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Impact of childhood and adult socioeconomic position on change in functional aging

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DATA AVAILABILITY: Data from the Swedish Adoption/Twin Study of Aging are publicly at National Archive of Computerized Data on Aging (NACDA)

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ABSTRACT

Objectives: To examine lifecourse models by investigating the roles of childhood and adult socioeconomic position (SEP) in longitudinal changes in a functional aging index (FAI).

Methods: Up to 8 waves of testing, covering 25 years, were available from the Swedish Adoption Twin Study of Aging: N=654, intake age = 50-82. A two-slope latent growth curve model was applied to the data, and the impact of including childhood and adult SEP as covariates of the intercept (at age 70) and slopes (before and after age 70) was tested.

Results: Both childhood and adult SEP contributed to the best-fitting model. Childhood SEP was significantly associated with intercept and slope 1 (before age 70) of the latent growth curve model ($p < .05$). Association of adult SEP with slope 2 (after age 70) trended toward significance ($p < .10$). There was a significant interaction effect of childhood and adult SEP on the intercept ($p < .05$). As a result, intercept at age 70 was highest and change after age 70 was fastest for those whose SEP decreased from childhood to adulthood.

Conclusions: Both childhood and adult SEP impact change in functional abilities with age, supporting both critical period and social mobility models. The social environment is modifiable by policies at local, national, and international levels, and these policies need to recognize that early social disadvantage can have long-lasting health impacts.

Words = 227

Key words: socioeconomic position, functional abilities, longitudinal, social mobility

Public Significance: Economic disadvantage in both childhood and adulthood can impact the pace of physical aging. In fact, that the number of years lost to economic disadvantage may be equal to or greater than number of years lost due to major risk factors for chronic disease. Initiatives targeting health inequalities should be focused on interventions early in the life course.

Reducing socioeconomic position (SEP) inequalities in health outcomes is a primary goal of health policy (WHO, 1985). Evidence suggests, however, that income inequality has been increasing in recent years (Hoffmann, Lee, & Lemieux, 2020), as have inequalities in health and life expectancy (S. Lynch, 2003; Mirowsky & Ross, 2003; Pappas, Queen, Hadden, & Fisher, 1993; van Kippersluis, Van Ourti, O'Donnell, & van Doorslaer, 2009). SEP measured as social class and education has been identified as a fundamental cause of these health disparities (Phelan, Link, & Tehranifar, 2010). Different socioeconomic groups have different health behavioral patterns (Pampel, Krueger, & Denney, 2010), and experience different types of stressors. For example, people with low socioeconomic position can experience both the magnified stress of life events (e.g., sudden illness, job loss) and the chronic stress of poor working conditions and extensive financial strain (Baum, Garofalo, & Yali, 1999; Pearlin, 1989). Evidence suggests that people in low socioeconomic positions may be less able to take advantage of developing health information, due to limited access to the information and limited means to adapt to new information (Finkel & Ernst Bravell, 2020; Mirowsky & Ross, 2003). As a result of these factors, people with low SEP tend to have worse cognitive and physical function in late adulthood (Darin-Mattsson, Fors, & Kåreholt, 2017; Wolfova, Csajbok, Kagstrom, Kåreholt, & Cermakova, 2021).

A life-course approach is necessary for a full understanding of the impact of SEP on the aging process, as the influence of the environment can change and accumulate over the lifespan (Alwin & Wray, 2005; Corna, 2013; Luo & Waite, 2005; Lyu & Burr, 2016; Zimmer, Hanson, & Smith, 2016). Specifically, the life-course approach emphasizes that the timing of events (e.g., childhood vs. adulthood) can play a significant role in the ultimate impact of the experience (Elder, Johnson, & Crosnoe, 2003). Several models have been proposed to identify when in the lifespan critical events or experiences may produce long-term effects on outcome variables. The critical period model suggests that early-life disadvantage can set in motion biological processes

