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Are neighbourhood inequalities in adult health explained by socio-economic and psychosocial determinants in adolescence and the subsequent life course in northern Sweden? A decomposition analysis

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ABSTRACT

This study explains neighbourhood deprivation inequalities in adult health for a northern Swedish cohort by examining the contribution of socio-economic and psychosocial determinants from adolescence (age 16), young adulthood (age 21) and midlife (age 42) to the disparity. Self-reported information from 873 participants was drawn from questionnaires, with complementary neighbourhood register data. The concentration index was used to estimate the inequality while decomposition analyses were run to attribute the disparity to its underlying determinants. The results suggest that socio-economic and psychosocial factors in midlife explain a substantial part, but also that the inequality can originate from conditions in adolescence and young adulthood.

1. Background

In recent years, three broad strands of social epidemiological literature – focusing on neighbourhoods, life course and inequalities, respectively – have developed without much direct conceptual, empirical or methodological integration. First, 'neighbourhood and health' research acknowledges that health is unevenly distributed across residential areas, but attempts to explain this inequality have focused primarily on concurrent circumstances in adulthood. Secondly, the 'life course perspective' states that adult health may also be affected by exposures during earlier life periods, but instead of focusing on circumstances to understand health disparities, this research has been mostly concerned with explaining overall population health. Thirdly, while 'social inequalities in health' have received much empirical attention, the focus in this field has been mostly directed towards health differences at the individual level (e.g. by income or education), with efforts to explain the disparity being largely contemporaneous. Ultimately, the present study merge lines of social epidemiological inquiry that have evolved chiefly in parallel during the last decades. As a result, it explores the issue of neighbourhood deprivation inequalities in adult health by examining the contribution of socio-economic and psychosocial determinants from adolescence, young adulthood and midlife to the disparity.

Following the increased interest in residential variations in ill-health, studies examining the so-called 'neighbourhood effects on health' have typically used cross-sectional data to explore whether the inequality could be attributed to the context of the area, either independently of or in combination with concurrent individual characteristics (Arcaya et al., 2016; Diez Roux and Mair, 2010; Oakes et al., 2015). Based on this research, the uneven distribution of health across differently deprived areas have been approached primarily from a contemporaneous perspective. As a result, the possibility that neighbourhood socio-economic inequalities in adult health (henceforth referred to as neighbourhood *deprivation* inequalities in health) can be explained by exposures during earlier life periods have so far been largely overlooked (Osypuk, 2013).

In contrast to the cross-sectional dominance of neighbourhood and health research, studies drawing upon the life course epidemiological approach (Ben-Shlomo and Kuh, 2002) acknowledge that the implications of daily living conditions are not always immediate, but can have effects on health that do not become visible until decades later (Lynch and Smith, 2005). Supporting this notion is research that indicate how individual (Agahi et al., 2014; Hyde et al., 2006) and contextual (Gustafsson, Bozorgmehr, Hammarström, & Sebastian, 2017) factors from the previous life course are important for self-reported health problems in adulthood. As explained by Diderichsen et al. (2001), however, the determinants *of health* and of *health inequalities* are not necessarily the same. As a result, contemporary life course research has

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typically focused on factors that may be helpful to understand – and ultimately improve – the health of the population, rather than those that can be used to reduce the disparities (Dahlgren and Whitehead, 2006).

In light of the growing awareness of social inequalities in health, the disproportionate concentration of ill-health among disadvantaged groups has nevertheless received attention in a parallel line of inquiry. Despite the fact that conceptual descriptions of this matter usually include both contextual and life course perspectives (Arcaya et al., 2015; Marmot et al., 2012; Solar and Irwin, 2010), there is a scarcity of empirical studies that integrate them simultaneously to explain the disparities. For example, most research in this field has focused on health differences by income or education, thereby overlooking the socio-economic inequalities in adult health that exist at higher contextual levels, such as between neighbourhoods (Macintyre and Ellaway, 2003). In addition, studies that attempt to identify conditions that underpin the inequalities with decomposition methods (e.g. Hosseinpoor et al., 2006; McGrail et al., 2009; Morasae et al., 2012; Sortsø et al., 2017) have, with few exceptions (see Mosquera, San Sebastian, Ivarsson, Weinehall, & Gustafsson, 2017) not applied a life course perspective. To our knowledge, none have used decomposition methods to explain neighbourhood deprivation inequalities in adult health.

To guide us in our attempt to empirically explore the uneven distribution of adult health between differently deprived neighbourhoods, the conceptual framework of the study is presented below.

1.1. Conceptual framework

When it comes to determinants of not only *health* but of *health inequalities*, a strong determinant of health can be expected to matter for a corresponding inequality in health to the extent the determinant is also i) unequally distributed across social groups (in our case neighbourhoods) and ii) sufficiently frequent to be able to make an impact on a population-level phenomenon such as health inequality. As clarified by Jokela (2015), explaining neighbourhood deprivation inequalities in adult health thus requires identifying conditions which influence people's health and simultaneously relates to their area of residence.

As emphasized in the social epidemiological literature, socio-economic circumstances (e.g. education, employment and income/financial resources) are essential in the development of ill-health and disease (Glymour et al., 2014). These factors are important because they affect people's cognitive and emotional skills (e.g. education) as well as their material living conditions and access to various health-enhancing resources (e.g. income and occupation) (Galobardes et al., 2006). In addition, socio-economic factors can also act indirectly on health through psychosocial pathways e.g. stress, lack of support and low control (Marmot, 2004).

