The Presence of the Past
A Life Course Approach to the Social Determinants of Health and Health Inequalities in Northern Sweden

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If your only tool is a hammer, all your problems will be nails

– Twain/Maslow/Kaplan/Unknown
Table of Contents

Abstract .............................................................................................................................. iii
Populärvetenskaplig sammanfattning ................................................................. v
List of papers ................................................................................................................ viii
Introducing social and life course epidemiology ..............................................1
   Social determinants of health – a helpful concept for public health? .......... 2
   Social inequalities in health – overlapping with social determinants of health? ... 5
   Social determinants of health, and of health inequalities – separate concepts? ... 9
   The life course approach – adding insights to health and health inequalities? .... 11
   The importance of life periods for health and health inequalities .......... 12
   The importance of life course processes for health and health inequalities .... 14
Previous research in social and life course epidemiology ..........19
   Life course processes – contributing to morbidity and mortality? .............. 19
   Life course socio-economic position – influencing self-reported health? ....... 21
   Life course psychosocial and material factors – affecting self-reported health? ... 23
   Life course determinants – explaining contextual inequalities in health? ....... 25
Rationale and objective .............................................................................................. 26
The setting .................................................................................................................... 28
   Sweden from the early 1980s until the late 2000s............................................ 28
   Northern Sweden .................................................................................................... 28
The health outcome ................................................................................................. 30
   Functional somatic symptoms ......................................................................... 30
Methods ....................................................................................................................... 32
   Participants and procedures ............................................................................. 32
   Data collection ................................................................................................... 33
   Measures .......................................................................................................... 35
   Primary outcomes ............................................................................................. 35
   Main exposures .................................................................................................. 36
   Covariates ......................................................................................................... 41
   Analytical strategy ............................................................................................. 42
      Path analysis .................................................................................................. 42
      Multiple linear regression .......................................................................... 43
      Diagonal reference models ......................................................................... 43
      Decomposition analysis .............................................................................. 44
   Ethical and legal considerations ..................................................................... 45
Results ......................................................................................................................... 46
   The implications of life course social determinants for midlife ill-health ....... 46
   The implications of life course social determinants for midlife health inequalities... 48
Discussion .................................................................................................................... 51
   Social determinants are jointly and independently important..................... 51
   Social determinants of the present appear most important ....................... 53
The past is present and the present is the past.................................................. 54
Methodological considerations ............................................................................. 56
  Issues of design, population and sample ......................................................... 56
  Issues of measurements ..................................................................................... 57
  Issues of analysis and confounding ................................................................... 59
Concluding remarks ..............................................................................................61
  Implications for public health and paths for future research .......................... 62
Acknowledgements ..............................................................................................66
Tack .......................................................................................................................68
References ............................................................................................................ 70
Abstract

Background. Positioned at the intersection between the social and life course epidemiological sub-fields, this thesis builds on the idea that the health implications of life and living conditions can extend over years and decades before becoming expressed in the population patterns of ill-health. The overall purpose was to assess how multiple types of social determinants of health across the life course may contribute to ill-health and health inequalities in midlife. Several gaps in knowledge served as the basis for four research questions that focused on: 1) the intermediate role of socio-economic, material and psychosocial factors in young adulthood, in the long-term association between adolescent socio-economic position and midlife ill-health; 2) the implications of poor social capital in adolescence and accumulated over the life course for midlife ill-health; 3) the consequences of intra-generational social mobility for midlife ill-health and 4) the contribution of socio-economic, material and psychosocial circumstances in adolescence, young adulthood and middle-age to midlife neighbourhood deprivation inequalities in ill-health.

Methods. The setting of the thesis is Sweden spanning over nearly three decades, from the early 1980s and until the mid-2010s. With information drawn from the Northern Swedish Cohort the study population consists of 1,083 pupils (506 girls and 577 boys) who attended, or should have attended, the last year of compulsory school in 1981. The data used came from questionnaires answered by the participants in the follow-ups at the ages of 16 (in 1981), 21 (in 1986), 30 (in 1995) and 42 (in 2007). The attrition rate was low with 1,010 out of the 1,071 students who were alive over the 26-years participated in all waves (94.3%). Data was also included from the Swedish registers for the same ages as the surveys on the participants’ neighbourhoods and sociodemographic characteristics on all other residents in these areas. The health outcome was functional somatic symptoms, referring to the occurrence of common physical complaints such as musculoskeletal pain, headache, palpitations and fatigue. To capture various social determinants of health, socio-economic, material and psychosocial factors were operationalised as main exposures. The research questions were analysed using: 1) path analysis, 2) multiple linear regression, 3) diagonal reference models and 4) a decomposition analysis.

Results. With regard to the four research questions, the results firstly indicated that the long-term association between adolescent socio-economic position and midlife ill-health was linked by socio-economic position in young adulthood and further via material and psychosocial factors in middle-age. Secondly, that poor social capital in adolescence also could play a role in the development of adult ill-health, but that this influence seem to be largely dependent on recent or current
conditions in adulthood. Thirdly, that downward mobility in the socio-economic hierarchy during middle-age may have little to no health implications, while upward movements could have a small positive effect on health. Fourthly, that ill-health was concentrated in more socio-economically deprived neighbourhoods and that this inequality was to a small extent attributed to conditions in earlier life period and mainly to factors in adulthood.

**Conclusions.** Based on patterns cutting across the original research questions, the findings from this thesis indicate broadly that socio-economic, material and psychosocial conditions may be meaningful for midlife ill-health and health disparities, jointly and independently from each other. The results also suggests that determinants in the present on the surface appear to be more important for midlife ill-health and health inequalities than those of the past, but at the same time that life circumstances in the earlier life course may not be irrelevant. Rather than representing permanent or resilient health implications, however, the long-term influence of adolescent conditions seem to reflect mainly social processes that are conditional on recent or concurrent adult factors. In sum, the results indicate that a continuum of various life and living conditions may be a key phenomenon underlying ill-health and health disparities in midlife. Specifically, this thesis illustrates how the past may become part of the present through the accumulation and chains of unfavourable circumstances over the life course and conversely, how the present health reflects and embodies a life-long past.

