	7.0	51.1	41.9
		Missing	106	.3	22.6	51.9	25.5
	Regular tooth brushing	<1–5 times/week	7443	17.8	17.6	62.9	19.5
		About once/day	19,421	46.4	8.3	56.5	35.2
		Several times/day	14,807	35.4	5.8	49.5	44.7
		Missing	151	.4	13.9	60.9	25.2
Social support	Nuclear family	No	9192	22.0	15.6	59.0	25.4
		Yes	32,398	77.5	7.2	54.0	38.8
		Missing	232	.5	17.7	59.0	23.3
	Talking about issues to father	Difficult/No father	22,363	53.5	9.3	54.8	35.9
		Easy	18,572	44.4	8.4	55.3	36.3
		Missing	887	2.1	17.6	62.6	19.8
	Talking about issues to mother	Difficult/No mother	11,384	27.2	10.1	55.2	34.7
		Easy	29,930	71.6	8.5	55.1	36.4
		Missing	508	1.2	18.3	59.5	22.2
	Talking about issues to friends	Difficult/No friends	6379	15.2	10.1	55.2	35.7
		Easy	34,833	83.3	8.7	55.1	36.2
		Missing	610	1.5	17.5	60.7	21.8

Dwelling ownership of father, mother, maternal and paternal grandparents

Dwelling ownership was classified as either owner-occupied (owned a house or had shares in the housing unit) or rented (living in a rented apartment).

Employment status of father and mother

Employment status was based on the indicated response (employed, unemployed, unknown) about one's main activity. The category 'unemployed' also included those who had at least one month of unemployment during the preceding twelve months of the census. Because most grandparents had retired, this variable was used for parents only.

Reserve capacity

Reserve capacity was measured in three distinct dimensions of intra- and interpersonal factors. Within each dimension of reserve capacity (AHLS data), correlations and associations of the variables were calculated. We found moderate positive correlations (Spearman's) and statistically significant associations (Pearson chi-square tests) within the items described per dimension.

(a) *Perceived health* included three items: reported chronic disease, injury or disability that restricts daily activities (no/yes); a summary index of weekly perceived stress symptoms (stomachaches, tension or nervousness, irritability or outbursts of anger, trouble falling asleep or waking at night,

Table 2. Bivariate associations of each predictor variable with education level in adulthood (using low education as reference category), adjusting for sex and age at end of follow-up.

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence			Odds ratios, 95% confidence intervals	
			Middle	High
Family variables				
Education	Father	Low	1.0	1.0
		Middle	1.3 (1.3–1.5)***	2.1 (1.9–2.3)***
		High	1.8 (1.5–2.1)***	8.3 (7.0–9.8)***
	Mother	Low	1.0	1.0
		Middle	1.4 (1.3–1.5)***	2.6 (2.4–2.8)***
		High	1.9 (1.5–2.4)***	9.4 (7.6–11.6)***
	Paternal grandparents	Low	1.0	1.0
		Middle	1.1 (.9–1.2)	1.5 (1.3–1.7)***
		High	1.2 (.9–1.6)	2.9 (2.1–4.0)***
		Unknown	.8 (.8–.9)***	.8 (.8–.9)***
	Maternal grandparents	Low	1.0	1.0
		Middle	1.0 (.9–1.2)	1.4 (1.2–1.6)***
		High	1.2 (.8–1.6)	3.1 (2.2–4.3)***
		Unknown	.8 (.8–.9)***	.9 (.8–.9)**
Dwelling ownership	Father	Rented	1.0	1.0
		Owner-occupied	2.1 (1.9–2.3)***	4.0 (3.6–4.4)***
	Mother	Rented	1.0	1.0
		Owner-occupied	2.2 (2.0–2.4)***	4.3 (3.9–4.7)***
	Paternal grandparents	Rented	1.0	1.0
		Owner-occupied	1.3 (1.1–1.5)***	1.6 (1.4–1.9)***
		Unknown	1.0 (.9–1.2)	1.2 (1.0–1.3)*
	Maternal grandparents	Rented	1.0	1.0
		Owner-occupied	1.5 (1.3–1.8)***	2.1 (1.9–2.5)***
		Unknown	1.2 (1.0–1.3)*	1.4 (1.3–1.7)***
Employment status	Father	Unemployed	1.0	1.0
		Employed	1.4 (1.3–1.6)***	2.4 (2.2–2.8)***
	Mother	Unemployed	1.0	1.0
		Employed	1.6 (1.4–1.8)***	2.4 (2.1–2.6)***
Adolescence variables				
School achievement		Low	1.0	1.0
		Average	3.0 (2.7–3.3)***	10.7 (9.6–12.0)***
		High	5.6 (4.5–7.0)***	53.6 (43.0–66.8)***
Reserve capacity				
Perceived health	Chronic disease	Yes	1.0	1.0
		No	1.2 (1.1–1.4)**	1.3 (1.1–1.5)***
	Perceived stress symptoms	4–8/week	1.0	1.0
		2–3/week	1.3 (1.2–1.5)***	1.6 (1.4–1.8)***
		1/week	1.6 (1.4–1.8)***	1.8 (1.6–2.1)***
		None	1.7 (1.5–2.0)***	2.0 (1.8–2.3)***
	Self-rated health	Poor	1.0	1.0
		Average or good	1.4 (1.1–1.8)**	1.5 (1.2–2.0)**
		Very good	1.5 (1.2–1.9)**	1.9 (1.5–2.5)***
Health-promoting behaviour	Physical activity	Does not exercise	1.0	1.0
		Occasional/low efficient exerciser	1.3 (1.2–1.5)***	1.8 (1.6–2.0)***
		Active efficient exerciser	1.4 (1.2–1.6)***	2.3 (2.1–2.6)***
		Very active efficient exerciser	1.6 (1.4–1.8)***	2.9 (2.5–3.3)***
	Regular tooth brushing	<1–5 times/week	1.0	1.0
		About once/day	1.7 (1.6–1.9)***	3.2 (2.8–3.5)***
		Several times/day	1.9 (1.7–2.2)***	4.9 (4.4–5.5)***
Social support	Nuclear family	No	1.0	1.0
		Yes	2.2 (2.0–2.4)***	3.8 (3.4–4.2)***
	Talking about issues to father	Difficult/No father	1.0	1.0
		Easy	1.1 (1.0–1.2)	1.1 (1.0–1.2)*
	Talking about issues to mother	Difficult/No mother	1.0	1.0
		Easy	1.1 (1.0–1.2)	1.1 (1.0–1.2)
	Talking about issues to friends	Difficult/No friends	1.0	1.0
		Easy	1.0 (.9–1.1)	.9 (.8–1.0)

*$p < .05$; **$p < .01$; ***$p < .001$ – Significance levels.

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, good to average, poor.

(b) *Health-promoting behaviour* 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.

(c) *Social support* 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 or difficult). Those who did not have a father (5%), mother (1%) or friends (.5%) were included in the 'difficult' category.

School achievement

Adolescents were categorized as having low, average or high academic achievement. The respondents were asked to assess whether their end-of-term school performance was much better, slightly better, average, slightly poorer or much poorer than the class average. For 12–14-year-olds (all in comprehensive schools), those who reported much better performance were classified as 'high', those with slightly better performance as 'average' while the rest were all classified as having 'low' achievement. For 16–18-year-olds, in addition to self-assessment of their school performance, school status (academic upper secondary school/vocational school/not attending school) was also used. Their achievement was classified as follows: high (in academic upper secondary school with better performance); average (in vocational school with better performance or academic upper secondary school with average performance); and, low (in vocational school with poor to average performance or high school with poor performance or not at school).

Statistical analysis

Descriptive statistics were presented as percentages for categorical variables. We used multinomial logistic regression analysis to investigate the associations of predictor variables with the outcome. In both bivariate and multivariate analyses, we adjusted for sex and age at the end of follow-up because of unequal follow-up times among the participants.