Over and above being central determinants of health, socio-economic conditions also largely determine people's area of living. According to Hedman et al. (2011), households tend to sort themselves into different neighbourhoods based on their i) similarity to the other residents and/or ii) ability to 'choose' an area that fits preferences and needs – processes that are overall largely determined by socio-economic characteristics (Hedman and van Ham, 2012). When residing in a particular place, however, Popay et al. (2003) have identified psychosocial circumstances such as low control and feelings of isolation as pathways to poor health for people in deprived neighbourhoods.

Ultimately, socio-economic factors in adulthood can contribute to neighbourhood deprivation inequalities in adult health by influencing people's health and their area of residence, either independently of or indirectly through psychosocial conditions. In accordance with the life course approach, however, focusing only on concurrent factors in adulthood may be insufficient, because neither people's health nor their life chances are affected solely by contemporaneous circumstances (Kuh et al., 2003). Instead, adult outcomes can be attributed to experiences from earlier life periods through various processes, exemplified by formulations of the *life course models* within this field (Lynch and Smith, 2005). The *sensitive period model* suggests that exposures during specific periods of development e.g. childhood (Irwin et al., 2007) or adolescence (Sawyer et al., 2012) can have long-term effects that act independently of exposures later in life (Mishra et al., 2010). In contrast, the notion of *risk accumulation* states that the likelihood of poor adult outcomes increases as detrimental exposures accumulate over time (Kuh et al., 2003), indicating that exposures can either be sequentially linked (chain of risk) or simply co-occurring (cumulative risk) (Lynch and Smith, 2005).

Building on this framework, neighbourhood deprivation inequalities in adult health could be rooted in the socio-economic and psychosocial conditions people experience earlier in life, either independently of or through adult conditions. This aligns with research that suggest, for example, that family socio-economic conditions earlier in life are associated with both adult neighbourhood of residence (Gustafsson et al., 2013) and self-rated health (Hyde et al., 2006). The present study therefore examines the contribution of socio-economic and psychosocial factors from three life periods (adolescence, young adulthood and midlife) to neighbourhood deprivation inequalities in adult health.

1.2. Study context

In this paper, the issue of neighbourhood deprivation inequalities in adult health is studied for a group of adolescents who grew up in a northern Swedish municipality in the early 1980s and who have been followed throughout young adulthood in the mid-1980s up until middle age in 2007 (Hammarström & Janlert, 2012). While the participants initially lived in northern Sweden, some of them have nevertheless relocated to other parts of the country over time.

Historically, Sweden has been characterized by a strong welfare state with a focus on, for example, redistribution, a generous social security system and universal health care. Since this will possibly reduce the number of severely disadvantaged people, health inequalities could potentially be smaller in the present study than in other contexts. Despite this, however, income inequalities have increased markedly in Sweden since the early 1990s and become particularly widespread throughout the 2000s (OECD, 2011). In combination with an intensified re-commodification process during this period, the welfare system has been seriously eroded (Fritzell et al., 2014), which means that the current study is situated in what Raphael (2014) described as a 'declining welfare state'.

As an indicator of the participant's health, functional somatic symptoms (FSS) is used in the present study. FSS pertain to a clustering of physical complaints that cannot be confidently attributed to an organic disease (Kroenke, 2003). The reasons for using this indicator follow from the fact that FSS is usually seen as a response to chronic stress and states of negative affect (Schenk et al., 2017), and is thus largely affected by adverse life circumstances (Tak et al., 2015) including the socio-economic context of the neighbourhood (Gustafsson, Hammarström, & San Sebastian, 2015). In addition, it display a socio-economic gradient that may increase with age and which seems to be attributed to social and material inequalities from across the life course (San Sebastian, Hammarström, & Gustafsson, 2015).

2. Methods

2.1. Sample

The Northern Swedish Cohort provided prospective data collected five times (in 1981, 1983, 1986, 1995 and 2007) through self-administered questionnaires on all individuals who attended 9th grade in Luleå municipality in 1981 (Hammarström & Janlert, 2012). Out of the 1071 students still alive across the 26-year period, 94.3% participated in all follow-ups (n = 1010).

For the current study, information on the health complaints and

living conditions of the participants was retrieved from 1981, 1986 and 2007 surveys (participants aged 16, 21 and 42 years). Information on the neighbourhood of residence as well as sociodemographic information on all residents in these neighbourhoods was collected from the Swedish registers and linked to all individuals participating in the last follow-up. In accordance with SAMS (small-area market statistics), the neighbourhood in which the participants resided at each subsequent year of data collection was constructed as a polygon, demarked by roads or similar physical borders and averaging about 1000 individuals (Gustafsson & San Sebastian, 2014). Specifically, information on the SAMS area in which the study participants were residing on December 31st in 1980, 1986 and 2007 has been collected. To better capture the neighbourhood context of the family, area of residence in 1980 was chosen instead of 1981 (Gustafsson & San Sebastian, 2014).