**Keywords:** social determinants, life course, health inequalities, long-term health consequences, functional somatic symptoms, northern Sweden.
Populärvetenskaplig sammanfattning

Epidemiologi är en vetenskaplig disciplin inriktad mot att studera förekomsten, fördelningen och bestämningsfaktorerna för olika hälso- och sjukdomsrelaterade tillstånd i befolkningen. Denna avhandling är placerad i korsningen mellan och förenar två av dess underliggande kunskapsområden – social epidemiologin och livsloppsepidemiologin. Sammantaget utgör dessa grenar från föreställningen om att socioekonomiska (t.ex. yrke och utbildning), materiella (t.ex. inkomst och bostad) och psykosociala (t.ex. stöd och inflytande) livsomständigheter är viktiga för förekomsten av ohälsa i befolkningen och för dess ojämlik fördelning mellan social grupper. Mer specifikt antas dock att hälsokonsekvenserna av dessa förhållanden inte alltid, nödvändigtvis eller enbart är omedelbara. Bortsett från det faktum att ekonomiska och sociala förutsättningar i familjen under uppväxten kan påverka hälsan hos ungdomar direkt, antas sådana omständigheter kunna forma deras hälsotillstånd även på längre sikt – både avskilt från och tillsammans med senare livsvillkor. Mot denna bakgrund var avhandlingens syfte att studera hur olika livs- och levnadsförhållanden över livsloppet kan bidra till ohälsa och ojämlikheter i hälsa i medelåldern.

En rad kunskapsluckor i den social- och livsloppsepidemiologiska litteraturen låg till grund för fyra specifika forskningsfrågor. Dessa fokuserade på: 1) betydelsen av socioekonomiska, materiella och psykosociala livsvillkor i det tidiga vuxenlivet för relationen mellan socioekonomiska omständigheter i ungdomen och ohälsa i medelåldern; 2) konsekvenserna av bristande socialt kapital i ungdomen och sammantaget över livsloppet för ohälsa i medelåldern; 3) följderna av upptåt- och nedåtgående socioekonomisk mobilitet i vuxen ålder för ohälsa i medelåldern och 4) den grad till vilken ojämlikheter i hälsa mellan bostadsområden kan härledas till underliggande skillnader i socioekonomiska, materiella och psykosociala livs- och levnadsvillkor i ungdomen, det tidiga vuxenlivet och medelåldern.

Deltagare och metod
inhämtats från svenska register. Förekomsten av relativt vanliga fysiska besvär i form av huvud-, muskel- och magväkt, trötthet, sömnsvårigheter, illamående och hjärtklappning – så kallade funktionella somatiska symptom – användes som ett sammantaget mått på ohälsa i avhandlingen. En rad materiella, socioekonomiska och psykosociala livs- och levnadsförhållanden verkade i sin tur som de primära exponeringarna för att förklara förekomsten av och den ojämlika fördelningen i detta hälsoutfall. De statistiska analysmetoder genom vilka forskningsfrågorna studerats inkluderade 1) ’path analysis’, 2) multipel linjär regression, 3) diagonalanalys referens modeller och 4) dekompositionsanalys.

Centrala fynd och slutsatser
Resultaten från denna avhandling visar hur ett komplext samband mellan olika socioekonomiska, materiella och psykosociala levnadsförhållanden, vid separata livsloppspanor men också över tid, kan ha betydelse för utvecklingen av ohälsa (forskningsfrågor 1-3) och ojämlikheter i hälsa (forskningsfråga 4) i medelåldern. Mer specifikt framkom att 1) socioekonomiska problem i ungdomen och senare ohälsa kan sammankopplas genom erfarenheter av materiella och psykosociala svårigheter tidigt i vuxenlivet. Vidare indikerade resultaten att 2) bristande socialt kapital i ungdomen kan spela en viktig roll i utvecklingen av ohälsa i medelåldern, men att ett sådant inflytande till stor del verkar vara beroende av senare eller den sammantagna erfarenheten av bristande social kapital genom livet. Dessutom visade resultaten att 3) nedåtgående mobilitet i den socioekonomiska hierarkin i vuxen ålder inte hade någon hälsoeffekt kunde urskiljas från uppfåtande rörlighet. Slutligen framkom genom resultaten att 4) en ojämlikhet i hälsa mellan olika bostadsområden vilken kunde tillskrivas bakomliggande skillnader i socioekonomiska, psykosociala och materiella villkor i hög grad. Primärt förklarades ojämlikheten av samtida omständigheter i vuxen ålder men även till viss del av levnadsförhållandena i ungdomen och det tidiga vuxenlivet.

I ljuset av ovanstående fynd framhålls genom denna avhandling för det första en genomgripande betydelse av olika socioekonomiska, materiella och psykosociala livsomständigheter för ohälsa samt för ojämlikhet i hälsa. Mer specifikt förefaller samtliga levnadsförhållanden vara viktiga, både tillsammans och oberoende av varandra. För det andra pekar resultaten på styrkan i rådande livsomständigheter i vuxen ålder samt att dessa tycks vara en viktigare källa till ohälsa och ojämlikhet i hälsa i medelåldern än tidigare levnads villkor. Genom att endast göra en ytlig jämförelse kan samtida livsomständigheter således ses som mer väsentliga än de förgångna. Detta representerar dock en förhastad och förenklad slutsats, vilken bortser från viktiga nyanser i resultaten. För det tredje framkom det nämligen hur livsvillkor i ungdomen och i det tidiga vuxenlivet inte bör ses som obetydliga. Istället för att ha direkta och permanenta konsekvenser verkar denna långsiktiga
påverkan dock karaktäriseras av sociala processer där tidiga omständigheter är beroende av och samspevar med senare händelser.