Three multivariate models were fitted using a backward elimination approach. Variables included were only those statistically significant in bivariate analyses (Table 2). The first model named Model 1 examined family SES variables; Model 2 included the Model 1 variables plus school achievement; and, Model 3 (final model) consisted of all statistically significant family socioeconomic variables, school achievement and reserve capacity variables. Due to the numerous predictors considered in each model, statistical significance was set at $p < .01$ for retaining variables in the models. Model fit was assessed using Akaike information criterion (AIC) values and likelihood ratio tests. The model parameters were presented as odds ratios (ORs) with 95% confidence intervals (CIs). All analyses were performed using STATA version 12.1.

Results

A third (35.7%) of the adolescents achieved high education in adulthood, about half (55.2%) attained a middle education and less than a tenth (9.1%) had low adult education level. Table 1 presents the distributions of the predictor variables by adolescents' adult education level. Generally, the proportion of adolescents who obtained high adult education level increased with better family socioeconomic circumstances, high achievement in school and positive reserve capacity characteristics. The opposite

Table 3. Multivariate associations of each predictor variable with education level in adulthood (using low education as reference category) in three models, adjusting for sex and age at end of follow-up.

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence			Model 1[a]		Model 2[b]		Model 3 (final model)[c]	
			Odds ratios, 95% confidence intervals		Odds ratios, 95% confidence intervals		Odds ratios, 95% confidence intervals	
			Middle	High	Middle	High	Middle	High
Family variables								
Education	Father	Low	1.0	1.0	1.0	1.0	1.0	1.0
		Middle	1.2 (1.1–1.3)**	1.7 (1.6–1.9)**	1.2 (1.1–1.3)**	1.5 (1.4–1.7)**	1.2 (1.1–1.3)**	1.5 (1.4–1.7)**
		High	1.4 (1.2–1.7)**	4.5 (3.7–5.4)**	1.2 (1.0–1.5)	2.7 (2.3–3.3)**	1.1 (.9–1.4)	2.6 (2.1–3.1)**
	Mother	Low	1.0	1.0	1.0	1.0	1.0	1.0
		Middle	1.3 (1.2–1.4)**	1.9 (1.8–2.1)**	1.2 (1.1–1.3)**	1.8 (1.6–1.9)**	1.3 (1.2–1.4)**	1.8 (1.6–2.0)**
		High	1.4 (1.1–1.8)*	3.6 (2.9–4.6)**	1.2 (1.0–1.5)	2.4 (1.9–3.0)**	1.3 (1.0–1.6)	2.5 (2.0–3.2)**
Dwelling ownership	Father	Rented	1.0	1.0	1.0	1.0	1.0	1.0
		Owner-occupied	1.4 (1.2–1.6)**	1.7 (1.5–2.0)**	1.4 (1.2–1.6)**	1.8 (1.6–2.2)**	1.4 (1.2–1.5)**	1.7 (1.5–2.0)**
	Mother	Rented	1.0	1.0	1.0	1.0	1.0	1.0
		Owner-occupied	1.6 (1.4–1.9)**	2.3 (2.0–2.7)**	1.5 (1.3–1.7)**	1.9 (1.6–2.2)**	1.3 (1.1–1.5)**	1.5 (1.3–1.7)**
	Maternal grand-parents	Rented	1.0	1.0	1.0	1.0	1.0	1.0
		Owner-occupied	1.3 (1.2–1.5)**	1.6 (1.4–1.8)**	1.3 (1.1–1.5)**	1.5 (1.3–1.8)**	1.3 (1.1–1.5)*	1.5 (1.3–1.8)**
		Unknown	1.0 (.9–1.2)	1.2 (1.0–1.4)	1.0 (.9–1.1)	1.1 (.9–1.3)	1.0 (.8–1.1)	1.0 (.9–1.2)
Employment status	Father	Unemployed	1.0	1.0	1.0	1.0	1.0	1.0
		Employed	1.2 (1.0–1.3)*	1.5 (1.4–1.8)**	1.1 (1.0–1.3)	1.4 (1.2–1.6)**	1.0 (.9–1.2)	1.2 (1.1–1.4)*
	Mother	Unemployed	1.0	1.0	1.0	1.0	1.0	1.0
		Employed	1.3 (1.2–1.5)**	1.6 (1.4–1.8)**	1.3 (1.2–1.4)**	1.4 (1.3–1.6)**	1.2 (1.1–1.4)**	1.4 (1.2–1.5)**
Adolescence variables								
School achievement		Low			1.0	1.0	1.0	1.0
		Average	–	–	2.8 (2.5–3.1)**	9.0 (8.0–10.1)**	2.6 (2.3–2.9)**	7.9 (7.0–8.9)**
		High			5.1 (4.1–6.4)**	38.9 (31.1–48.6)**	4.6 (3.7–5.8)**	32.4 (25.9–40.6)**
Reserve capacity								
Perceived health	Chronic disease	Yes					1.0	1.0
		No	–	–	–	–	1.3 (1.1–1.4)**	1.3 (1.2–1.5)**
	Perceived stress symptoms	4–8/week					1.0	1.0
		2–3/week					1.2 (1.1–1.4)*	1.5 (1.3-.1.7)**
		1/week	–	–	–	–	1.4 (1.3–1.7)**	1.6 (1.4–1.9)**
		None					1.6 (1.4–1.8)**	1.8 (1.5–2.0)**

Health-promoting behaviour	Physical activity	Does not exercise					1.0	1.0
		Occasional/low efficient exerciser					1.2 (1.0–1.3)*	1.4 (1.2–1.6)**
		Active efficient exerciser	–	–	–	–	1.2 (1.0–1.3)*	1.5 (1.4–1.8)**
		Very active efficient exerciser					1.2 (1.1–1.4)*	1.6 (1.4–1.8)**
	Regular tooth brushing	<1–5 times/week					1.0	1.0
		About once/day	–	–	–	–	1.5 (1.3–1.6)**	2.1 (1.9–2.4)**
		Several times/day					1.5 (1.4–1.7)**	2.5 (2.2–2.9)**
Social support	Nuclear family	No					1.0	1.0
		Yes	–	–	–	–	1.7 (1.5–1.8)**	2.3 (2.0–2.5)**

Note: Includes statistically significant variables from Table 2.
[a]Model 1: Family SES.
[b]Model 2: Family SES + school achievement.
[c]Model 3: Family SES + school achievement + reserve capacity variables.
*$p < .01$; **$p < .001$ – Significance levels.

was observed among those with low adult education level. No marked differences in distribution of family- and adolescent-related variables were found among those with middle adult education level.

The odds of getting either middle or high adult education relative to low education increased when parents and grandparents had middle or high education (Table 2). There was also higher likelihood of obtaining either middle or high adult education level compared to low when family members owned their dwellings and when parents were employed. Parental and grandparental socioeconomic circumstances were more strongly associated with a high adult education than middle education. Adolescents who were high achievers in school had markedly greater odds of obtaining a middle or high adult education level than a low one. In terms of reserve capacity, positive categories predicted higher likelihood of getting either middle or high education. Clear gradients existed in the associations of most variables within dimensions of perceived health and health-promoting behaviour with adult education level. In the social support dimension, family structure was strongly associated with both adult education levels while talking to father was weakly related to high adult education only.