2.2. Variables

2.2.1. Health outcome

The health outcome was functional somatic symptoms (FSS) at age 42, an index based on three items scored 0–2 which together covered ten different symptoms (cardiopulmonary/autonomic, gastrointestinal, musculoskeletal and general symptoms) occurring during the last 12 months. With the question 'Do you have (or have you had during the last 12 months) any of the following symptoms: 1) headache or migraine; 2) other stomach ache; 3) nausea; 4) backache, hip pain or sciatica; 5) fatigue; 6) breathlessness; 7) dizziness; 8) overstrain?'; the first item covered eight symptoms (response options 'No' = 0, 'Yes, mild' = 1 and 'Yes, severe' = 2). By giving a positive response to the question 'Have you experienced any nervous problems during the last 12 months?' the participants were then allowed to select between six different types of nervous problems, with palpitations being one alternative. Together with the question 'How often have you had nervous problems during the past 12 months?,' the frequency ('Never' = 0, 'Sometimes' = 1 and 'Always' = 2) of palpitations was measured and included in the index. The last question 'Have you had sleeping difficulties during the past 12 months?', with the response options 'Never' = 0, 'Sometimes' = 1 and 'Often'/'Always' = 2, measured frequency of sleeplessness. After being summarized, Cronbach's alpha was 0.78 and the measure has displayed acceptable factorial invariance and internal consistency over time (Hammarström et al., 2016).

2.2.2. Inequality indicator

The inequality indicator was neighbourhood deprivation at age 42, a measure used to estimate the socio-economic context of the area. This measure is based on register data and has been used and described in more detail elsewhere (see Gustafsson & San Sebastian, 2014). In short, it was developed in accordance with previous research but also depended on information available in the registers, resulting in an index based on eight indicators. The percentage of neighbourhood residences with: 1) *low income* (an annual disposable income ≤ the 10th percentile of the Swedish population at the corresponding year); 2) *high income* (an annual disposable income ≥ the 90th percentile of the Swedish population at the corresponding year, reverse-coded); 3) *housing allowance;* 4) *wealth* (paying any wealth tax, reverse-coded);5) *non-employment* (those over 18 who had their main income from unemployment, early retirement, or sickness benefits); 6) *single parents*; 7) *low education* (people over 25 with only primary education); and 8) *high education* (people over 25 with 2 or more years of secondary education, reverse coded) created the index.

Each indicator was standardized and the mean across the Z-scores used to acquire an estimate of neighbourhood deprivation. The variable was then reverse-coded, indicating less deprivation at higher values (range 3.13–10.74), to make it follow the theory and methodology of the concentration index (O'Donnell et al., 2008). Cronbach's alpha was 0.86.

2.2.3. Life course socio-economic and psychosocial determinants of neighbourhood deprivation inequalities in adult health

Life course socio-economic factors were assessed using parental occupation and unemployment, as well as own occupation, education, employment status and financial resources as indicators (Galobardes et al., 2006). In contrast, psychosocial variables were selected to capture social circumstances which can potentially influence people's outlook and behaviour (Martikainen et al., 2002). To acknowledge the possibility of health selection – that neighbourhood inequalities in health are explained by the systematic sorting of people into neighbourhoods by their health (Mair et al., 2008) – FSS from the previous life course was included, together with demographic characteristics, as control variables.

According to Wagstaff et al. (2011), including the inequality indicator to explain the disparities is important to prevent other factors from picking up its effect. In other words, excluding this variable from the decomposition analysis could lead to an overestimation of factors to which are highly correlated with neighbourhood deprivation or to the residuals being larger. After being divided into quintiles, neighbourhood disadvantage at age 42 was therefore included also as a determinant in the decomposition analysis.

2.2.3.1. Adolescence (age 16)

2.2.3.1.1. Socio-economic factors. *Parental occupation,* two parents in social group 3 (Johansson, 1970) (yes = 1; no = 0). *Parental unemployment,* someone in the family being unemployed during the last 12 months (yes = 1; no = 0). *Neighbourhood deprivation,* the operationalization at age 16 was similar to the one at age 42 (see above) but included *low occupation* and *high occupation* (the highest occupation in the household being unskilled manual worker and professional/self-employed, respectively) instead of high and low education because these latter indicators were not available in the registers during this year. Before being included in the decomposition analysis, this variable was divided into quintiles indicating highest (= 1) to lowest (= 5) level of deprivation. Internal consistency was $\alpha = 0.93$.

2.2.3.1.2. Psychosocial factors. *Parental loss,* parents split up, parent (s) die or parents never lived together (yes on either item = 1; no = 0). *Parental illness,* parent(s) with physical illness, mental or alcohol problems (yes = 1; no = 0).

2.2.3.2. Young adulthood (age 21)

2.2.3.2.1. Socio-economic factors. *Occupation,* manual work (= 1) and non-manual/self-employed (= 0). *Low cash margin,* the inability to raise 5,000 SEK within a week (yes = 1; no = 0). *Unemployment,* currently unemployed (yes = 1; no = 0). *Neighbourhood deprivation,* the operationalization at age 21 was identical to the one at age 16 (see above) but with an internal consistency of $\alpha = 0.88$.

2.2.3.2.2. Psychosocial factors. *Social influence,* whether the participant 1) could decide sufficiently often; 2) speak his/her mind; 3) felt he/she was appreciated by others; and 4) found it hard to get others to listen. The response options yes = 0 and no = 1 were summarized and dichotomized at the 80th percentile (> = 1). *Social isolation*, mostly or always alone during the days (yes = 1; no = 0).