that may not be revealed until late adulthood (Ben-Shlomo & Kuh, 2002; Kuh & Ben-Shlomo, 2004). As a result, the model predicts that childhood measures of SEP will impact late-life health outcomes, even in the context of concurrent adult SEP. The accumulation of risks model also posits the continued impact of early life environment factors via consistent increases in their impact on health disparities across the lifespan (Ferraro & Shippee, 2009; Willson, Shuey, & Elder, 2007). Thus, both childhood and adult SEP would be expected to have an additive influence health outcomes. Social mobility models allow for the dynamic processes of change in SEP to alter the impact of early life experiences (Hallqvist, Lynch, Bartley, Lang, & Blane, 2004; Pudrovska & Anikputa, 2014; Zimmer et al., 2016). Consequently, moving from lower SEP in childhood to higher SEP in adulthood might moderate or even reverse the negative impact of early disadvantage. In contrast, decreasing SEP in mid- and late-life might outweigh the benefits of early life advantage. In other words, social mobility models predict that childhood and adult SEP interact in their contribution to late-life health outcomes.

Various approaches have been used to test life-course models of the impact of SEP at various points in the lifespan on health outcomes. Studies have focused on the relative impact of both childhood and adult SEP (Luo & Waite, 2005; Lyu & Burr, 2016; Yang, Schorpp, Boen, Johnson, & Harris, 2020; Zimmer et al., 2016) and the role of changes in SEP from childhood to adulthood and from early to later adulthood (Hallqvist et al., 2004; Poulton et al., 2002; Yang, Gerken, Schorpp, Boen, & Harris, 2017). Outcome variables have included self-reported health or functional limitations (Luo & Waite, 2005), mortality (Hart, Smith, & Blane, 1998; J. W. Lynch et al., 1994), measured cognition (Lyu & Burr, 2016), registry-based health data (Zimmer et al., 2016), and measured health variables such as inflammation or cardiorespiratory fitness (Poulton et al., 2002; Yang et al., 2017; Yang et al., 2020). As a result, outcomes have been mixed. Many studies report that both childhood and adult SEP are important for health outcomes (Hart et al.,

1998; Luo & Waite, 2005; Lyu & Burr, 2016; Zimmer et al., 2016), but other studies do not find evidence for a continuing role of childhood SEP (J. W. Lynch et al., 1994; Poulton et al., 2002). Other studies find that the impact of childhood SEP is mediated through adult SEP (Chapman, Fiscella, Duberstein, Coletta, & Kawachi, 2009; Lyu & Burr, 2016; Yang et al., 2017). Investigations of life-course trajectories in SEP have produced mixed results, suggesting complex temporal dynamics for the impact of socio-economic disadvantage on health outcomes (Poulton et al., 2002; Yang et al., 2017).

In contrast to approaches incorporating of life-course changes in SEP, most studies of the impact of SEP on health rely on cross-sectional information about the outcome variables, or short-term longitudinal designs (e.g., (Luo & Waite, 2005; Yang et al., 2017; Yang et al., 2020); research examining the relationship between childhood and adult SEP and *rate of change with age* in objectively measured health and functioning is relatively rare (Lyu & Burr, 2016). In linear latent growth curve analyses of change in body mass index over 20 years (Lee & Park, 2020) and functional limitations over 8 years (Haas, 2008), both childhood and adult SEP were associated with the intercept of the model, but only childhood SEP was associated with the rate of change with age. In contrast, a study with 12-years of follow-up data on cognition found no effect of childhood SEP, per se, on rates of changes, but did find that rates of cognitive decline were reduced among participants whose mothers had higher education (Zimmer et al., 2016). Longitudinal research indicates mixed results for individuals who experienced changes in SEP across the life course (Harber-Aschan et al., 2020; Landös et al., 2019; Torres, Rizzo, & Wong, 2018). Mixed SEP (Harber-Aschan et al., 2020) or changes from high to low SEP (or vice versa) from childhood to adulthood (Landös et al., 2019; Torres et al., 2018) were associated with worse or mixed aging trajectories in functional limitations and health.

The aim of the current analysis was to expand on previous work and investigate the

associations between SEP across the lifecourse and late-life functional abilities by applying latent growth curve models to longitudinal measures of functional aging over 25 years of follow-up, and to test the fit of three theoretical models of the potential pathways through which SEP has an effect on FAI in aging (e.g., accumulation of risks model, critical periods model, social mobility model). Based on previous research, we predict that both childhood and adult SEP will be associated with a latent growth curve model of changes with age in functional aging, providing support for the accumulation of risks model. We also predict that only childhood SEP will be associated with rates of change in functional aging, as predicted by the critical periods model. We will examine the interaction of childhood and adult SEP to investigate the social mobility model.