In multivariate analyses, parental socioeconomic variables were found to be associated with adult education level. However, among grandparental variables, only maternal grandparents' dwelling ownership retained its statistically significant associations (Table 3, Model 1). The strength of the associations observed for family socioeconomic circumstances were similar to those found in the bivariate analyses but the odds ratios were attenuated. Family socioeconomic circumstances strongly predicted high adult education than a middle education. When school achievement was added (Model 2), the odds ratios for the associations of almost all socioeconomic predictors with high education level decreased distinctly but minimal or no changes were seen in the associations with middle education level. School achievement was independently and strongly associated with both middle and high education. When reserve capacity variables were added (Model 3), the odds ratios obtained for socioeconomic circumstances of the family did not vary considerably from those in Model 2 but there were marked reductions in the associations of both parents' employment status and dwelling ownership with high adult education level. The odds ratios for school achievement also decreased but this remained the strongest predictor of adult education level. Independent associations of reserve capacity variables with adult education level were also found, with clear gradients for perceived stress symptoms and health-promoting behavioural factors. As regards social support, only family structure was related to adult education level. The final model showed that one's family socioeconomic circumstances significantly predicted one's adult education level but both school achievement and reserve capacity tended to decrease their effects.

Excluding unknown grandparents

Multivariate analyses excluding data from those with unknown grandparents showed slightly increased associations between some of the predictors (parents' education, school achievement and perceived stress symptoms in the perceived health dimension) and adult education level (Appendix 1). On the other hand, father's employment status and chronic disease in the perceived health dimension lost their statistically significant associations with the outcome. Overall results, however, showed the same directions and magnitude of associations as the analyses which included data from this group.

Discussion

Main findings of this study

The socioeconomic circumstances of parents and grandparents directly predicted adult education level. School achievement and reserve capacity dimensions of perceived health, health-promoting behaviour and social support in adolescence also positively and independently predicted adult education. Moreover, these tended to decrease the effect of family socioeconomic circumstances on educational level. Using polytomous categories for the outcome allowed us to disentangle the effects of the

predictors on different adult education levels. Results showed that all predictors were more strongly related with high than middle education.

Family socioeconomic circumstances

Consistent with previous research, our study found that family socioeconomic circumstances are positively associated with adult education level (Brekke, 2015; Fergusson et al., 2008; Koivusilta et al., 2013; Merritt & Buboltz, 2015; Slominski et al., 2011). We also provide evidence about the persistence of grandparents' effect on grandchildren's later educational outcomes, elucidating the origin of socioeconomic inequalities. Several mechanisms have been proposed for these associations. According to the Family Investment Model (FIM), greater SES implies greater parental material investments through financial transfers for tuition or maintenance during education (Albertini & Radl, 2012; Conger et al., 2010; Martin et al., 2010), primarily to prevent downward social mobility of children (Albertini & Radl, 2012). Likewise, wealthy grandparents might help finance their grandchildren's education through such monetary transfers (Chan & Boliver, 2013). High SES families value education more and have higher educational aspirations for their children compared to low SES families (Albertini & Radl, 2012; Fergusson et al., 2008; Martin et al., 2010). Conversely, low SES families are more likely exposed to stressful events such as unemployment which hinder their access to economic resources and limit their children's educational achievements (Fergusson et al., 2008).

Varying socioeconomic backgrounds also lead to different parenting practices, values and priorities which affect developmental and educational outcomes of children (Conger et al., 2010; Martin et al., 2010). Lower SES in childhood and adolescence were found to be associated with greater problem behaviours (Martin et al., 2010), probably due to poor quality of parenting which affect children's cognitive development and educational performance (Astone & McLanahan, 1991; Bird, 2007).

Adolescent-related predictors

Other than family SES, our results showed similar evidence with literature that school achievement was a strong predictor of adult education level (Brekke, 2015; Koivusilta et al., 2013; Slominski et al., 2011). Academic achievement implies academic ability and attachment level to school (Astone & McLanahan, 1991). During adolescence, school achievement likely influences enrolment in higher education (Brekke, 2015; Koivusilta et al., 2013). Thus, high achievers have been found to complete more years of schooling (Slominski et al., 2011).

Current research suggests that psychosocial resources in early childhood influence socioeconomic trajectories (Conger et al., 2010; Kroenke, 2008). However, there is limited evidence on psychosocial resources as a possible pathway to educational outcomes as these are more commonly considered in SES-health relationships. Moreover, there is a broad spectrum of psychosocial characteristics but to-date, few were studied and found to be associated with educational success: greater optimism, satisfaction (Boehm, Chen, Williams, Ryff, & Kubzansky, 2015), locus of control (Murasko, 2007) and self-efficacy (Merritt & Buboltz, 2015). We covered a different set of resources, including both psychosocial and behavioural factors, which were independently and positively associated with adult education level. Our findings enhanced available literature on reserve capacity and showed that good perceived health, health-promoting behaviour and social support protect adolescents from having a low adult education level. We surmise that these factors influence educational inequalities probably through the same mechanisms by which the reserve capacity framework causes SES-health related disparities (Gallo et al., 2009; Matthews et al., 2010). In other words, individuals with high reserve capacity are able to manage stressful school environments and meet academic demands, building competencies and skills necessary to pursue higher education (Matthews et al., 2010).

Although our findings did not show statistically significant associations between social support from friends and adult education level, related literature pointed to the existence of peer effects on education. Essentially, supportive and caring friendships positively influence school adjustment and

academic motivations (Nelson & DeBacker, 2008) while having academically weak peers tend to reduce one's academic performance (Winston & Zimmerman, 2004).

Limitations of this study

We note some limitations of our study. First, since the study was not initially conceptualized to measure reserve capacity, we used best available proxy measures. Despite this, our indicators measured important aspects of this multidimensional concept (Matthews & Gallo, 2011) but more research is needed to validate our findings. Second, almost half of the grandparents' data on socioeconomic circumstances were not available in the database of Statistics Finland. In order to preserve a robust sample size, we considered these groups as separate category and included in our analyses. Further analyses showed that if we had excluded these groups, we would have obtained similar results, albeit, some of the associations would slightly be overestimated (Appendix 1). Last, we acknowledge that other predictors of adult education level such as the school environment (Ryan & Patrick, 2001) and associated costs of continuing higher education and educational aspirations (Becker & Hecken, 2009) were unmeasured in our study. Future research should also try to account for the effect of these factors or assess other factors among those with preference for middle education instead of higher education.

Conclusions

Our study highlights the role of family socioeconomic circumstances in attaining high adult education and contributes to further understanding of the interplay between familial and personal factors in adolescence. Indeed, family socioeconomic circumstances, including those of grandparents, produced a dynamic effect in adolescence and influenced educational outcomes. However, since these associations were mediated by school achievement and reserve capacity in adolescence, it seemed that these personal predictors play more important roles in higher educational attainment (Koivusilta et al., 2013; Murasko, 2007; Slominski et al., 2011). Our findings suggest that formulating interventions which build reserve capacity and improve school performance, especially among adolescents from families with disadvantaged socioeconomic backgrounds, could likely reduce educational inequalities.

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References

Adler, N. E., & Newman, K. (2002). Socioeconomic disparities in health: Pathways And policies. *Health Affairs (Project Hope), 21*(2), 60–76.

Albertini, M., & Radl, J. (2012). Intergenerational transfers and social class: Inter-vivos transfers as means of status reproduction?*. *Acta Sociologica, 55*(2), 107–123. doi:10.1177/0001699311431596

Astone, N. M., & McLanahan, S. S. (1991). Family structure, parental practices and high school completion. *American Sociological Review, 56*(3), 309–320. doi:10.2307/2096106

Becker, R., & Hecken, A. E. (2009). Higher education or vocational training? An empirical test of the rational action model of educational choices suggested by Breen and Goldthorpe and Esser. *Acta Sociologica, 52*(1), 25–45.