2.2.3.3. Midlife (age 42)

2.2.3.3.1. Socio-economic factors. *Occupation,* manual work (= 1) and non-manual/self-employed (= 0). *Low cash margin,* the inability to raise 15000 SEK within a week (yes = 1; no = 0). *Unemployment,* unemployed for at least a year since age 30 (yes = 1; no = 0). *Education,* university degree/post-secondary education or equivalent (= 0) and higher secondary education or less (= 1). *Financial strain,* forced to withhold from different activities due to financial reasons during the last 12 months. The number of 'often' responses (as compared to 'seldom', never', 'not applicable') were summarized and dichotomized at the 80th percentile (> = 1). *Neighbourhood*

deprivation at age 42 is the same variable as the inequality indicator (see above) but divided into quintiles, from highest (= 1) to lowest (= 5) level of deprivation.

2.2.3.3.2. Psychosocial factors. Social influence, whether the participant 1) could decide sufficiently often; 2) speak his/her mind; 3) felt he/she was appreciated by others; and 4) found it hard to get others to listen. The response options yes = 0 and no = 1 were summarized and dichotomized at the 80th percentile (> = 1). *Social isolation*, the number of people 1) with similar interests the participant knows; 2) the participant meets regularly every week; 3) that are available to visit at any time; and 4) the participant can speak openly to. These items were summarized and dichotomized at the 20th percentile (< = 1). *Separation,* split up from a long-term relationship since age 30 (yes = 1; no = 0). *Exposure to threat and violence*, experiences of 1) physical violence; 2) threats of violence; 3) personal prosecution through mean words and actions at the workplace; and 4) sexual harassment at the workplace (yes on either item = 1; no = 0).

2.2.3.4. Control variables

2.2.3.4.1. Previous health status. Functional somatic symptoms at age 16 and 21, operationalized identically as at age 42 (see above) but with an internal consistency of α = 0.69 (both age 16 and 21).

2.2.3.4.2. Demographic factors. Civil status, married/in a cohabitating relationship at age 42 (yes = 1; no = 0). *Having children* (yes = 1; no = 0) at age 42. *Sex*, man (= 1) and woman (= 0).

2.3. Analytical strategy

As indicated by initial data screenings, the strengths of associations (according to Pearson's r, cross-validation using Spearman's rho yielded similar results) between all variables in the models were weak to moderate (r < 0.45). In addition, the variance inflation factor (VIF) indicates that multicollinearity is present only at a low level in the analysis (all values < 1.5).

To study the issue of neighbourhood deprivation inequalities in adult health, the analysis was performed in two steps. First, to estimate whether FSS at age 42 was unevenly distributed across neighbourhoods at age 42, the concentration curve (CC) and the corresponding concentration index (C) (Wagstaff et al., 1991) were estimated. Secondly, to examine the contribution of the determinants to this inequality i.e. the concentration index C, Wagstaff et al. (2003) decomposition analysis was applied. In this latter analysis, determinants were estimated separately for each life period in model 1 (factors from age 16), model 1a (factors from age 21) and model 1b (factors from age 42). Thereafter, their joint contribution was assessed in model 2 (factors from age 16 and 21) and model 3 (factors from age 16, 21 and 42).

2.3.1. Step 1 – Estimating neighbourhood deprivation inequalities in adult health

In our case, the concentration curve (*CC*) was obtained by plotting the cumulative proportion of ill-health (FSS at age 42) against the cumulative proportion of the population ranked by the inequality indicator (neighbourhood deprivation at age 42), starting with the most disadvantaged. If FSS is equally distributed across neighbourhoods, CC will coincide with the diagonal (45-degree) line. By contrast, if the CC lies above the diagonal, then FSS is higher in deprived neighbourhoods and if the CC lies below then FSS is higher in more advantaged neighbourhoods. Based on this, the concentration index *C* is defined as twice the area between the CC and the 45-degree line and takes a value between − 1 and 1 (Wagstaff et al., 2011). Accordingly, C will be zero if CC coincides with the diagonal, be positive if it lies below and negative if it lies above. In accordance with O'Donnell et al. (2008) the concentration index C for FSS at age 42 can be expressed as:

$$C = \frac{2}{\mu}\sum_{i=1}^{n} h_i r_i - 1 \quad (1)$$

where h is the outcome of interest and h_i is therefore the level of FSS for individual i; μ is the mean of FSS; n is the size of the analytical sample and r_i is the fractional rank of individual i according to the level of deprivation in his/her neighbourhood.