METHOD

Participants

Data came from the Swedish Adoption/Twin Study of Aging (SATSA; (Finkel & Pedersen, 2004). Recruitment and testing procedures have been described previously. In brief, starting in 1984, twins who reached age 50 were recruited from the population-based Swedish Twin Registry for in-person testing (IPT); new twins who reached age 50 were recruited at each IPT up through IPT5 (Lichtenstein et al., 2002). Up to 10 waves of IPT took place between 1984 and 2014 in locations convenient to participants, such as district nurses' offices, health-care schools, long-term care clinics, or at the participant's home. Intervals between IPTs averaged 3 years. Component measures of the Functional Aging Index were not available from IPT1; therefore, the sample was drawn from the 750 individuals who participated in IPT2 and after. All 4 component measures of function from at least one testing occasion were available for 654 individuals (87.2%). Sample characteristics are reported in Table 1. The sample ranged in age from 50 to 82 at intake and mean age at intake was 62.13 ($SD = 8.17$); 57.03% of the sample were women. All participants were of European ancestry. Mean number of waves of

participation was 4.03 ($SD = 2.39$). Three or more waves of data required to support growth curve modeling were available for 62.69% of the sample; 5 or more waves of data were available from 39.05% of the sample. Although 16.21% of the sample had only 1 wave of participation, they were included in analyses because growth curve models take missing data into account by giving more weight to individuals with the most time points. Longitudinal follow-up ranged from 0 to 27 years, with a mean of 15.25 years ($SD = 7.96$).

Measures

Socioeconomic Position (SEP). Questions about parental occupation and education included in the first survey mailed out to SATSA participants in 1984 were used as measures of childhood SEP. The same two scales were used to assess occupation and education for childhood and adult SEP: for childhood the items referred to parents, for adult SEP the items referred to the participant. If the participant was retired, then their primary lifetime occupation was coded. Education was scored on a 4-point scale: 1 (only compulsory education: 6 to 7 years for this age group), 2 (lower secondary or vocational), 3 (upper secondary), and 4 (university education). Occupation was translated to a 7-point scale: 1 = homemaker (at the time listed as 'housewife'), 2 = work with no special education, 3 = work with training, 4 = work with apprenticeship or considerable experience, 5 = work with vocational school or higher training, 6 = work with responsibility or academic studies (e.g., BA degree), 7 = work with considerable responsibility or higher academic degree (Lichtenstein, Harris, Pedersen, & McClearn, 1993). For both childhood and adult measures of SEP, relevant occupation and education variables were standardized and summed. For use in data analyses, the resulting variables were standardized and winsorized at 3 standard deviations to remove positive skew, descriptive statistics are reported in Table 1. Finally, median split was used to create high and low SEP groups for both childhood and adult SEP.

Functional Aging Index (FAI). The World Health Organization has championed an approach to aging research that focuses on healthy aging as a process of developing and maintaining functional abilities as opposed to simply the absence of disease (WHO, 2015). Functional abilities help to ensure quality of life and wellbeing in late adulthood (Rudnicka et al., 2020). Therefore, our outcome variable is an index of functional aging measured in-person that can complement existing measures of biological aging and frailty by focusing on functional capacity (Finkel, Sternäng, Jylhävä, Bai, & Pedersen, 2019). At each IPT, measures of functional aging were collected by research nurses: grip strength (best of 3 trials on each hand), pulmonary function (best peak expiratory flow (PEF) from two trials), and gait (time to walk 3 meters and return). A measure of sensory function combined self-report items about vision and hearing. Before calculation of FAI, grip strength was regression-corrected for sex and PEF was corrected for body volume through dividing it by the individual's squared height in meters (Sternäng, Palmer, Kabir, Hasan, & Wahlin, 2018). The four measures were standardized respectively and then summed to compose a general FAI (Finkel et al., 2019). Higher FAI indicates worse functioning; FAI is expected to increase with age. Descriptive statistics for FAI at intake are reported in Table 1.