Bird, K. (2007). *The intergenerational transmission of poverty: An overview* (Chronic Poverty Research Centre Working Paper No. 99). doi:10.2139/ssrn.1629262

Boehm, J. K., Chen, Y., Williams, D. R., Ryff, C., & Kubzansky, L. D. (2015). Unequally distributed psychological assets: Are there social disparities in optimism, life satisfaction, and positive affect? *PLoS ONE, 10*(2), e0118066. doi:10.1371/journal.pone.0118066

Brekke, I. (2015). Health and educational success in adolescents: A longitudinal study. *BMC Public Health, 15*, 619. doi:10.1186/s12889-015-1966-0

Chan, T. W., & Boliver, V. (2013). The grandparents effect in social mobility: Evidence from British birth cohort studies. *American Sociological Review, 78*(4), 662–678. doi:10.1177/0003122413489130

Cinar, A. B., Tseveenjav, B., & Murtomaa, H. (2009). Oral health-related self-efficacy beliefs and toothbrushing: Finnish and Turkish pre-adolescents' and their mothers' responses. *Oral Health & Preventive Dentistry, 7*(2), 173–181.

Conger, R. D., Conger, K. J., & Martin, M. J. (2010). Socioeconomic status, family processes, and individual development. *Journal of Marriage and Family, 72*(3), 685–704. doi:10.1111/j.1741-3737.2010.00725.x

Entwisle, D., Alexander, K., & Olson, L. (2005). First grade and educational attainment by age 22: A new story. *American Journal of Sociology, 110*(5), 1458–1502. doi:10.1086/428444

Erola, J., & Moisio, P. (2007). Social mobility over three generations in Finland, 1950–2000. *European Sociological Review, 23*(2), 169–183. doi:10.1093/esr/jcl027

Fergusson, D. M., Horwood, L. J., & Boden, J. M. (2008). The transmission of social inequality: Examination of the linkages between family socioeconomic status in childhood and educational achievement in young adulthood. *Research in Social Stratification and Mobility, 26*(3), 277–295. doi:10.1016/j.rssm.2008.05.001

Freudenberg, N., & Ruglis, J. (2007). Reframing school dropout as a public health issue. *Preventing Chronic Disease, 4*(4). Retrieved June 9, 2016, from http://www.cdc.gov/pcd/issues/2007/oct/07_0063.htm

Gallo, L. C., Espinosa de los Monteros, K., & Shivpuri, S. (2009). Socioeconomic status and health: What is the role of reserve capacity? *Current Directions in Psychological Science, 18*(5), 269–274.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129*(1), 10–51.

Koivusilta, L., West, P., Saaristo, V. M. A., Nummi, T., & Rimpelä, A. (2013). From childhood socio-economic position to adult educational level – Do health behaviours in adolescence matter? A longitudinal study *BMC Public Health, 13*(1), 711. doi:10.1186/1471-2458-13-711

Kroenke, C. (2008). Socioeconomic status and health: Youth development and neomaterialist and psychosocial mechanisms. *Social Science & Medicine, 66*(1), 31–42. doi:10.1016/j.socscimed.2007.07.018

Kuh, D., Ben-Shlomo, Y., Lynch, J., Hallqvist, J., & Power, C. (2003). Life course epidemiology. *Journal of Epidemiology and Community Health, 57*(10), 778–783. doi:10.1136/jech.57.10.778

Liu, H., & Hummer, R. A. (2008). Are educational differences in U.S. self-rated health increasing?: An examination by gender and race. *Social Science & Medicine, 67*(11), 1898–1906. doi:10.1016/j.socscimed.2008.09.021

Mackenbach, J. P., Kulhanova, I., Bopp, M., Deboosere, P., Eikemo, T. A., Hoffmann, R., … EURO-GBD-SE Consortium. (2015). Variations in the relation between education and cause-specific mortality in 19 European populations: A test of the "fundamental causes" theory of social inequalities in health. *Social Science & Medicine, 127*, 51–62. doi:10.1016/j.socscimed.2014.05.021

Martin, M. J., Conger, R. D., Schofield, T. J., Dogan, S. J., Widaman, K. F., Donnellan, M. B., & Neppl, T. K. (2010). Evaluation of the interactionist model of socioeconomic status and problem behavior: A developmental cascade across generations. *Development and Psychopathology, 22*(3), 695–713. doi:10.1017/S0954579410000374

Matthews, K. A., & Gallo, L. C. (2011). Psychological perspectives on pathways linking socioeconomic status and physical health. *Annual Review of Psychology, 62*, 501–530. doi:10.1146/annurev.psych.031809.130711

Matthews, K. A., Gallo, L. C., & Taylor, S. E. (2010). Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. *Annals of the New York Academy of Sciences, 1186*, 146–173. doi:10.1111/j.1749-6632.2009.05332.x

Merritt, D. L., & Buboltz, W. (2015). Academic success in college: Socioeconomic status and parental influence as predictors of outcome. *Open Journal of Social Sciences, 3*, 127–135. doi:10.4236/jss.2015.35018

Møllegaard, S., & Jæger, M. M. (2015). The effect of grandparents' economic, cultural, and social capital on grandchildren's educational success. *Research in Social Stratification and Mobility, 42*, 11–19. doi:10.1016/j.rssm.2015.06.004

Murasko, J. E. (2007). A lifecourse study on education and health: The relationship between childhood psychosocial resources and outcomes in adolescence and young adulthood. *Social Science Research, 36*(4), 1348–1370. doi:10.1016/j.ssresearch.2007.01.001

Nelson, R. M., & DeBacker, T. K. (2008). Achievement motivation in adolescents: The role of peer climate and best friends. *The Journal of Experimental Education, 76*(2), 170–189.

Pakpour, A. H., & Sniehotta, F. F. (2012). Perceived behavioural control and coping planning predict dental brushing behaviour among Iranian adolescents. *Journal of Clinical Periodontology, 39*(2), 132–137. doi:10.1111/j.1600-051X.2011.01826.x

Robbins, L. B., Pender, N. J., Ronis, D. L., Kazanis, A. S., & Pis, M. B. (2004). Physical activity, self-efficacy, and perceived exertion among adolescents. *Research in Nursing & Health, 27*(6), 435–446. doi:10.1002/nur.20042

Ryan, A. M., & Patrick, H. (2001). The classroom social environment and changes in adolescents' motivation and engagement during middle school. *American Educational Research Journal, 38*(2), 437–460. doi:10.3102/00028312038002437

Slominski, L., Sameroff, A., Rosenblum, K., & Kasser, T. (2011). Longitudinal predictors of adult socioeconomic attainment: The roles of socioeconomic status, academic competence, and mental health. *Development and Psychopathology, 23*(1), 315–324. doi:10.1017/S0954579410000829

Spittel, M. L., Riley, W. T., & Kaplan, R. M. (2015). Educational attainment and life expectancy: A perspective from the NIH office of behavioral and social sciences research. *Social Science & Medicine, 127*(1982), 203–205. doi:10.1016/j.socscimed.2014.11.017

United Nations. (n.d.) Sustainable development goals. Goal 4: Ensure inclusive and quality education for all and promote lifelong learning. Retrieved from http://www.un.org/sustainabledevelopment/education/

Winston, G., & Zimmerman, D. (2004). Peer effects in higher education. In C. Hoxby (Ed.), *College choices: The economics of where to go, when to go, and how to pay for it* (pp. 395–424). Chicago, IL: University of Chicago Press.

Appendix 1. Comparison of final model on associations with education level in adulthood (using low education as reference category), adjusting for sex and age at end of follow-up between population with unknown GP data and without unknown GP data.