2.3.2. Step 2 – Explaining neighbourhood deprivation inequalities in adult health

To estimate the contribution of life course determinants to the inequality, decomposition analyses were then run, in which the concentration index C from the above analysis was the dependent variable (O'Donnell et al., 2008). This regression-based method allows *C* (the inequality) to be attributed to a set of *k* determinants where the contribution of each factor is obtained by multiplying its elasticity (a regression coefficient for the association between the determinant and FSS at age 42 that is weighted by the frequency of the determinant) with its concentration index (reflecting the distribution of the determinant with respect to neighbourhood disadvantage at age 42). As described by O'Donnell et al. (2008) for any linear additive regression model of health (*y)*:

$$y = \alpha + \sum_k \beta_k x_k + \varepsilon \quad (2)$$

the concentration index for *y, C*, is written as:

$$C = \sum_k (\beta_k \overline{X}_k/\mu) C_k + GC_\varepsilon/\mu \quad (3)$$

here, μ is the mean of y, $\overline{X}_k$ is the mean of X_k, C_k is the concentration index for X_k and GC_ε is the generalized concentration index for the error term (ε). Eq. (3) shows that C is equal to the weighted sum of concentration indexes of the *k* determinants, and where the weight for X_k is the elasticity of *y* with respect to X_k. The last component GC_ε/μ is the residual, reflecting the inequality that is *not* explained by the systematic variation in the determinants across neighbourhoods. For a well-specified model, this should approach zero (O'Donnell et al., 2008).

2.3.3. Sensitivity analysis

To assess whether the clustering of FSS within neighbourhoods at age 42 could be a source of bias, a sensitivity analysis considering random effects in the decomposition analysis was performed (specifically, the 'regr outcome $X' command in STATA 13 was replaced with the 'mixed outcome $X || residential_areas: ' command). Since this did not change the estimates, the findings from the single-level models were retained and reported in the result section.

2.4. Ethical considerations

Ethical approval was granted by the Regional Ethical Review Board in Umeå (Dnr 07-057M and 2011-326-32M) and since all participants were informed about the study, answering the survey was considered consent to participate.

3. Results

Descriptive statistics are presented in Table 1. When aged 16, the mean level of FSS was 3.28, a number which decreased to 2.82 by age 21 but then rose again to 4.20 by age 42. In 1981 at age 16, the participants resided in 71 different neighbourhoods with an average deprivation level of 5.07. While the number of neighbourhoods increased to 199 in 1986 (age 21), the deprivation mean remained similar at 4.96. At age 42, the sample lived in 342 different neighbourhoods with an average deprivation level of 8.05.

Results from the main analyses are presented in Fig. 1 and Table 2. The concentration curve (Fig. 1) is above the diagonal line of equality and the corresponding concentration index (row labelled 'Inequality' in Table 2) small but significant, C = −0.041 (95% CI: − 0.071, − 0.011), with a negative value indicating that FSS was concentrated to people living in more deprived neighbourhoods.

Table 1
Descriptive statistics for all variables in the models; mean (standard deviation) for functional somatic symptoms and neighbourhood deprivation, and N (proportions, %) for the rest of the variables (n = 873).

Measures	Estimate		
	Age 16	Age 21	Age 42
Functional somatic symptoms	3.28 (2.503)	2.82 (2.485)	4.20 (3.296)
Socio-economic variables			
Neighbourhood deprivation	5.07 (0.803)	4.96 (0.693)	8.05 (1.147)
Manual parental occupation	321 (36.8%)		
Parental unemployment	170 (19.5%)		
Manual occupation		547 (62.7%)	293 (33.6%)
Low cash margin		237 (27.1%)	88 (10.1%)
Unemployment		69 (7.9%)	141 (16.2%)
Low education			473 (54.2%)
Financial strain			123 (14.1%)
Psychosocial variables			
Parental loss	194 (22.2%)		
Parental illness	184 (21.1%)		
Low social influence		88 (10.1%)	132 (15.1%)
Social isolation		108 (12.4%)	184 (21.1%)
Separation			308 (35.3%)
Harassed			115 (13.2%)
Control variables			
Having children			207 (23.7%)
Married/cohabitating			715 (81.9%)
Sex, men	455 (52.1%)		

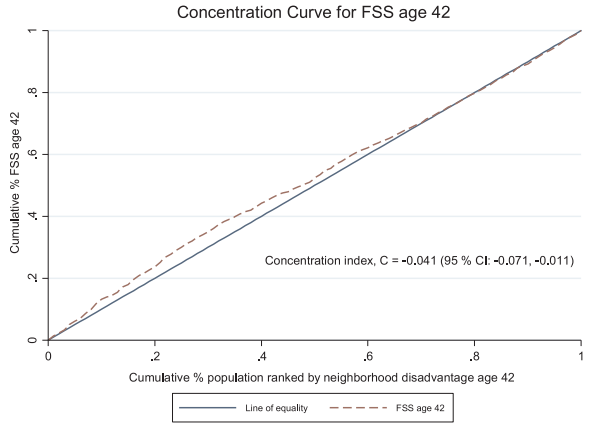

Fig. 1. Concentration curve for cumulative FSS at age 42 by cumulative percentage of the population, ranked by concurrent neighbourhood deprivation.

In the next step, the inequality was decomposed in a stepwise manner. First, the separate contribution of determinants from adolescence (age 16), young adulthood (age 21) and mid-adulthood (age 42) was estimated, respectively. In model 1, the estimate for parental occupation at age 16 in the 'Coeff' column for example, points to a positive association between this socio-economic indicator and FSS at age 42, after adjusting for all other variables at age 16 (B = 0.19). Moreover, the negative 'CI' (− 0.143) indicates that people with manually working parents at age 16 were concentrated to more deprived neighbourhoods at age 42. According to the 'Cont' estimate (*Elast* x *CI*), having manually working parents at age 16 contributed with − 0.002 to the overall concentration index (−0.041), or 5.7%. All in all, results in model 1 indicate that approximately 35% of the neighbourhood deprivation inequality in FSS at age 42 was attributed to adolescent factors, with 12% being explained by socio-economic (individual level = 7%; neighbourhood disadvantage = 5%) and about 15% by psychosocial factors.