Statistical Analysis

Due to the range in age at each wave, an age-based latent growth curve (LGC) model was used to estimate trajectories of change with age in FAI. The structural model can be considered as a multi-level random coefficients model (Bryk & Raudenbush, 1992; McArdle & Anderson, 1990). The model provides estimation of fixed effects, i.e., fixed population parameters as estimated by the average growth model of the entire sample, and random effects, i.e., inter-individual variability in intraindividual change in growth model parameters. The age basis serves as a marker for the age of the subject at each time of measurement, adjusted for the centering age. Therefore, age basis coefficients are defined as an individual's observed age at each measurement occasion minus the centering age (70 years).

Accelerating change with age in FAI is best captured using a two-slope model: slope 1 estimates the rate

of change up to age 70 and slope 2 estimates the rate of change after age 70 (Finkel et al., 2019). The random and fixed effects parameter estimates were obtained using PROC Mixed in SAS 9.4 and models were corrected for twinning by modeling both between and within pair variance in the random effects. Childhood and adult SEP were added to the LGC model as covariates of the intercept, slope 1, and slope 2. To test the effect of adding these covariates to the LGC models, four LGC models were compared: base model, adding only child SEP, adding only adult SEP, and adding both childhood and adult SEP and their interactions. Life-course models of timing of impact of SEP are tested in the fourth model: (a) if childhood SEP is a significant covariate in the context of adult SEP, then the critical periods model is supported, (b) if both childhood and adult SEP are significant covariates, then the accumulation of risks model is supported, and (c) if the interaction of childhood and adult SEP is significant, then the social mobility model is supported. Nested LGC models can be compared using a likelihood ratio test (LRT), which is the difference in the model fit statistic (log likelihood) for the two models, with degrees of freedom equal to the difference in parameters estimated. LGC model were fit using continuous child and adult SEP as covariates and using the dichotomized child and adult SEP variables. Results did not differ; results with dichotomized covariates are reported to support graphical presentation of results.

SATSA data have been made publicly available at the National Archive of Computerized Data on Aging (NACDA) and can be accessed at <https://www.icpsr.umich.edu/web/NACDA/studies/3843>

RESULTS

Results of fitting the two-slope LGC model to FAI are presented in Table 2. Given the sample size and amount of longitudinal data, 2421 data points were available for model fitting. Using the LRT to compare nested models indicated that adding only childhood SEP to the base model (model 2) did not result in a significant improvement in model fit (LRT = 2.10, df = 3, $p = 0.55$). Adding only adult SEP to the base model (model 3) did result in an improvement in model fit, as did adding both child and adult SEP and the interaction of child and adult SEP (model 4). Parameter estimates from model 4 are

presented in Table 3. Intercept, slope 1 (up to age 70) and slope 2 (after age 70) were all significantly different from zero. Slope 2 (9.66) was twice as large as slope 1 (4.37), indicating the rate of change in functioning doubles after age 70. Both childhood SEP and the interaction of childhood and adult SEP were significant covariates of the intercept. For example, the parameter estimate of -2.17 for the effect of child SEP on the intercept indicates that higher childhood SEP was associated with a 2 point reduction (on a T-score metric) in mean FAI at age 70 (lower scores on FAI indicate better functioning). Only childhood SEP was a significant covariate of slope 1: higher childhood SEP was associated with a reduced slope or slower rates of decline in FAI. Adult SEP covaried with slope 2, but the effect only trending towards significance at $p < .10$.

Childhood SEP did not differ between men and women ($t(652) = 1.52, p = .12$), although men had a significantly higher adult SEP than women ($t(652) = 3.51, p < .01$), as expected. The correlation between adult SEP and FAI at intake did not differ for men ($r = -.28$) and women ($r = -.25$): $z = 0.39, p = .69$. The impact of including sex in the LGC models was tested but did not alter the primary results.

Parameter estimates in Table 3 were used to estimate latent growth curves for the four groups in Figure 1: low childhood SEP and low adult SEP (child LO adult LO), low childhood SEP and high adult SEP (child LO adult HI), high childhood SEP and low adult SEP (child HI adult LO), and high SEP in both childhood and adulthood (child HI adult HI). Examining the estimated growth curves indicates that higher scores on FAI indicate worse functioning in all four groups, rate of change (increase in functional problems) in FAI increases dramatically after age 70, indicating accelerating aging in functional abilities. However, there are marked differences between groups in this aging pattern. Intercept (at age 70) tended to be higher for those with low adult SEP and lower for those with high adult SEP; nevertheless, the highest intercept was found for the group with high child SEP and low adult SEP. Conversely, the lowest intercept was in the group with low child SEP and high adult SEP. Similarly, groups with high adult SEP had slower rates of change before and after age 70 compared groups with low adult SEP. Childhood

SEP played a role in rates of change, before age 70 for those with high adult SEP and after age 70 for those with low adult SEP. The slowest change prior to age 70 occurred in the group with low child SEP and high adult SEP. The fastest change before age 70 occurred for those with low adult and low childhood SEP, after age 70 in the group with high childhood SEP and low adult SEP.