Sammanfattningsvis illustrerar denna avhandling hur en kontinuitet av livs- och levnadsförhållanden – vilka färdas från vårt förflutna till nuet, inte så mycket av kroppen utan genom livet vi lever – till stor del bidrar till utvecklingen av senare ohälsa och ojämlikhet i hälsa. Det intressanta är således inte huruvida händelser i dårدت eller nutid är viktigast, utan hur det förflutna gör sig känt genom sitt formativa inflytande på det nuvarande livet; och omvänd, hur nutiden reflekerar och förkroppsligar ett livslångt förflutet. Detta är problemformuleringar som den aktuella avhandlingen har gett några preliminära svar på.

**Implikationer och riktning för framtida forskning**

Ur ett vidare perspektiv bör resultaten från avhandlingen ses i relation till en ökad förekomst av mental ohälsa och psykosomatiska besvär hos barn och ungdomar i Sverige under de senaste decennierna. Sammantaget visar fynden nämligen hur sådana hälsoproblem hos unga människor kan fortsätta formas över livet genom sociala processer bundna till en kontinuitet av livs- och levnadsvillkor. Eftersom hälsokonsekvenser av förhållanden i ungdomen inte verkade vara direkta eller ökoperatorliga på grundval av avhandlingens resultat bör tidiga insatser inrättas, men inte nödvändigtvis ses som den enda lösningen. Fynden i avhandlingen tyder nämligen på att interventioner senare över livsloppet även skulle kunna minska risken för senare uppkomst av ohälsa och ojämlikhet i hälsa. När det gäller tidiga, förebyggande insatser vilka ämnar till att förbättra unga människors liv bör dessa ses som ett första alternativ då de kan ha både kort- och långsiktiga konsekvenser. På inrådan av den svenska folkhälsomyndigheten samt kommissionen för jämlik hälsa bör första hand skolmiljön och arbetsmarknaden stärkas för att främja hälsan och förbättra livschanserna för ungdomar och unga vuxna. Inom skolans värld bör segregationen mellan skolorna bli mindre, elevhälsovården förbättras samt lärarnas professionella utveckling öka och administrativa bördas. Strategier för sysselsättning och försörjning över det fortsatta livsloppet bör i sin tur öka socialförsäkringsersättnings, förbättra arbets- och anställningsvillkor samt avlägsna hinder för att delta och etablera sig på arbetsmarknaden.

Slutligen, vår samlade kunskap om de livs- och levnadsförhållande som påverkar hälsan och bidrar till ojämlikheter i hälsa grundar sig i dagsläget huvudsakligen på beskrivande epidemiologiska studier vilka sällan visar tydliga vägar framåt. Kommande forskning bör således ägna större uppmärksamhet åt interventioner och policies med syfte att utvärdera vad som fungerar (eller inte) för vem, under vilka omständigheter och hur för att främja hälsan och minska ojämlikheterna i hälsa. Vidare bör forskningen fortsätta i riktningen om att utröna diverse kausala samband. Detta med avsikten att generera kunskapsunderlag som verkligen är folkhälso- och praktiken till gagn.
List of papers

This thesis builds on the following papers, which are specified as I-IV and placed at the end of the thesis.


Introducing social and life course epidemiology

Historically, public health has been particularly concerned with promoting the health of everyone, as indicated by a commitment to increase the average level of health in the population (Verweij & Dawson, 2007). Over the last few decades, however, the scope has broadened along with revived interest in explaining why privileged groups tend to be healthier and live longer than those further down the social ladder (Munthe, 2008). The way through which these social inequalities in health (health inequalities or health disparities) are typically documented is by assessing the extent to which health systematically varies between groups who by some social or socio-economic indicator are seen as more or less disadvantaged (Kawachi, Subramanian & Almeida-Filho, 2002). To this day, public health could therefore be said to have two overall or main goals; improving population health and preventing, reducing, or preferably eliminating health inequalities between a priori defined social strata (Braveman, Krieger & Lynch, 2000).

In terms of providing information to achieve both of these aims, epidemiology – a scientific branch focusing on the occurrence, distribution and determinants of health-related states or events (Porta, 2014) – has major influence on public health policy and practice. For a long time, this discipline has been oriented along with the biomedical model of disease and focused mostly on unhealthy lifestyles and habits (Krieger, 2011a). In response to this reductionist, individualistic and ‘downstream’ attention to biology and behaviours, during the last few decades the field of social epidemiology has evolved (Berkman, Kawachi & Glymour, 2014; Kawachi & Subramanian, 2018). This field has risen more specifically from a need to ‘refocus upstream’ – from individual risk factors to the consequences of social, societal and structural life and living conditions for population patterns of health and disease. As a result, it builds on and includes three partially separate, albeit interrelated, concepts that are central to this thesis: social determinants of health, social inequalities in health and social determinants of health inequalities (Graham, 2004; Solar & Irwin, 2010).

In short, the social determinants of health highlight the consequences of social factors especially with regard to ‘population average health’ (Starfield, 2006). The related concept of social inequalities in health focuses, in turn, on systematic differences in health across strata while recognising that the mechanisms for, and factors underlying, these disparities may vary across the sub-groups. The social determinants of health inequalities then combine the two by making an effort to identify specific social determinants of health that contribute to the disparities.
The ways by which these concepts are more specifically understood and applied in this thesis will be described in more detail below.