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence			Final model[a] $N = 36,517$		Final model[a] $N = 15,328$ (unknown GP data excluded in analysis)	
			Odds ratios, 95% confidence intervals		Odds ratios, 95% confidence intervals	
			Middle	Middle	Middle	High
Family variables						
Education	Father	Low	1.0	1.0	1.0	1.0
		Middle	1.2 (1.1–1.3)**	1.5 (1.4–1.7)**	1.3 (1.1–1.5)**	1.7 (1.4–1.9)**
		High	1.1 (.9–1.4)	2.6 (2.1–3.1)**	1.2 (.9–1.6)	3.1 (2.2–4.2)**
	Mother	Low	1.0	1.0	1.0	1.0
		Middle	1.3 (1.2–1.4)**	1.8 (1.6–2.0)**	1.2 (1.1–1.4)*	1.8 (1.6–2.2)**
		High	1.3 (1.0–1.6)	2.5 (2.0–3.2)**	1.8 (1.2–2.8)*	3.5 (2.3–5.4)**
Dwelling ownership	Father	Rented	1.0	1.0	1.0	1.0
		Owner-occupied	1.4 (1.2–1.5)**	1.7 (1.5–2.0)**	1.4 (1.1–1.6)*	1.7 (1.4–2.2)**
	Mother	Rented	1.0	1.0	1.0	1.0
		Owner-occupied	1.3 (1.1–1.5)**	1.5 (1.3–1.7)**	1.3 (1.1–1.6)*	1.6 (1.2–2.0)**
	Maternal grandparents	Rented	1.0	1.0	1.0	1.0
		Owner-occupied	1.3 (1.1–1.5)*	1.5 (1.3–1.8)**	1.2 (1.0–1.4)	1.4 (1.1–1.7)*
		Unknown	1.0 (.8–1.1)	1.0 (.9–1.2)	–	–
Employment status	Father	Unemployed	1.0	1.0		
		Employed	1.0 (.9–1.2)	1.2 (1.1–1.4)*	–	–
	Mother	Unemployed	1.0	1.0	1.0	1.0
		Employed	1.2 (1.1–1.4)**	1.4 (1.2–1.5)**	1.2 (1.0–1.4)	1.3 (1.1–1.6)*
Adolescence variables						
School achievement		Low	1.0	1.0	1.0	1.0
		Average	2.6 (2.3–2.9)**	7.9 (7.0–8.9)**	2.6 (2.1–3.1)**	7.6 (6.2–9.2)**
		High	4.6 (3.7–5.8)**	32.4 (25.9–40.6)**	6.4 (4.1–10.0)**	42.0 (26.9–65.4)**
Reserve capacity						
Perceived health	Chronic disease	Yes	1.0	1.0		
		No	1.3 (1.1–1.4)**	1.3 (1.2–1.5)**	–	–
	Perceived stress symptoms	4–8/week	1.0	1.0	1.0	1.0
		2–3/week	1.2 (1.1–1.4)*	1.5 (1.3-.1.7)**	1.5 (1.2–1.9)**	2.2 (1.7–2.8)**
		1/week	1.4 (1.3–1.7)**	1.6 (1.4–1.9)**	1.5 (1.2–1.9)**	2.1 (1.7–2.7)**
		None	1.6 (1.4–1.8)**	1.8 (1.5–2.0)**	1.8 (1.5–2.2)**	2.7 (2.2–3.4)**

(*Continued*)

Appendix 1. (*Continued*).

Family socioeconomic circumstances, school achievement and reserve capacity in adolescence			Final model[a] $N = 36,517$		Final model[a] $N = 15,328$ (unknown GP data excluded in analysis)	
			Odds ratios, 95% confidence intervals		Odds ratios, 95% confidence intervals	
			Middle	Middle	Middle	High
Health-promoting behaviour	Physical activity	Does not exercise	1.0	1.0	1.0	1.0
		Occasional/low efficient exerciser	1.2 (1.0–1.3)*	1.4 (1.2–1.6)**	1.1 (.9–1.3)	1.2 (1.0–1.5)
		Active efficient exerciser	1.2 (1.0–1.3)*	1.5 (1.4–1.8)**	1.0 (.9–1.2)	1.3 (1.1–1.6)*
		Very active efficient exerciser	1.2 (1.1–1.4)*	1.6 (1.4–1.8)**	1.1 (.9–1.4)	1.5 (1.2–1.9)**
	Regular tooth brushing	<1–5 times/week	1.0	1.0	1.0	1.0
		About once/day	1.5 (1.3–1.6)**	2.1 (1.9–2.4)**	1.4 (1.2–1.6)**	2.1 (1.7–2.5)**
		Several times/day	1.5 (1.4–1.7)**	2.5 (2.2–2.9)**	1.5 (1.2–1.8)**	2.6 (2.1–3.2)**
Social support	Nuclear family	No	1.0	1.0	1.0	1.0
		Yes	1.7 (1.5–1.8)**	2.3 (2.0–2.5)**	1.8 (1.6–2.1)**	2.6 (2.3–3.1)**

Note: Includes statistically significant variables from Table 2.
[a]Final Model: Family SES + school achievement + reserve capacity variables.
*$p < .01$; **$p < .001$ – Significance levels.

PUBLICATION III

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

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

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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: To study whether family SES affects 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 from 1985 to 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 to the model.

Conclusions: The results show that family SES affects the child's adult education level through psychosocial and biobehavioural pathways, but the biological pathway is mediated by school achievement.

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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, as well as school, peer and other environmental influences, shape socio-emotional development and lead to formation and adoption of new behaviours (Viner et al. 2012), consequently affecting 'successful' transitions into adulthood (Johnson et al. 2011). Hence, intermediate pathways from childhood exposures to educational trajectories may be elucidated in adolescence.

In early life, the socioeconomic status (SES) of the family is an important exposure 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. 2018). 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, higher SES families tend to invest more in the health and education of their children than lower SES families do (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 effects likely 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), risky health behaviours (Koivusilta and Rimpelä

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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. 2008) 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 an increase in body fat (de Muinich Keizer 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 characterised by family conflict (Bleil et al. 2013) and stressful life events (Sun et al. 2017), for example, father absenteeism, divorce and single parent families (Bellis et al. 2006) is likewise linked to altered pubertal timing. Notably, research has documented mixed findings of socioeconomic inequalities in timing of puberty (de Muinich Keizer 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 Muinich Keizer and Mul 2001; Parent et al. 2003), possibly due to improved childhood health status (de Muinich Keizer and Mul 2001; Bellis et al. 2006) and nutrition (Parent et al. 2003; Bellis et al. 2006; Kyweluk et al. 2018). 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) due to environmental stress, which hastens reproductive maturation (Obeidallah et al. 2000; James-Todd et al. 2010; Xu et al. 2018).

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 hypothesised 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 whether pubertal timing and reserve capacity are such pathways through which SES influences educational trajectories. We added health-promoting behaviours, namely tooth brushing and physical activity, to the reserve capacity framework, 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. In this study, reserve capacity covers three dimensions, namely: perceived health, health-promoting behaviour and social support; with each dimension shown to independently predict adult education (Acacio-Claro et al. 2018). 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. 2018). 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 directly and indirectly (i.e. mediated by any of the adolescent pathways). 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 recognised important life stage transition (Viner et al. 2012).

Subjects 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

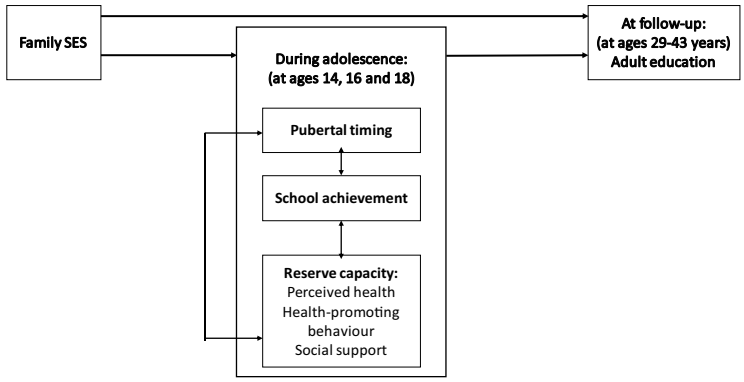

Figure 1. Conceptual model for the relationship of family SES with adult education level through adolescent pathways (biological, reserve capacity and school achievement).