When estimating the separate contribution of factors from young adulthood, results in model 1a indicate that factors at age 21 explain about 30% of the inequality, to which previous health status (FSS at age 21 = 15%) and psychosocial factors (6.5%) contributed the most. Similarly, in model 1b the results suggest that factors from mid-adulthood separately accounted for almost 92%. In this regard, socio-economic factors explained about 62% (individual level = 53%; neighbourhood disadvantage = 9%) and psychosocial conditions 38% of the neighbourhood deprivation inequality in FSS.

Secondly, when determinants from adolescence and young adulthood were included in model 2, the joint contribution of adolescent factors decreased from 35% to 18% and from young adulthood from 30% to 24%. Results from this model nevertheless suggest that approximately 42% of the inequality was explained by determinants from these life periods together. While previous health status explained the most (FSS at age 21 = 14%), the total contribution of socio-economic (6.4%) and psychosocial (6.5%) factors was numerically similar.

Lastly, by including factors at all ages as well as demographic control variables in model 3, the total proportion of inequality explained increased to 99%, with 89% being attributed to determinants from mid-adulthood (as compared to 92% in model 1b). Adding factors from midlife reduced the joint contribution of determinants from adolescence and young adulthood to about 4% and 2.5%, respectively. In this model, socio-economic factors at age 42 were the largest contributors with about 60% (individual level = 45%; neighbourhood disadvantage = 15%) followed by psychosocial factors with 28%. With regard to the other control variables, having children explained about 11% whilst gender did not contribute anything to the overall inequality.

4. Discussion

Situated in the intersection between the neighbourhood and health, health inequality and life course epidemiological literatures, the present study attempted to explain neighbourhood deprivation inequalities in adult health for a northern Swedish cohort that has been followed for almost three decades, from adolescence to mid-adulthood. To do this, the contribution of socio-economic and psychosocial factors from three life periods to the disparity was estimated. The results suggest that neighbourhood deprivation inequalities in adult health are largely attributed to socio-economic conditions at both individual and neighbourhood levels, and to a slightly smaller degree, to psychosocial factors in midlife. The analysis also finds that the disparity can be rooted in the health and life circumstances people experience throughout adolescence and young adulthood, but that this influence seems to be largely dependent on concurrent conditions.

As indicated by the small, but negative concentration index, the results from the initial analysis suggest that poor health is concentrated in more socio-economically deprived neighbourhoods, pointing to a disparity in favour of the privileged. Albeit employing a novel method to estimate neighbourhood-based inequalities, this finding corresponds well with 'neighbourhood and health' research in Sweden (Gustafsson & San Sebastian, 2014) and elsewhere (Astell-Burt and Feng, 2015; Rocha et al., 2017). The greatest contribution of the current report is, however, the decomposition of this inequality by socio-economic and psychosocial factors from the previous and concurrent life course.

According to the results in model 1b, which indicate that occupation, education and a strained financial situation in adulthood were i) associated with poorer health, ii) more common among residents in deprived neighbourhoods and iii) sufficiently common circumstances, a large proportion of the neighbourhood deprivation inequality in adult health was attributed to individual level socio-economic factors in midlife. This aligns well with our conceptual framework, which highlights how these conditions largely determine people's area of residence (Hedman et al., 2011) as well as their health (Glymour et al., 2014). In addition, while including concurrent neighbourhood deprivation as a determinant was statistically motivated, this turned out to be a fairly influential indicator in the analysis. Based on this finding, over and above the contribution of individual factors, the neighbourhood socio-

Table 2
The contribution of determinants in adolescence (age 16), young adulthood (age 21) and midlife (age 42) to the neighbourhood deprivation inequality in midlife FSS (n = 873). Presenting the contribution of determinants separately from age 16 (model 1), age 21 (model 1a) and age 42 (model 1b). The joint contribution of determinants from age 16 and 21 (model 2), and age 16, 21 and 42 (model 3).