During the same time period of the 1980-1990s when social epidemiology was taking shape, the complementary field of life course epidemiology emerged (Ben-Shlomo, Cooper & Kuh, 2016; Ben-Shlomo & Kuh, 2002; Blane, Netuveli & Stone, 2007; Kuh, Ben-Shlomo, Lynch, Hallqvist & Power, 2003). In the early years of development, life course epidemiology was a response to a growing polarisation between epidemiological approaches focused on either prenatal or infant growth or adult lifestyles and behaviours impacting later life ill-health and disease (Ben-Shlomo et al., 2016). Since then, this field has expanded into an integrated study of the ways by which various factors at different levels (e.g., individual, household and context) act independently, cumulatively or synergistically over the life stages to influence adult disease risks (Kuh et al., 2003).

With the brief overview outlined so far as a springboard, the forthcoming sections will be dedicated first to the thesis’s conceptual underpinnings, followed by the empirical research done in areas that are of greatest relevance to the thesis. The conceptual part begins with an introduction to the three social epidemiological concepts just presented: the social determinants of health, social inequalities in health and social determinants of health inequalities. Drawing from the related field of life course epidemiology, a life course perspective on these areas is then integrated, which represents the thesis’s point of departure. In the empirical section thereafter, an overview of what has been the main focus in social and life course epidemiological literature is initially outlined. After that follows a brief presentation of previous research in areas that are especially central to the thesis, albeit that have received less attention in the fields. With the intention of identifying gaps in knowledge, this last part before arriving at the objective, methods, results and discussion thus provides a rationale for the thesis.

Social determinants of health – a helpful concept for public health?

Defined broadly as the ‘conditions in which people are born, grow, work, live and age, and the wider set of forces and systems which influence the situations of daily life’ (WHO, 2011b), social determinants of health has been considered a central concept in the social epidemiological literature (Krieger, 2001). Clearly, this is an all-encompassing, comprehensive and imprecise definition (Graham, 2004). By not making a direct reference to the importance of specific conditions for health, as written now, the health relevance of social determinants has to be considered implicit in the definition.
In theory, a *determinant* of health should reflect a causal relationship in either a positive or negative direction between some experience or exposure and a health-related state or event (Porta, 2014). In terms of being conceptualised as potential causes of ill-health and as predictors of future events, these *determinants* or *determining conditions* should be seen as ‘essential factors (...) which set certain limits or exert certain pressures’ (Krieger, 2001, p. 697). Thus, rather than being deterministic in the sense of a fatalistic determinism where everything is already decided and ‘inevitable’ (Williams, 1983, pp. 99-101), the social determinants of health represent a system that is essentially probabilistic (Porta, 2014).

![Figure 1. The social determinants of health framework describing the conditions that shape population health and health inequalities. Source: amended from Solar and Irwin (2010) and Starfield (2007).](image)

In standard models (see Figure 1), social determinants are typically classified broadly as either structural or intermediary (Solar & Irwin, 2010). Structural determinants refer to societal contexts and mechanisms related to e.g., the labour market, the educational system and the welfare state as well as to cultural norms and political institutions. They are also represented by social or socio-economic positions of groups and individuals that arise from these macro-level processes and according to which populations are stratified along lines of power, money, prestige, coercion and access to resources (Lofters & O’Campo, 2012; Wilkinson, 1999). Although such a distinction is not made explicit in Figure 1, structural determinants of health can be meaningfully assessed through various indicators of social position at different levels. At the individual-level, usually by income, education, occupation, gender or ethnicity and at the contextual-level, typically by residential areas or neighbourhoods.
In turn, intermediary determinants refer to various life circumstances and living conditions that arise from people’s social position as indicated more specifically e.g., by so-called material and psychosocial factors (Solar & Irwin, 2010). Material factors comprise conditions such as residential crowding, poor housing, financial strain, hazardous working environments and occupational insecurities (Moor, Spallek & Richter, 2017). Psychosocial factors instead focus on circumstances related to subjective experiences and social exposures of a more ‘objective’ nature such as e.g., negative life events, lack of support and low control over life that are manifested in interpersonal relations and mediated by psychological mechanisms (Martikainen, Bartley & Lahelma, 2002).

Within this thesis, social capital is one example of a specific psychosocial factor. Notwithstanding that both the meaning and usefulness of the concept of social capital has been questioned (Macinko & Starfield, 2003; Moore, Haines, Hawe & Shiell, 2006; Muntaner, Lynch & Smith, 2001; Navarro, 2004), social capital is henceforth viewed as a psychosocial mechanism in general (Solar & Irwin, 2010) and as an extension of social relations influencing health in particular (Popay, 2000). Following from the idea that such relations can generate health-relevant resources by experiences of participation, support and influence, social capital is perceived as a diverse, age-specific and complex construct that consists of various dimensions (Kawachi, Subramanian & Kim, 2008; McPherson et al., 2014; van der Gaag & Webber, 2008).

In the literature, intermediary material and psychosocial determinants are often treated as conceptually separate (Solar & Irwin, 2010). This follows mainly from them forming the basis of two dominant and partially competing explanations of health inequalities (Arcaya, Arcaya & Subramanian, 2015) – a circumstance that in itself has been the point of departure in lively discussions about their relative importance. Scholars that place a strong emphasis on material conditions have, for example, maintained that attention directed towards psychosocial factors will ignore power imbalances, result in ‘victim blaming’ and be potentially limited to inform public health policy and practice (Lynch, Smith, Kaplan & House, 2000; Muntaner, 2004; Muntaner, Rai, Ng & Chung, 2012; Navarro, 2004). Advocates for psychosocial circumstances have, in turn, stressed that a focus on material standards of living may neither be an adequate explanation nor a sufficient line of action (Marmot, 2004; Marmot & Wilkinson, 2001; Szreter & Woolcock, 2004; Wilkinson, 1999; Wilkinson & Pickett, 2009). Instead, given that the stress that arise from social disadvantage is real and matters, psychosocial factors should be recognised as central, albeit indirect, determinants of health in their own right (Martikainen et al., 2002).