Given the unexpected results for groups that changed SEP from childhood to adulthood (low to high and high to low), follow-up analyses were conducted. Demographic variables for the four groups are presented in Table 4. The smallest group (N = 88) included individuals who were raised in above median SEP households but in adulthood had SEP below the median. There were no significant differences in gender distribution across the 4 groups (chi-square (df = 3) = 4.05, $p = 0.26$), although the percent female was highest in the group with low SEP in both childhood and adulthood. Two-way ANOVAs compared participation (number of IPTs) and length of follow-up across the four groups. Results indicated only main effects of adult SEP for participation ($F(1,653) = 22.37, p < .01$) and follow-up ($F(1,653) = 6.55, p < .05$). Participants with higher adult SEP participated in one more IPT and had 1.75 more years follow-up, on average. A two-way ANOVA was to compare age at intake across the four groups found only the main effect of adult SEP was significant ($F(1, 653) = 22.94, p < .01$). Individuals with low adult SEP had an age of intake that was 2 to 4 years older, on average, than individuals with high adult SEP. Age-based LGC models align the data by age, but an older age of intake can indicate individuals born earlier in the 20th century. A two-way ANOVA was used to compare birthyear across the four groups and again only the main effect of adult SEP was significant ($F(1, 653) = 31.18, p < .01$). Individuals with low adult SEP were born 4 to 6 years earlier than those with high adult SEP (birthyear and age at intake can differ due to the recruitment of new participants into the study up to 2001). No interaction between child and adult SEP was found for any of the variables reported in Table 4.

DISCUSSION

Using a two-slope latent growth curve model of FAI, we found that even when adult SEP was included in the model, childhood SEP was associated with the intercept (at age 70) and the rate of change in FAI up to age 70, providing support for the critical period model. Adult SEP was only marginally associated with the rate of change after age 70, providing modest support for the accumulation of risks model. Finally, the interaction of child and adult SEP had a significant impact on the intercept of the model, providing support for the social mobility model. As a result, individuals with higher adult SEP tended to have a lower intercept and slower rates of change with age in FAI than individuals with lower adult SEP. Individuals who changed SEP status from childhood to adulthood demonstrated divergent patterns. The intercept was highest and the rate of change after age 70 was fastest for those whose SEP decreased from childhood to adulthood. Conversely, the intercept was lowest and the rate of change up to age 70 was slowest for individuals whose SEP increased from childhood to adulthood.

Similar to previous investigations of the impact of SEP on longitudinal patterns of change in functioning with age (Haas, 2008; Lee & Park, 2020), we found that child SEP and the interaction of child and adult SEP were associated with the intercept of the model. Using a two-slope model allowed us to investigate differences in association of SEP with aging across the adult lifespan, i.e., before and after functional aging accelerates around age 70. As a result, we found a significant association of childhood SEP with rate of change in FAI before age 70 and a trending association of adult SEP with the rate of change in FAI after age 70. Lee and Park (2020) used a linear growth curve model and Haas (2008) used a time-based quadratic model; neither approach allows for the examination of differences in association of aging trajectories with SEP in young-old vs. old-old. The effect of childhood SEP on trajectories of change in FAI, at least up to age 70, provides support for the critical period model suggesting that early-life disadvantage can have effects even on late-life outcomes (Ben-Shlomo & Kuh, 2002; Kuh & Ben-

Shlomo, 2004). The diverging patterns of change for individuals with lower and higher adult SEP support the accumulation of risks model (Ferraro & Shippee, 2009; Willson et al., 2007).