	Model 1 (only factors age 16)				**Model 2 (factors age 16 and 21)**				**Model 3 (factors age 16, 21 and 42)**			
	Coeff	**Elast**	**CI**	**Cont (%)**	**Coeff**	**Elast**	**CI**	**Cont (%)**	**Coeff**	**Elast**	**CI**	**Cont (%)**
Age 16												
FSS	0.26**	0.205	− 0.017	− 0.003 (8.50)	0.09*	0.071	− 0.017	− 0.001 (2.97)	0.08	0.066	− 0.017	− 0.001 (2.71)
ND	− 0.04	− 0.021	0.095	− 0.002 (4.93)	− 0.02	− 0.008	0.095	− 0.0008 (1.91)	0.03	0.017	0.095	0.002 (− 3.87)
Par. Occup.	0.19	0.016	− 0.143	− 0.002 (5.70)	0.26	0.022	− 0.143	− 0.003 (7.89)	0.06	0.005	− 0.143	− 0.0007 (1.82)
Par. Unemp.	0.20	0.009	− 0.064	− 0.0006 (1.42)	− 0.21	− 0.009	− 0.064	0.0006 (− 1.49)	− 0.20	− 0.007	− 0.064	0.0004 (− 1.09)
Par. Loss	0.38	0.020	− 0.128	− 0.002 (6.26)	0.11	0.006	− 0.128	− 0.0008 (1.89)	0.15	0.008	− 0.128	− 0.001 (2.49)
Par. Illness	0.46	0.023	− 0.150	− 0.003 (8.44)	0.25	0.013	− 0.150	− 0.002 (4.63)	0.09	0.004	− 0.150	− 0.0007 (1.64)
Subtotal %				**35.25**				**17.82**				**3.74**
Age 21	**Model 1a (only factors age 21)**											
	Coeff	**Elast**	**CI**	**Cont (%)**	**Coeff**	**Elast**	**CI**	**Cont (%)**	**Coeff**	**Elast**	**CI**	**Cont (%)**
FSS	0.49**	0.331	− 0.018	− 0.006 (15.21)	0.45**	0.307	− 0.019	− 0.006 (14.08)	0.36**	0.247	− 0.019	− 0.005 (11.32)
ND	− 0.05	− 0.025	0.069	− 0.002 (4.20)	− 0.02	− 0.009	0.069	− 0.0006 (1.62)	0.01	0.004	0.069	0.0003 (0.66)
Occupation	0.17	0.025	− 0.068	− 0.002 (4.25)	0.12	0.017	− 0.068	− 0.001 (2.92)	− 0.05	− 0.007	− 0.068	0.0005 (− 1.19)
Low cash	− 0.09	− 0.006	− 0.153	0.001 (− 2.31)	− 0.09	− 0.006	− 0.153	0.0009 (− 2.31)	− 0.46	− 0.030	− 0.153	0.004 (− 11.13)
Unemp.	0.27	0.005	− 0.116	− 0.0006 (1.45)	0.17	0.003	− 0.116	− 0.0004 (0.93)	− 0.17	− 0.003	− 0.116	0.0004 (− 0.90)
Influence	0.85*	0.020	− 0.068	− 0.001 (3.36)	0.88*	0.021	− 0.068	− 0.001 (3.51)	0.30	0.007	− 0.068	− 0.0005 (1.20)
Isolation	0.56	0.016	− 0.079	− 0.001 (3.20)	0.55	0.016	− 0.079	− 0.001 (3.14)	0.42	0.012	− 0.079	− 0.0009 (2.36)
Subtotal %				**29.39**				**23.89**				**2.32**
Age 42	**Model 1b (only factors age 42)**											
	Coeff	**Elast**	**CI**	**Cont (%)**					**Coeff**	**Elast**	**CI**	**Cont (%)**
ND	− 0.02	− 0.009	0.403	− 0.004 (9.13)					− 0.03	− 0.015	0.403	− 0.006 (14.60)
Occupation	0.17	0.013	− 0.230	− 0.003 (7.67)					0.17	0.014	− 0.230	− 0.003 (7.84)
Low cash	0.77	0.018	− 0.345	− 0.006 (15.53)					0.61	0.014	− 0.345	− 0.005 (12.26)
Strain	1.06*	0.035	− 0.236	− 0.008 (20.45)					0.84*	0.028	− 0.236	− 0.007 (16.23)
Education	0.22	0.028	− 0.109	− 0.003 (7.57)					0.27	0.034	− 0.109	− 0.004 (9.26)
Unemp.	0.09	0.003	− 0.165	− 0.0006 (1.47)					0.01	− 0.0003	− 0.165	− 0.0001 (0.15)
Separation	0.31	0.026	− 0.129	− 0.003 (8.34)					0.25	0.021	− 0.129	− 0.003 (6.63)
Influence	1.29**	0.046	− 0.165	− 0.007 (18.66)					0.91*	0.033	− 0.165	− 0.005 (13.23)
Isolation	1.07**	0.054	− 0.023	− 0.001 (3.05)					0.85*	0.043	− 0.023	− 0.001 (2.43)
Harrased	1.63**	0.051	− 0.064	− 0.003 (8.10)					1.22**	0.038	− 0.065	− 0.002 (6.03)
Subtotal %				**91.90**								**88.69**
Control variables									**Coeff**	**Elast**	**CI**	**Cont (%)**
Sex									− 0.62*	− 0.077	− 0.023	0.002 (− 4.27)
Civil status									− 0.09	− 0.005	− 0.219	0.001 (− 2.62)
Children									− 0.48	− 0.093	0.050	− 0.005 (11.27)
Subtotal %												**4.38**
Total % expl							**41.71**				**99.14**	
Inequality (SE)		**− 0.041 (0.015)**										

* p < 0.05 and ** p < 0.01.
Coeff, a regression coefficient estimating the association between the determinant and FSS at age 42; *Elast* (elasticity), the *Coeff* weighted by the frequency of the determinant; *CI* (concentration index), the distribution of the determinant with respect to neighbourhood disadvantage at age 42; *Cont,* the contribution to the overall concentration index in absolute (same as the C) and relative (percent) scale.

economic inequalities themselves seem to explain a considerable proportion of the unequal distribution of health between differently deprived areas. Moreover, irrespective of whether psychosocial factors act independently of or follow from socio-economic conditions (which we cannot disentangle), our conceptual framework suggested that they should explain a portion of the inequality. In line with Popay et al. (2003), our results indicated that a lack of influence and a feeling of isolation can be pathways to poor health for people in deprived neighbourhood. This follows from the fact that psychosocial factors in our study were not only strongly associated with health but also sufficiently common and unequally distributed favouring the privileged.