Discussions about the social determinants of health has been fruitful because they recognise the importance of social, structural and societal conditions for health,
while shifting focus away from medical care, behaviours and lifestyles (Krieger, 2011b). Notwithstanding this progress, scholars have emphasised how the social determinants of health framework pays only minor attention to the influences of political, economic and societal contexts for people’s lives (Galea & Link, 2013; Ng & Muntaner, 2014; O’Campo & Dunn, 2012; Raphael, 2006). Specifically, this concept has been criticised for concentrating on ‘middle range’ conditions rather than on ‘macro-level’ processes (Putnam & Galea, 2008). As a result, the public health relevance of the framework and the ability of research to translate into meaningful action has been seriously questioned (Muntaner, Sridharan, Solar & Benach, 2009). This critique builds at least partially on the fact that some social determinants of health – e.g., those building on inter-individual relations – may be difficult to address (Putnam & Galea, 2008). It also follow from the idea that interventions targeting proximal living condition will most likely be less effective to promote population health and reduce health disparities than strategies within the broader political, economic and cultural systems (Ng & Muntaner, 2014).

Through the establishment of the Commission on Social Determinants of Health (2008) by the World Health Organization (WHO) in 2005, the importance of social, societal and structural factors for population health has gained increasing attention globally and nationally. As an example, in 2015 the Swedish government launched a similar Commission for Equity in Health (Dir 2015:60) as part of the goal to eradicate health inequalities within the next generation. In spite of these efforts, interventions intended for altering lifestyles and behaviours have still often been favoured in public health policy over those aimed for social change (Lynch, 2017; Navarro, 2007). Notwithstanding the limitations outlined above, continued conceptual, methodological and empirical research on the social determinants of health therefore remains crucial. This is because now more than ever there is a need to strengthen the view of health and disease as a societal rather than an individual responsibility (Braveman, Egerter & Mok enchaupt, 2011; Marmot, 2015).

Social inequalities in health – overlapping with social determinants of health?

In contrast to the social determinants of health framework, which draws attention to the health implications of a range of social factors, the interrelated term social inequalities in health instead highlights the extent to which health systematically varies by social position (Whitehead, 2007). The concepts of social determinants of health and social inequalities in health thus share both similarities and display differences that seldom and inconsistently are made explicit in the literature. This is why the following section outlines four such aspects. Rather than intending to provide a complete and exhaustive conceptualisation the discussion represents a
presentation of ways through which the frameworks are understood and applied in this thesis.

First, socio-economic position – defined as the social and economic resources that influence the position people and groups hold within the societal structure – represents a central aspect in both the social determinants of health and social inequalities in health frameworks (Lynch & Kaplan, 2000). For this thesis, as well as generally in the field, socio-economic position is usually preferred over similar terms such as class or status (Marmot, 2017). This is because it is generic, neutral-sounding and while it still encompass and may be used interchangeably with class or status, socio-economic position makes no judgement as to the theoretical basis of the concept. Instead, it simply serves to classify individuals along a gradient (Galobardes, Shaw, Lawlor, Davey Smith & Lynch, 2006).

In social epidemiology, indicators such as occupation, education and income at the individual level and neighbourhoods or residential areas at the contextual level, are commonly used to measure socio-economic position (Krieger, Williams & Moss, 1997). Importantly, this concept is a social construct seen as arising from institutions, mechanisms and processes in the political, economic and cultural context (Solar & Irwin, 2010). This means that socio-economic position does not refer simply to people’s education or income, but to a ranking of power, prestige and access to resources which is the reason why it is often seen as a strong marker of social stratification (Lofters & O’Campo, 2012). From the social inequalities in health perspective, socio-economic position has thus been a dominant indicator of health disparities (Graham, 2004) and with research pointing to gaps that are large and persisting (Mackenbach et al., 2008; Marmot, Allen, Bell, Bloomer & Goldblatt, 2012). In the social determinants of health framework, socio-economic position has therefore come to be defined specifically as a structural determinant of health (Solar & Irwin, 2010). Between the social determinants of health and social inequalities in health perspectives, a conceptual overlap can subsequently be identified and where the similarity can be partly attributed to the meaning of socio-economic position in both frameworks.

Secondly, two partially competing explanations focusing on the role of material and psychosocial factors for health disparities has been dominant in the literature (Arcaya et al., 2015; CSDH, 2008). These have risen, in part, from the fact that individuals and groups further down the socio-economic ladder usually face more material (e.g., poor housing situations and hazardous working conditions) and psychosocial (e.g., stressful life events and low control over life) difficulties than their privileged peers (Solar & Irwin, 2010). From a social inequalities in health perspective, health disparities may thus be explained to some degree by socio-economic group’s different exposure to proximal life and living conditions. In the social determinants of health framework, this group of factors correspond with
those that has been labelled as intermediate determinants of health. This means that within both concepts, proximal or intermediary determinants of health are central; as determinants of health in the social determinants of health framework and as explanations of health inequalities within the social inequalities in health framework.