The significant interaction effect of child and adult SEP on the intercept of the growth curve model for FAI indicated not only that SEP in both childhood adulthood play a role in functioning in later adulthood, but that changes in SEP over the life-course and timing of disadvantage may play an important role, as predicted by the social mobility model (Hallqvist et al., 2004; Luo & Waite, 2005). Growth curves indicated that the highest intercept and fastest rate of change was found for individuals who experienced downward mobility: from high childhood SEP to low adult SEP. In contrast, the lowest intercept and the slowest rate of change prior to age 70 was found for individuals who experienced upward mobility: from low childhood SEP to high adult SEP. Similarly, Lyu and Burr (2016) reported that downward mobility was associated with lower (but not the lowest) mean cognitive score and stable high SEP or upward mobility was associated with higher mean cognitive scores. Harber-Aschan and colleagues (2020) reported that adults with mixed SEP demonstrated the lowest intercept on a measure of cognitive and physical health, although adults with consistently low SEP exhibited faster rates of decline.

In comparison with the critical period model, social mobility theory suggests the possibility that experiences of midlife can reverse the negative consequences of early life disadvantage (Hallqvist et al., 2004; Pudrovska & Anikputa, 2014; Zimmer et al., 2016). Results for the group with low child SEP and high adult SEP provide some support for the social mobility model. Similarly, Landös and colleagues (2019) reported that higher adult SEP minimized the impact of disadvantage in childhood on measures of activities of daily living for men, but not for women. The social mobility model also suggests that change in the opposite direction is possible: experiences in midlife could outweigh the benefits of early life advantage. In the current study, the group with the highest intercept and the fastest rate of aging had experienced a decline from high childhood SEP to low adult SEP. The FAI aging trajectory for this

group could also result from health selection (Luo & Waite, 2005). Individuals with poor health and functional limitations may experience downward drift in SEP and “select” into lower SEP as a result of reduced employment or educational opportunities (Williams, 1990).

Follow-up investigation of the two groups who experienced changes over the lifespan in SEP indicated that there were no significant gender differences between groups: men and women were equally likely to have stable or changing SEP. Although previous analyses have found that women tend to have a higher intercept in growth curve models of FAI (Finkel et al., 2019), gender could not explain group differences in growth curve parameters. However, the groups with lower adult SEP tended to be older at intake and thus from a somewhat earlier generation. Sweden experienced dramatic changes in educational policy and economy during the 20th century (Sundin & Willner, 2007). Swedish parliament decided to extend the duration of compulsory schooling from 6 to 7 years (SFS 1936:305) for children ages 7-14 starting in 1937, so generations born before 1930 would likely have averaged 1 year less of education than later born cohorts. Some researchers highlight the vital role of education, *per se*, in the association between SEP and health outcomes (Mirowsky & Ross, 2003; Ross & Wu, 1996). Education represents human capital in the abilities to acquire and leverage health information and research suggests that education has a larger impact on health than income and occupational status components of SEP (S. Lynch, 2003, 2006; Mirowsky & Ross, 2003). Thus, it is possible that one year less of education in childhood resulted in both lower adult SEP and less ability to take advantage of health information and thus faster rates of aging in FAI.

Limitations of the current analyses include many of the statistical assumptions common to structural equation models. The data are assumed to be missing at random and the sample is assumed to be relatively homogeneous. As with any longitudinal sample, attrition occurred in SATSA. However, using an age-based growth curve model instead of a time-based model allowed us to maximize power, especially for individuals with more participation waves. Even though the sample was representative of

the population at intake, non-random dropout through the course of the longitudinal studies results in increasingly select samples of adults who are healthy enough to participate. Participants with higher adult SEP participated in one more IPT, on average, than participants with lower adult SEP. Because participants could miss one IPT and be included in the next, differences in IPT participation translated to a modest 1.75-year difference in length of follow-up, on average; thus, the impact of group differences in participation was likely small. Strengths of the study include the use of objectively measured functional abilities. In addition, research nurses visited the participants at their current residence; therefore, data collection could continue even after onset of illness or entry in to care. As a result, wave-to-wave dropout in was quite low (about 8%) and results primarily from mortality, but dropout accumulates across waves. Consequently, our analyses have likely underestimated the extent of change with age. As with many measures of SEP, the combination of occupation and education used here is likely less valid for women than men, particularly in these age cohorts (Featherman & Stevens, 2021). Measures such as subjective SEP or financial strain tend to be less problematic and more predictive of health outcomes than objective SEP (Cundiff & Matthews, 2017; Hoebel & Lampert, 2020; Singh-Manoux, Marmot, & Adler, 2005) and could be used in future tests of these theories. Finally, Sweden is considered a leader in social welfare, which could limit the generalizability of these results to other countries with less well-developed social safety nets. In fact, significant income inequalities and social inequalities still exist in Sweden (Gastwirth, 2014) and were even more pronounced prior to the initiation of the social welfare system beginning in the 1950s, when many of the twins would have experienced the impact of childhood SEP.