1966 and 1980 were drawn for each study year from the Population Register Centre. Even though the AHLS has been 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. The 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 29–43 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. The Joint Commission on Ethics of the University of Turku and the Turku University Hospital 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 highest educational level attained by the adolescent. The exact degree codes according to the Finnish Standard Classification of Education were 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 categorised in the same way as that of adolescents' education. 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 were used. The minimum age of both 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 the 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), and 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), and 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) categorised as no symptoms, one symptom/week, 2–3/week, 4–8/week; and, self-rated health categorised 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 using 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 categorised based on self-assessment of their school performance as having: highest, 2nd highest, 2nd lowest or lowest academic achievement. The 14-year-old respondents (in comprehensive schools) were asked to assess whether their end-of-term school report was much better (highest), slightly better (2nd highest), average (2nd 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); 2nd highest (in vocational school with better performance or academic upper secondary school with average performance); 2nd 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. 2007). 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%). The majority of adolescents with low-to-middle adult education had parents with similarly attained education. Among those with available data, the average age of pubertal onset for boys was 13.1 ± 1.3 years, while for girls it was 12.6 ± 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 degrees of relationship with the latent variable. Among the nine variables, perceived stress symptoms and self-rated health, both of which are included in the perceived health dimension, contributed most to the measurement of the latent reserve capacity in both boys and

Table 1. Characteristics of participants according to 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	
	n	%	*n*	%	*n*	%	*n*	%
Age at baseline (years)								
14	4,182	34.0	1828	34.9	3,624	30.1	2951	35.6
16	4,412	35.9	1873	35.8	4,325	35.8	2972	35.9
18	3,701	30.1	1535	29.3	4,107	34.1	2366	28.5
Pubertal timing								
Early	2,731	22.2	1211	23.1	1,684	14.0	1213	14.6
Average	4,884	39.7	2449	46.8	7,709	63.9	5327	64.3
Late	3,067	25.0	1127	21.5	2,565	21.3	1714	20.7
No data	1,613	13.1	449	8.6	98	0.8	35	0.4
Parents' education								
High	1,227	10.0	1659	31.7	1,011	8.4	2178	26.3
Low/Middle	11,063	90.0	3577	68.3	11,039	91.6	6108	73.7
No data	5	0.0	0	0.0	6	0.0	3	0.0
Reserve capacity								
Perceived health dimension								
Chronic disease								
No	11,194	91.0	4796	91.6	10,759	89.2	7521	90.7
Yes	1,101	9.0	440	8.4	1,297	10.8	768	9.3
Perceived stress symptoms								
None	6,221	50.6	2647	50.6	3,636	30.2	2724	32.9
1/week	2,576	21.0	1119	21.4	2,657	22.0	1906	23.0
2–3/week	2,435	19.8	1117	21.3	3,535	29.3	2426	29.2
4–8/week	1,063	8.6	353	6.7	2,228	18.5	1233	14.9
Self-rated health								
Very good	4,502	36.6	2061	39.4	2,882	23.9	2525	30.5
Average/good	7,511	61.1	3080	58.8	8,833	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
Health-promoting behaviour dimension								
Physical activity								
Very active efficient exerciser	2,938	23.9	1677	32.0	1,824	15.1	1805	21.8
Active efficient exerciser	3,554	28.9	1735	33.2	3,242	26.9	2740	33.1
Occasional/low efficient exerciser	3,020	24.6	1094	20.9	3,966	32.9	2513	30.3
Does not exercise	2,740	22.3	719	13.7	3,000	24.9	1219	14.7
No data	43	0.3	11	0.2	24	0.2	12	0.1
Regular tooth brushing								
Several times/day	2,101	17.1	1584	30.2	5,644	46.8	4601	55.5
About once/day	5,967	48.5	2794	53.4	5,358	44.4	3309	39.9
About 1–5 times/week or less	4,151	33.8	846	16.2	1,031	8.6	360	4.4
No data	76	0.6	12	0.2	23	0.2	19	0.2
Social support dimension								
Nuclear family (with both parents)								
Yes	9,268	75.4	4471	85.4	8,577	71.1	6838	82.5
No	2,937	23.9	748	14.3	3,406	28.3	1419	17.1
No data	90	0.7	17	0.3	73	0.6	32	0.4
Talking about issues to father								
Easy	6,375	51.8	2763	52.8	4,003	33.2	3026	36.5
Difficult	3,762	30.6	1780	34.0	4,477	37.1	3435	41.4
Very difficult/No father	1,794	14.6	613	11.7	3,326	27.6	1762	21.3
No data	364	3.0	80	1.5	250	2.1	66	0.8
Talking about issues to mother								
Easy	8,454	68.7	3692	70.5	8,593	71.3	5984	72.2
Difficult	2,875	23.4	1246	23.8	2,622	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
Talking about issues to friends								
Easy	9,432	76.7	3945	75.4	10,392	90.7	7540	91.0
Difficult	2,093	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
School achievement								
Highest	1,026	8.3	1972	37.6	1,539	12.8	3611	43.6
2nd highest	2,987	24.3	2046	39.1	3,718	30.8	3204	38.6
2nd lowest	5,081	41.3	1014	19.4	4,453	36.9	1231	14.8
Lowest	3,009	24.5	182	3.5	2,212	18.4	221	2.7
No data	192	1.6	22	0.4	134	1.1	22	0.3

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 hypothesised 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 is also illustrated in the bottom parts of Figures 2 and 3.

Table 2. Results from confirmatory factor analyses (CFA) of reserve capacity model regressed on nine observed variables presented as standardised (β) coefficients.

	Boys		Girls	
	β	*p*-value	β	*p*-value
Observed variable				
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				
Perceived health				
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
Health-promoting behaviour				
Physical activity with				
Regular tooth brushing	0.12	< 0.001	0.10	< 0.001
Social support				
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	0.430
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
Fit indices				
RMSEA	0.04		0.03	
CFI	0.97		0.97	

SEM analyses

To disentangle the influence of the biological pathway from those of other intermediate pathways, we assessed how effects of puberty on adult education vary when only family SES was considered (model 1), then reserve capacity (model 2) and school achievement (model 3) were sequentially added (Table 3). Results showed that delayed pubertal timing increased the probability of low adult education in boys (models 1 and 2), but lost statistical significance once the school achievement pathway was included. On the other hand, family SES consistently predicted the probability of adult education, regardless of adolescent pathways added into the models in both boys and girls.

Model 3 is referred to as the full model and is illustrated 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 the same dimension of reserve capacity, were not shown.

Detailed results from SEM analyses of the full model 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 (Figures 2 and 3) fit the data well based on the presented fit indices. The hypothesised pathways are described further 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

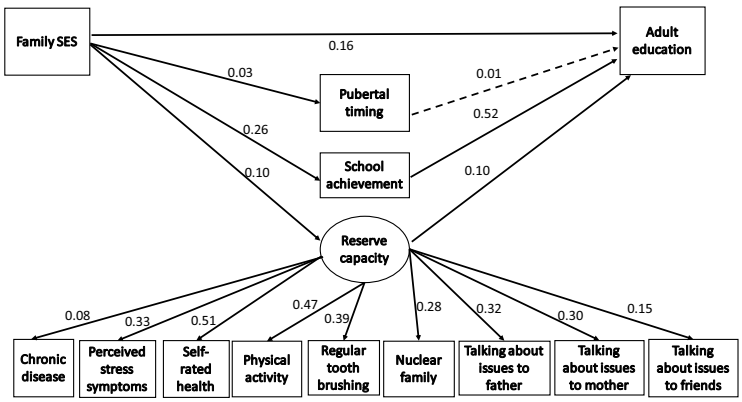

Figure 2. Boys: Structural equation model depicting relationships among family socioeconomic status (SES), pubertal timing, school achievement and reserve capacity in adolescence and adult education level (RMSEA = 0.05; CFI = 0.90). The values along the paths are standardised regression coefficients. Solid lines indicate statistically significant paths ($p < 0.001$).