Thirdly, following at least partially from their greater experiences of and exposure to negative life and living conditions, lower socio-economic groups generally face greater obstacles in the realisation of health and more health risks. Based on this notion, it has been argued most of the circumstances influencing their health are not within their power to directly influence and control (Le Grand, 2013). In other words, individuals and groups that are less well-off cannot be seen as having the same opportunities for health as their privileged peers (Asada, Hurley, Norheim & Johri, 2014). The fact that they tend to carry a disproportionately high burden of disease and premature mortality as a subsequent result is a major reason why social inequalities in health to date are generally perceived as unfair and ethically wrong (Arcaya et al., 2015; Kawachi et al., 2002; Le Grand, 2013; Sen, 2002; Whitehead, 1992). With attention directed towards a social inequalities in health perspective, issues centring on fairness thus becomes especially critical. Since the social determinants of health framework is not directly and exclusively concerned with health differences that arise from social stratification, discussions about the unfairness of ill-health and disease does not have the same explicit presence. This is thus another feature according to which the two concepts partly differ.

The fact that social inequalities in health are usually perceived as unethical means that inequality translate into an inequity (Arcaya et al., 2015; Daniels, Kennedy & Kawachi, 1999; Woodward & Kawachi, 2000). The terms health inequality, health disparity and health inequity therefore are often used interchangeably (Braveman, 2014a). Since this is typically a source of confusion, in this thesis the terms inequality or disparity will be applied rather than inequity to align with the broader social epidemiological literature. Implicit still is nevertheless the idea socio-economic differences in health are unfair, tragic and warrant public health attention (Mackenbach, 2012).

Fourthly, underlying the social determinants of health framework is the notion of a hypothesised relationship where social factors are seen as being causally related to a health-related state or event (Porta, 2014). By drawing attention specifically to the social, societal and structural conditions that powerfully shape both health behaviours and health itself, the social determinants of health concept sometimes is referred to as being concerned with ‘the causes of the causes’ (Marmot, 2005). In turn, as presented above, social inequalities in health, health inequalities and health disparities are distributional terms that refer to systematic socio-economic differences in health (Arcaya et al., 2015; Kawachi et al., 2002). When the social
Determinants of health concept thus builds, in part, on causal assumptions, social inequalities in health reflects a population distribution of health where the causal foundation is not necessarily known or assumed (Braveman, Kumanyika, et al., 2011). Given that hypotheses about causality are particularly integral to the social determinants of health framework while not being equally distinct in discussions social inequalities in health, this is another aspect by which the concepts partly differ.

In summary, as constructed for the purpose of this thesis, the social determinants of health and social inequalities in health frameworks differ in at least two ways (shown by the top and bottom arrows in Figure 2). Hypotheses about causality remain more present in the social determinants of health concept while concerns about fairness are somewhat more central when discussing social inequalities in health. As shown with the overlap of the circles (Figure 2), however, on two other accounts the frameworks can be seen as connected and dependent. Structural determinants of health (i.e., socio-economic position) in the social determinants of health framework are indicators of social stratification typically used from a social inequalities in health point of view to assess health inequalities. In turn, within the social determinants of health framework, intermediary determinants of health are seen as factors influencing health while by their unequal distribution between socio-economic groups, they have been as a partial explanation for social inequalities in health.

In light of the fact that there has been large gains in understanding and explaining health inequalities over the last few decades (Arcaya et al., 2015; Bartley, 2017; Kawachi et al., 2002; Link & Phelan, 1995; Mackenbach, 2012, 2016; Marmot et al., 2012; McCartney, Collins & Mackenzie, 2013; Phelan, Link & Tehranifar,
2010; Solar & Irwin, 2010; Starfield, 2007; Øversveen, Rydland, Bambra & Eikemo, 2017) this topic will be discussed in the following section.

**Social determinants of health, and of health inequalities – separate concepts?**

Addressing health inequalities is based on and follows from insights as to how the relationship between socio-economic position and health can best be understood. In this regard, two broad perspectives – the *social causation* and *health selection* hypotheses – which differ mainly with respect to their proposed causal direction between socio-economic position and health, have been dominant within the field (Mackenbach, 2012; Solar & Irwin, 2010; Warren, 2009).

As illustrated up to this point, the *social causation hypothesis* puts emphasis on the importance of social factors for health by postulating that health disparities arise, in part, from socio-economic groups different exposure to e.g., material and psychosocial conditions (Graham, 2009; Solar & Irwin, 2010). In turn, the *health selection hypothesis* – equivalent to a specific example of reverse causality – suggests that health inequalities result from processes where differences in health leads to differences in socio-economic standing (Mackenbach, 2012). People with poor health may, for example, drift downwards on the socio-economic ladder due to challenges of attaining or maintaining a desirable position (McCartney et al., 2013). While ideas about social causation and health selection should generally be seen as complementary (Solar & Irwin, 2010), they have often been treated as at least somewhat competing (Goldman, 1994). With a focus on causal inference, Kröger, Pakpahan and Hoffmann (2015) reviewed 34 studies spanning over 20 years to assess if social causation or health selection better explained health disparities. The authors concluded that both explanations may be at play, but that there seem to be variation in strengths due to different socio-economic indicators and life stages. Their systematic review thus shed light on a potentially reciprocal relationship between socio-economic position and health over the life course.

The social causation perspective also contains further nuances, represented by at least two alternative, but not mutually exclusive, mechanisms that may underlie the disparities. The first posits that health inequalities follow from a *differential effect, susceptibility or vulnerability* to intermediary determinants of health by socio-economic strata (Diderichsen, Evans & Whitehead, 2001; Diderichsen, Hallqvist & Whitehead, 2018; Solar & Irwin, 2010). The second one – which is most central to this thesis – indicates instead that health inequalities arise from a *differential exposure* to intermediate health determinants (Arcaya et al., 2015; Bartley, 2017; Solar & Irwin, 2010). In itself the differential exposure mechanism has two potential sources. First, differential exposure can occur when factors that are more distal to the inequality (e.g., in time or level) influence both health and
socio-economic position. This builds on the idea that adult health disparities may arise because factors early in life – e.g., parental socio-economic position – have the potential to influence socio-economic standing and health status in adulthood (Anderson, 2018; Blane, Davey Smith & Bartley, 1993; Kröger et al., 2015; Warren, 2009). In this regard, the differential exposure explanation corresponds with the so-called *indirect selection hypothesis* (Anderson, 2018; Warren, 2009) and as health inequalities are approached from a life course point of view, indirect selection becomes a central aspect. Secondly, differential exposure can occur also due to concurrent variations between socio-economic groups in their exposure to or experiences of intermediate determinants where these are contemporaneously *unequal* in their distribution.