Consonant with previous investigations of the impact of SEP on rates of change in health and cognitive outcomes, we found that both childhood and adult SEP impact the growth curve parameters. Long-term effects of high SEP in childhood were seen in slower rates of change up to age 70 and childhood SEP in combination with the more proximal effects of higher adult SEP were associated with

lower FAI intercept at age 70. Application of the two-slope growth curve model allowed for more fine-tuned investigation of when in the adult lifespan childhood and adult SEP impact FAI. Interactions between childhood and adult SEP provide support for social mobility models: dynamic changes in SEP over the life course resulted in dramatically different patterns of aging with evidence for both upward mobility and downward drift in SEP have an impact on aging trajectories for FAI. Evidence suggests that the number of years lost to adverse SEP is equal to or greater than number of years lost due to major risk factors for chronic disease (Stringhini et al., 2018). The social environment is modifiable by policies at local, national, and international levels, and these policies need to recognize that early social disadvantage can have long-lasting health impacts (Mirowsky & Ross, 2003). Initiatives targeting health inequalities should be focused on interventions early in the life course (Corna, 2013; Mirowsky & Ross, 2003). Researchers in future study designs may want to consider other variables that may have an added effect on access to education, occupational attainment, and social mobility (e.g., racism, sexism, exposure to violence/war, other disadvantages).

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Table 1. Descriptive Statistics

Variable	Values
N	654
% Female	57.03
Age Range at Intake	40 - 83
Mean Age at Intake (SD)	62.13 (8.17)
Range in # Waves	1 - 8
Mean # Waves (SD)	4.03 (2.4)
Range in Years Follow-up	0 - 27
Mean Years Follow-up (SD)	15.26 (7.96)
Mean FAI at Intake (SD)	48.29 (10.87)
Mean Childhood SEP (SD)	0.00 (2.07)
Mean Adult SEP (SD)	0.26 (1.69)

Table 2. Results of fitting latent growth curve model to Functional Aging Index

Model	Parameters	-2 Log Likelihood	LRT	df	Significance
					change
1. Base Model	16	16416.70			
2. Base model + child SEP	19	16414.60	2.10	3	$p = .55$
3. Base model + adult SEP	19	16384.20	32.50	3	$p < .01$
4. Base model + child SEP and adult SEP and interactions	25	16376.00	40.70	9	$p < .01$

Note: LRT = Likelihood Ratio Test. Base model: age-based two-slope (+/-70 years) latent growth curve model.

Table 3. Parameter estimates from latent growth curve model

Parameter	Estimate (SE)	Significance
Intercept	47.02 (0.74)	$p < .01$
Intercept x child SEP	-2.17 (1.11)	$p < .05$
Intercept x adult SEP	1.95 (1.32)	$p = .14$
Intercept x child SEP x adult SEP	3.14 (1.60)	$p < .05$
Slope 1	4.37 (0.48)	$p < .01$
Slope 1 x child SEP	-1.53 (0.74)	$p < .05$
Slope 1 x adult SEP	0.38 (0.94)	$p = .68$
Slope 1 x child SEP x adult SEP	2.06 (1.29)	$p = .11$
Slope 2	9.66 (0.75)	$p < .01$
Slope 2 x child SEP	0.39 (1.24)	$p = .75$
Slope 2 x adult SEP	2.31 (1.34)	$p < .10$
Slope 2 x child SEP x adult SEP	-1.98 (1.89)	$p = .30$

SEP is coded low = 0, high = 1. LGC model is centered at age 70, Slope 1 = before 70 years, slope 2 = after 70 years.

Table 4. Demographic variables for high and low SEP groups.