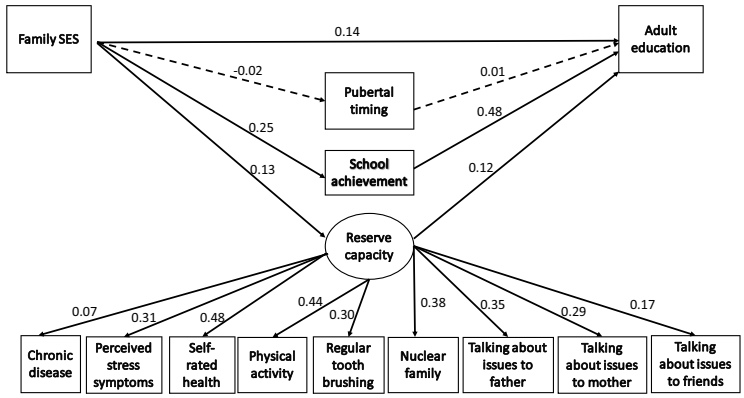

Figure 3. Girls: Structural equation model depicting relationships among family socioeconomic status (SES), pubertal timing, school achievement and reserve capacity in adolescence and adult education level (RMSEA = 0.04; CFI = 0.91). The values along the paths are standardised regression coefficients. Solid lines indicate statistically significant paths ($p < 0.001$).

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

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

Note: All models were adjusted for ages at baseline and follow-up.
*Statistically significant at $p < 0.5$.
[a]Model with family SES and puberty.
[b]Model 1 plus reserve capacity.
[c]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 standardised (β) coefficients.

Indirect effect of family SES through	Boys	Girls
Pubertal timing	0.00	0.00
School achievement	0.14*	0.12*
Reserve capacity	0.01*	0.02*
Covariance between pathways	**Boys**	**Girls**
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*

* Statistically significant at $p < 0.001$.

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 (β = –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. While, the paths from pubertal timing to adult education were not statistically significant, a positive coefficient ($\beta = 0.01$) indicated 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 directly and 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 a positive relationship with school achievement. In our study, this means that delayed pubertal timing was related to better reserve capacity in both boys and girls, but lower school achievement in boys. On the other hand, a weak reserve capacity was related to low school achievement.

Discussion

Summary and interpretation of results

We investigated the relationships among 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 the relationship between family SES and adult education. 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 inter-related, 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, directly and indirectly, via pathways of reserve capacity and school achievement. The significant effect of SES on 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 the economic inequality gap is narrower than in many 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 and 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 utilised 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 showed, 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 et al. (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 Muinich Keizer and Mul 2001; Parent et al. 2003). Living in low socioeconomic conditions might delay puberty because of a higher likelihood of malnutrition, acute or chronic illnesses and the 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 a higher body mass index (BMI) or being overweight (Downing and Bellis 2009; James-Todd et al. 2010) and experiencing stressful life events (James-Todd et al. 2010). However, 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 such as 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. 2018) 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 are congruent with previous evidence, albeit reserve capacity was measured using 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 inhibit them from developing and replenishing these resources 'to be kept in reserve'. While reserve capacity was initially conceptualised 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 requires 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 replicate 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 for girls showed no association between pubertal timing and adult education, other evidence has presented contrary findings (Hendrick et al. 2016; Gill et al. 2017). Previous research has shown that early maturing girls had a higher probability of being high school dropouts (Cavanagh et al. 2007; Hendrick et al. 2016) or having low-grade point averages (GPA) at the end of high school (Cavanagh et al. 2007). Gill et al. (2017) found that menarche occurring at later ages increased the schooling period. However, most studies have suggested that, beyond high school, the impact of early pubertal timing on educational outcomes ceases (Copeland et al. 2010; Hendrick et al. 2016). Still, as research on educational outcomes related to pubertal timing is 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. 2018), school achievement has the largest effect on adult education. This is to be expected, as good grades obtained in high school strongly predicted enrolment in higher education (Brekke 2015). In fact, Entwisle et al. (2005) demonstrated that academic performance as early as first grade influenced educational attainment. Our results also point 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 reduce human capital investments of parents for their children, including cognitive stimulation, thus affecting their cognitive development (Kroenke 2008; Conger et al., 2010).

Strengths and limitations of the study

Using large, nationwide samples with good response rates, a 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 behaviours. We needed to use proxy indicators, whereas related studies had used psychological scales or other structured tools, because reserve capacity was conceptualised at a much later time than when our surveys were conducted. Nevertheless, we have measured a valid construct as proven by the good fit indices obtained for this latent variable.

We have identified intermediate adolescent pathways (pubertal timing, reserve capacity and school achievement) which account for the relationship of family SES with adult education. Even though our models had good fit, we recognise that there are other structural and individual factors that have not been measured in our study which could 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 were not obtained from school-based or community-based surveys, so analysing those effects were beyond the scope of this study.

We acknowledge some methodological limitations related to one of the pathways and the outcome variable studied. 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 the 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. Still, the pubertal timing ages estimated in our study population closely resembled those described in other European countries which used more accurate staging methods for the same period (de Muinich Keizer and Mul 2001). For the outcome, we initially tried to use three categories of education (low, middle and high) where SEM results are ordered logistic regression coefficients. However, our current data did not support the proportional odds assumption required for the ordered three-category outcome. On the other hand, treating the categories as unordered or multinomial did not allow assessment of indirect effects, which is one of our main hypotheses. Thus, we decided to dichotomise adult education. Future research should assess if similar pathways operate for other categories of education such as middle education.

Generally our results, which expand on the work of earlier studies (Koivusilta and Rimpelä 2004, Acacio-Claro et al. 2018), have shown similar patterns, even with the different methodological techniques used (i.e. using a longer follow-

up period and different analytic procedures), thus adding to the robustness and reliability of our study.

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 statement

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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References

Acacio-Claro P, Doku DT, Koivusilta LK, Rimpelä AH. 2018. How socioeconomic circumstances, school achievement and reserve capacity in adolescence predict adult education level: a three-generation study in Finland. Int J Adolesc Youth. 23:382–397.

Aksglaede L, Olsen LW, Sørensen TIA, Juul A. 2008. Forty years trends in timing of pubertal growth spurt in 157,000 Danish school children. Plos One. 3:e2728.

Becker R, Hecken AE. 2009. Higher education or vocational training? An empirical test of the rational action model of educational choices suggested by Breen and Goldthorpe and Esser. Acta Sociol. 52:25–45.

Bellis MA, Downing J, Ashton JA. 2006. Adults at 12? Trends in puberty and their public health consequences. J Epidemiol Commun Health. 60:910–911.

Bleil ME, Adler NE, Appelhans BM, Gregorich SE, Sternfeld B, Cedars MI. 2013. Childhood adversity and pubertal timing: Understanding the origins of adulthood cardiovascular risk. Biol Psychol 93:213–219.

Brekke I. 2015. Health and educational success in adolescents: A longitudinal study. BMC Public Health 15:619.

Cavanagh SE, Riegle-Crumb C, Crosnoe R. 2007. Puberty and the education of Girls*. Soc Psychol Q. 70:186–198.

Cinar AB, Tseveenjav B, Murtomaa H. 2009. Oral health-related self-efficacy beliefs and toothbrushing: Finnish and Turkish pre-adolescents' and their mothers' responses. Oral Health Prev Dent. 7:173–181.

Conger RD, Conger KJ, Martin MJ. 2010. Socioeconomic status, family processes, and individual development. J Marriage Fam. 72:685–704.