Building on these two aspects of the differential exposure explanation, strong determinants of health can be expected to matter for corresponding inequalities in health to the extent they also relate to – either by affecting or being affected by – people’s socio-economic position. In circumstances when this occurs, specific *social determinants of health* can thus also be seen as *social determinants of health inequalities* (Dahlgren & Whitehead, 2006).

![Figure 3](image.png)

**Figure 3.** Illustrating the relationship between the social determinants of health and the social determinants of health inequality frameworks, with all dots on the right-hand side of the figure representing social determinants of health, but only those in the upper-right-hand side quadrant being social determinants of health inequalities.

Figure 3 provides an illustration of this relationship with the dots representing different determinants; the horizontal axis displaying the strength of association between the factors and health, moving from strongly related to unrelated. In turn, the vertical axis displays the strength of association between the factors and socio-economic position moving also from largely related to unrelated. Dots that
are located on the right-hand side of Figure 3, i.e., those that are more strongly linked to health, albeit irrespective of their position on the vertical axis, can be perceived as social determinants of health. By contrast, only dots situated in the upper-right-hand quadrant can simultaneously be seen as social determinants of health inequalities. Factors that relate to health but are not, or weakly, associated with socio-economic position (lower-right-hand quadrant) should therefore not systematically be categorised as social determinants of health inequalities.

The distinction between social determinants of health, and of health inequalities has, for some time, not been clearly articulated in broader discussions within the social determinants of health framework (Graham, 2004; Starfield, 2006). There has thus been a call for continued theoretical, conceptual and empirical attempts to identify social determinants of health inequalities, a task that requires a deeper understanding about the role that specific social determinants of health play in the development of health inequalities (Solar & Irwin, 2010).

To provide more insights as to the relationship between the frameworks of social determinants of health, social inequalities in health and social determinants of health inequalities, a life course perspective can be added to these concepts (Ben-Shlomo et al., 2016; Ben-Shlomo & Kuh, 2002; Blane et al., 2007; Kuh et al., 2003). The intention of this latter integrative framework is to further understand how different types of social determinants may contribute to ill-health and health inequalities over the course of life.

**The life course approach – adding insights to health and health inequalities?**

The integration a life course perspective in social epidemiology more generally and in relation to the above detailed concepts in particular, follows from the belief that the social determinants may have not only have instant, but also long-term implications for health (Cable, 2014). The approach also recognises that inherent in particular life periods are risks and opportunities that could potentially shape the health impact of social factors for the rest of the life span (Braveman, 2014b). Specifically, the life course approach builds on the idea that adult ill-health and health inequalities are shaped not only by concurrent factors in adulthood, but also by determinants in earlier life periods (Lynch & Smith, 2005). Based further on the so-called life course models, this strategy may provide insights to the ways by which the determinants can act independently, cumulatively or synergistically over the life course to shape the patterns of adult ill-health (Kuh et al., 2003).

At the intersection of life course and social epidemiological research, focus to date has been mostly directed towards the consequences of structural determinants – specifically socio-economic position – in childhood for cardiovascular morbidity
and mortality in adulthood (Davey Smith, Hart, Blane & Hole, 1998; Galobardes, Davey Smith & Lynch, 2006; Galobardes, Lynch & Davey Smith, 2004, 2008; Kaplan & Salonen, 1990). As a complement to this body of work and in accord with the notion that adolescence constitutes a central life period for the long-term health implications of social determinants (Ben-Shlomo et al., 2016; Sawyer et al., 2012; Viner et al., 2012), this thesis focuses on life throughout adolescence, young adulthood and middle-age.

**The importance of life periods for health and health inequalities**

According to Alwin (2012), life periods represent phases in a sequence of socially constructed and age-related statuses or roles. The sections that follow therefore elaborate on common characteristics of the life periods that are relevant to this thesis.

*Adolescence – the ‘second decade of life’*

According to WHO (2001) adolescence represents the ‘second decade of life’ and covers ages 10 to 19 years and with late adolescence being demarked by age 15 to age 19 (Sawyer et al., 2012). This is a phase of significant social, emotional and physiological change that could be potentially important for later health. At this time of the life course, one typically moves from being a dependent child to a partially autonomous young adult, a shift that creates opportunities for self-governance (Lerner, Brindis, Batanova & Blum, 2018) while also increasing the importance connectedness with and sensitivity to peers and the surrounding neighbourhood environment (Viner et al., 2012). Together with the broader social context, the onset of puberty may also activate and/or support other transitions through the development of various emotional, behavioural and social capacities (Patton & Viner, 2007).

In terms of adolescent health, this is a time when mortality is generally low but where the risk of e.g., injuries, substance use and sexually transmitted infections tend to increase (Gore et al., 2011). In addition, adolescence is also a period when mental disorders like depression and anxiety, and also psychosomatic symptoms or complaints such as different kinds of pains, headaches, dizziness and sleeping difficulties usually present themselves for the first time (Gore et al., 2011; Patel, Flisher, Hetrick & McGorry, 2007). Considering that these latter health problems tend to be more common at this life stage, adolescent mental and psychosomatic ill-health has become an area of concern for public health policy and research internationally (Patton et al., 2016; WHO, 2014) and in Sweden (Hagquist, 2009; Petersen et al., 2010; Public Health Agency of Sweden, 2018a, 2018b; The National Board of Health and Welfare, 2017; Wikman, 2018). This rising interest builds on the indication that mental and psychosomatic problems among young people are growing (Bor, Dean, Najman & Hayatbakhsh, 2014; Collishaw, 2014;
Potrebny, Wiium & Lundegård, 2017; Public Health Agency of Sweden, 2018a, 2018b) but also follow from the belief that improving the life and living conditions of youth is key to ensure the long-term achievement of human potential (Patton et al., 2016).