Variable	Child: Low SEP	Child: Low SEP	Child: High SEP	Child: High SEP
	Adult: Low SEP	Adult: High SEP	Adult: Low SEP	Adult: High SEP
N	227	141	88	198
% Female	62.32	58.14	55.00	52.49
Mean Birthyear (SD)	1923.45 (8.78)	1929.85 (9.86)	1925.60 (11.19)	1929.07 (10.81)
Mean Intake Age (SD)	64.50 (7.49)	59.86 (7.41)	62.86 (9.38)	60.87 (8.03)
Mean # IPTs (SD)	3.44 (2.22)	4.46 (2.42)	3.73 (2.38)	4.64 (2.39)
Mean Follow-up (SD)	14.17 (7.88)	15.95 (7.76)	15.01 (8.17)	16.75 (7.76)

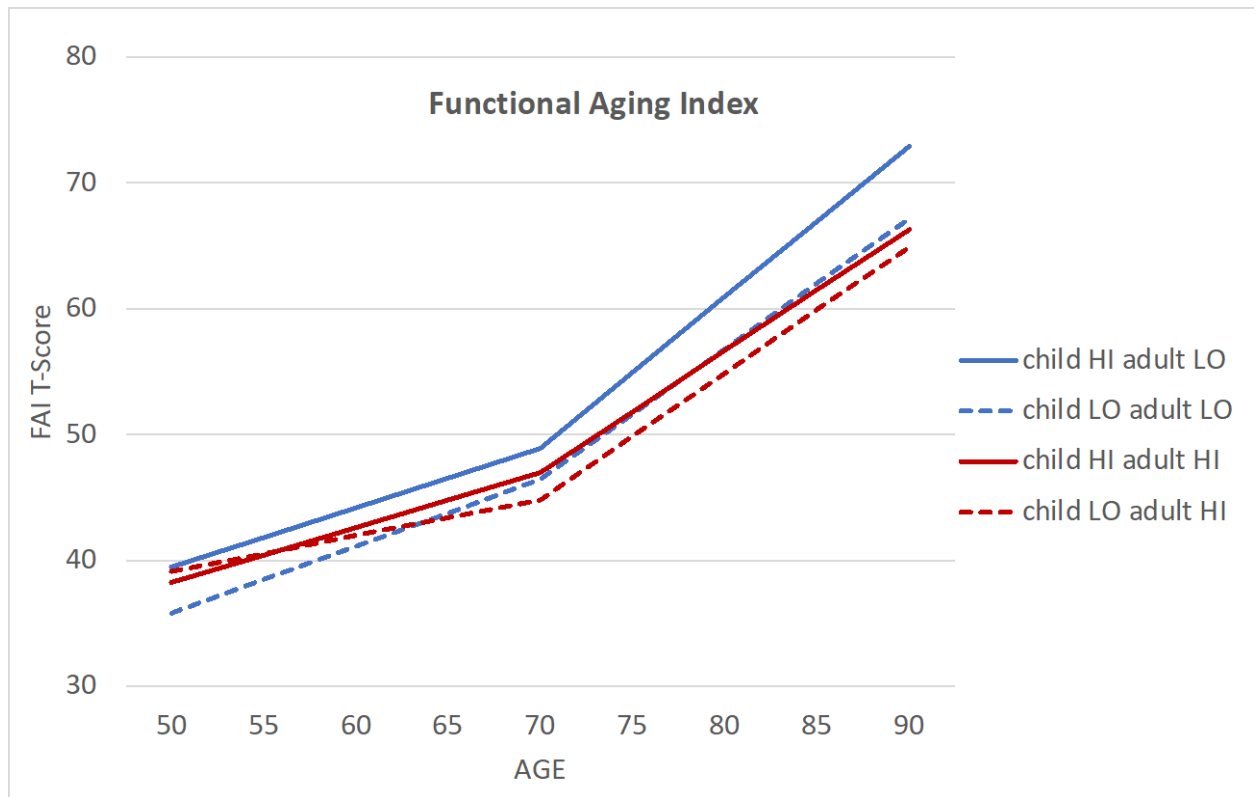


Figure 1: Estimated latent growth curves for model including both child and adult SEP as covariates.

Child HI = high SEP in childhood; child LO = low SEP in childhood; adult LO = low SEP in adulthood; adult HI = high SEP in adulthood.