Copeland W, Shanahan L, Miller S, Costello EJ, Angold A, Maughan B. 2010. Outcomes of early pubertal timing in young women: a prospective population-based study. Am J Psychiatry. 167:1218–1225.

de Muinich Keizer SM, Mul D. 2001. Trends in pubertal development in Europe. Hum Reprod Update 7:287–291.

Downing J, Bellis M. 2009. Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: A preliminary cross-sectional study. BMC Public Health 9:446.

Entwisle D, Alexander K, Olson L. 2005. First grade and educational attainment by age 22: a new story. American Journal of Sociology 110:1458–1502.

Euling SY, Herman-Giddens ME, Lee PA, Selevan SG, Juul A, Sorensen TI, Dunkel L, et al. 2008. Examination of US puberty-timing data from 1940 to 1994 for secular trends: panel findings. Pediatrics 121 Suppl 3: S172–S191.

Gallo LC, Matthews KA. 2003. Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? Psychol Bull 129:10–51.

Gallo LC, Espinosa de los Monteros K, Shivpuri S. 2009. Socioeconomic status and health: What is the role of reserve capacity? Curr Directions Psychol Sci 18:269–274.

Gill D, Del GM, Rawson TM, Sivakumaran P, Brown A, Sheehan NA, Minelli C. 2017. Age at menarche and time spent in education: a mendelian randomization study. Behav Genet 47:480–485.

Golub MS, Collman GW, Foster PMD, Kimmel CA, Rajpert-De Meyts E, Reiter EO, Sharpe RM, et al. 2008. Public health implications of altered puberty timing. Pediatrics 121:S218–S230.

Graber JA. 2013. Pubertal timing and the development of psychopathology in adolescence and beyond. Horm Behav 64:262–269.

Grace JB, Bollen KA. 2005. Interpreting the results from multiple regression and structural equation models. Bull Ecol Soc Am 86: 283–295.2.0.CO;2.

Hendrick CE, Cohen AK, Deardorff J, Cance JD. 2016. Biological and sociocultural factors during the school years predicting women's lifetime educational attainment. J Sch Health 86:215–224.

Hooper D, Coughlan J, Mullen MR. 2007. Structural equation modeling: guidelines for determining model fit. The Electronic Journal of Business Research Methods 6:53–60.

Jacobsen BK, Oda K, Knutsen SF, Fraser GE. 2009. Age at menarche, total mortality and mortality from ischaemic heart disease and stroke: the Adventist health study, 1976-88. Int J Epidemiol 38:245–252.

James-Todd T, Tehranifar P, Rich-Edwards J, Titievsky L, Terry MB. 2010. The impact of socioeconomic status across early life on age at menarche among a racially diverse population of girls. Ann Epidemiol 20: 836–842.

Johnson MK, Robert C, Elder GH. 2011. Insights on adolescence from a life course perspective. J Res Adolesc 21:273–280.

Keenan K, Culbert KM, Grimm KJ, Hipwell AE, Stepp SD. 2014. Timing and tempo: Exploring the complex association between pubertal development and depression in African American and European American girls. J Abnorm Psychol 123:725–736.

Koerselman K, Pekkarinen T. 2017. The timing of puberty and gender differences in educational achievement. Bonn, Germany: IZA Institute of Labor Economics initiated by Deutsche Post Foundation. Report nr IZA DP No. 10889.

Koivusilta L, Rimpelä A. 2004. Pubertal timing and educational careers: A longitudinal study. Ann Hum Biol 31:446–465.

Koivusilta LK, Rimpelä AH. 2006. Pubertal timing and health-related behaviours in adolescence - socio-economic outcomes in a follow-up study from Finland. Italian Journal of Public Health 3:41–52.

Koivusilta L, West P, Saaristo VMA, Nummi T, Rimpela A. 2013. From childhood socio-economic position to adult educational level - do health behaviours in adolescence matter? A longitudinal study. BMC Public Health 13:711.

Kroenke C. 2008. Socioeconomic status and health: Youth development and neomaterialist and psychosocial mechanisms. Soc Sci Med 66: 31–42.

Kyweluk MA, Georgiev AV, Borja JB, Gettler LT, Kuzawa CW. 2018. Menarcheal timing is accelerated by favorable nutrition but unrelated to developmental cues of mortality or familial instability in Cebu, Philippines. Evolution and Human Behavior 39:76–81.

Lakshman R, Forouhi NG, Sharp SJ, Luben R, Bingham SA, Khaw KT, Wareham NJ, Ong KK. 2009. Early age at menarche associated with cardiovascular disease and mortality. J Clin Endocrinol Metab. 94: 4953–4960.

Martin AJ, Steinbeck K. 2017. The role of puberty in students' academic motivation and achievement. Learn Individual Differences. 53:37–46.

Martin MJ, Conger RD, Schofield TJ, Dogan SJ, Widaman KF, Donnellan MB, Neppl TK. 2010. Evaluation of the interactionist model of socioeconomic status and problem behavior: a developmental cascade across generations. Dev Psychopathol. 22:695–713.

Matthews KA, Gallo LC. 2011. Psychological perspectives on pathways linking socioeconomic status and physical health. Annu Rev Psychol. 62:501–530.

Matthews KA, Gallo LC, Taylor SE. 2010. Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. Ann N Y Acad Sci. 1186:146–173.

Merritt DL, Buboltz W. 2015. Academic success in college: Socioeconomic status and parental influence as predictors of outcome. Open J Soc Sci. 03:127–135.

Muthén LK, Muthén BO. 1998-2012. Mplus User's guide. 7th ed. Los Angeles, CA.: Muthén & Muthén.

Obeidallah DA, Brennan RT, Brooks-Gunn J, Kindlon D, Earls F. 2000. Socioeconomic status, race, and girls' pubertal maturation: results from the project on human development in Chicago neighborhoods. J Res Adolesc. 10:443–464.

Organisation for Economic Cooperation and Development [OECD]. 2018. Equity in education: breaking down barriers to social mobility. Paris: PISA, OECD Publishing.

Pakpour AH, Sniehotta FF. 2012. Perceived behavioural control and coping planning predict dental brushing behaviour among Iranian adolescents. J Clin Periodontol. 39:132–137.

Parent A, Teilmann G, Juul A, Skakkebaek NE, Toppari J, Bourguignon J. 2003. The timing of normal puberty and the age limits of sexual precocity: Variations around the world, secular trends, and changes after migration. Endocr Rev. 24:668–693.

Robbins LB, Pender NJ, Ronis DL, Kazanis AS, Pis MB. 2004. Physical activity, self-efficacy, and perceived exertion among adolescents. Res Nurs Health 27:435–446.

Slominski L, Sameroff A, Rosenblum K, Kasser T. 2011. Longitudinal predictors of adult socioeconomic attainment: The roles of socioeconomic status, academic competence, and mental health. Dev Psychopathol 23:315–324.

Statistics Finland. Finnish Standard Classification of Education 2011 [Internet]; c2018. Available from: http://www.stat.fi/meta/luokitukset/koulutus/001-2011/index_en.html.

Sun Y, Mensah FK, Azzopardi P, Patton GC, Wake M. 2017. Childhood social disadvantage and pubertal timing: A national birth cohort from Australia. Pediatrics 139. http://doi.org/10.1542/peds.2016-4099

Viner RM, Ozer EM, Denny S, Marmot M, Resnick M, Fatusi A, Currie C. 2012. Adolescence and the social determinants of health. The Lancet 379:1641–1652.

Xu Y, Norton S, Rahman Q. 2018. Early life conditions, reproductive and sexuality-related life history outcomes among human males: A systematic review and meta-analysis. Evol Hum Behav 39:40–51.

Zhu J, Chan Y. 2017. Adult consequences of self-limited delayed puberty. Pediatrics 139:e20163177.