To this end, the results illustrate how concurrent socio-economic and psychosocial factors in adulthood are important to understand neighbourhood deprivation inequalities in adult health. As indicated by the analyses in which we estimate the joint contribution of factors from different life periods (model 1–3), however, it becomes apparent that a life course perspective can yield additional insights. When entered separately from later life circumstances in model 1, factors from adolescence explained about 35% of the inequality. By including conditions from young adulthood and midlife in models 2 and 3, however, the joint contribution of adolescent factors was successively reduced, to 18% and then 4%. Although we did not specifically test any of the life course hypotheses, this aligns most closely with the *chain of risk model*, which suggest that one hardship often builds on another (Kuh et al., 2003). Specifically, the results indicated that people's health and psychosocial experiences in adolescence could be a source for what develops into a continuity of poor health and adverse living conditions throughout young adulthood and midlife.

In conclusion, the results shed light on a complex interplay between various determinants, separately at different life periods but also over time. As a result, integrating perspectives such as residential context, inequality and life course, to explain the poorer health of people in deprived neighbourhood seems beneficial. Since the more specific pathways remain unidentified in this study, however, future research should continue to integrate different strands of social epidemiological literature to increase our understanding of social inequalities in health.

4.1. Methodological considerations

The primary strengths of the study include the combination of different literatures, the use of novel methods and the combination of prospective survey and register data extending over several life periods

with a very low attrition (Hammarstrom & Janlert, 2012). Despite this, the paper has a number of limitations.

First, while the study participants were fairly similar to the same age cohort in Sweden overall on a number of demographic measures (Hammarstrom & Janlert, 2012), in other aspects the sample is not as representative. The cohort was closed and initiated when immigration into Sweden was low which means that it is more ethnically homogeneous than the contemporaneous Swedish population. In addition, the participants grew up in a time of occupational instability and experienced a severe economic recession in the early 1990s. Caution should therefore be applied when generalizing the results to other geographical and historical contexts.

Second, in accordance with the 'modifiable area unit problem' (Lloyd, 2014), the concentration index is likely linked to the neighbourhood scale (SAMS units) in the study. However, when comparing three geographical demarcations and their implications for area inequalities in health in the UK, Stafford et al. (2008) found only marginal differences. Moreover, since the independent or mutually adjusted influence of each determinant was estimated in this study, the contribution of factors which are more distal to the inequality have likely been underestimated. In addition, when assessing the implications of neighbourhood socio-economic context on health, related factors at the individual level should be accounted for (Subramanian et al., 2003). Considering we do not have a symmetric individual level control for all neighbourhood indicators (e.g. income and wealth), we nevertheless have similar proxies that are detailed markers of financial situation (e.g. financial strain and low cash margin).

Third, there are continuous methodological developments to study health inequalities. Besides the decomposition analysis developed by Wagstaff et al. (2003) that we have applied, alternative strategies aimed at improving causal inference have recently been suggested. For example, Kessels and Erreygers (2016) use a structural equation modelling approach to develop a two-dimensional decomposition, one which account for the bivariate nature of health inequalities by estimating the covariance between health and the socio-economic indicator. To this, Heckley et al. (2016) introduced a recentered influence function (RIF) regression approach, which allows this covariance to be decomposed into its underlying determinants. Ultimately, the results may differ depending on the specific method applied. By adopting the Wagstaff et al. (2003) approach, which have been used in numerous studies to date (e.g. Hosseinpoor et al., 2006; McGrail et al., 2009; Morasae et al., 2012; Mosquera et al., 2017; Sortsø et al., 2017), we have followed the established method in the field (Heckley et al., 2016) to ensure that our results are comparable with the rest of the health inequality literature.

Fourth, FSS (Zijlema et al., 2013) and neighbourhood disadvantage (Diez Roux et al., 2001) was operationalized in accordance with previous studies, but while neighbourhood disadvantage was based on register data, FSS was self-reported, something that made it impossible to ascertain whether the symptoms were unrelated to an underlying disease. Moreover, on the one hand, area differences in health can possibly be underestimated when the health outcome is self-reported since people in deprived neighbourhoods tend to adjust their expectations to their current situation (Macintyre and Ellaway, 2003). On the other, the differences could potentially be overestimated since people may report poorer health to justify adversity (Black et al., 2017). In the present study, however, FSS is based on the experiences of physical symptoms (e.g. back pain) and should therefore be less dependent on expectations than e.g. self-rated health (SRH).

5. Conclusion

This study suggests that socio-economic and psychosocial factors in midlife are important to understand neighbourhood deprivation inequalities in adult health, but also that the inequality may originate, in part, from the health and living conditions people experience throughout adolescence and young adulthood. By drawing attention to a complex interrelationship between determinants across time and space, it illustrates how the integration of individual, contextual and life course perspectives can be useful in understanding social inequalities in health.

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Declarations of interest

none.

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