When it comes to health inequalities, it has been proposed that adolescence – and especially late adolescence – may be a period when socio-economic inequalities attenuate (West, 1997). As indicated through the equalisation hypothesis, social inequalities in health could potentially diminish in adolescence following growing independence, peer pressure and changing cultural exposures (Spencer, 2006), thus giving a certain degree of respite from the pervasive influence of family and parental socio-economic conditions. When examined, the idea about equalisation has received mixed empirical support, with some findings being largely in favour (e.g. Dibben & Popham, 2013) while others bringing about more doubts (e.g. Emerson, Graham & Hatton, 2006; Engström, Laflamme & Diderichsen, 2003; West & Sweeting, 2004). The conclusion nevertheless seems to be that although absolute equalisation appears unlikely, adolescence may still be a period when health disparities become slightly diminished compared with childhood and adulthood (Green, 2013).

By modifying childhood trajectories towards health and by providing a basis for the adaptation of new skills and abilities, adolescence should be recognised as a period when disease processes may be initiated and patterns for future health established (Sawyer et al., 2012). In other words, the challenges that adolescents face may not only have concurrent implications, but may also affect their health and life circumstances in the future.

Young or emerging adulthood – the ‘unsettled’ life period

Rather than being a mere transition to adulthood, the period from ages of 18-19 to about 29 years, when people are no longer adolescents but in many ways not adults either, has recently emerged as a distinct period of the life course (Arnett, 2014). According to Tanner (2011), this follows at least partially from an overall postponement of careers and family as resulting from extended opportunities for post-secondary education. Young adulthood is a period usually characterised by exploration, independence, ambiguity and expectations (Arnett, 2014). Related to preceding and following stages, it is more ‘unsettled’ as indicated for example by frequent changes of residences, relationships and occupations.

At this point, unhealthy behaviours tend be more frequent than in adolescence, while mental and psychosomatic health problems usually continue to be equally common (Tanner, 2011). Resulting from the ‘demographic instability’ of frequent residential moves, emerging adults often spend at least some time under more
disadvantaged life circumstances, e.g., living in a poor residential area (van Ham, Manley, Bailey, Simpson & Macleennan, 2012). In general, however, this is a time when various domains such as life satisfaction and well-being typically improve (Tanner, 2011) and in terms of living in a deprived neighbourhood, this may not be harmful as long as it is temporary (Hedman, Manley, van Ham & Öst, 2015).

Midlife – the ‘afternoon of life’
Midlife, middle-age or mid-adulthood (used interchangeably) has at times been described as the ‘afternoon of life’. In relation to previous periods, this is a long life stage with rather fuzzy boundaries. The common conception is nevertheless that those in middle-age are 40 to 60 years old, but the exact delimitations vary between classifications and an additional 10 years has sometimes been added to either side so that it may also span from approximately ages 30 to 70 (Lachman, 2004).

Midlife has been presented as a rewarding yet challenging period where people are often in the midst of handling multiple roles, including e.g., trying to balance work with family duties (Lachman, Teshale & Agrigoroaei, 2015). By being seen commonly as the providers, the ones to make decisions, have control and reassure stability, middle-aged adults regularly face a variety of demands and expectations (Willis, Martin & Rocke, 2010). Although mid-adulthood has been described also as a period characterised by resilience and plasticity (Lachman et al., 2015), this may nevertheless be a stage when people are especially sensitive to unexpected life changes (Willis et al., 2010). Furthermore, middle-age is a time when many health problems start to surface and is also when social inequalities in health are usually the greatest or at least particularly marked (Adler & Stewart, 2010; Ford, Ecob, Hunt, Macintyre & West, 1994; Merlo et al., 2003). Explanations for this development – i.e., a rise in ill-health and a widening of health inequalities during midlife – can be found at least partially in the so-called life course models (Kuh et al., 2003).

The importance of life course processes for health and health inequalities
Jointly, the life course models propose a number of different, albeit interrelated, processes through which the implications of social determinants over the course of life can become expressed in population patterns of adult ill-health and disease (Ben-Shlomo et al., 2016; Ben-Shlomo & Kuh, 2002; Blane et al., 2007; Kuh et al., 2003). Specifically, time and timing of exposures has been emphasised (Lynch & Smith, 2005).

Focusing on timing builds more generally on the importance of considering when events or experiences occur, but in particular it originates from the premise that
there may be ‘time windows’ when the effects will be magnified and long-lasting (Kuh et al., 2003). This notion has been captured specifically under the *sensitive period model*, which suggests that the health implications of social determinants may be stronger at certain developmental stages (Ben-Shlomo et al., 2016). As illustrated in Figure 4, this means that exposures during the previous life course may have implications for later health that act at least partially independent of exposures during subsequent life periods. This is not a new idea, but something that was initially expressed by the *critical period model*, which formed the basis of Barker and colleagues’ (1993) concerns about biological programming (Ben-Shlomo & Kuh, 2002).

Underlying the original critical period model is the concept of well-demarcated and deterministic ‘windows of opportunity’ within developmental science and the assumption that exposures in infancy and childhood could alter bodily structures, systems or functions. The idea about critical periods thus evolved to propose that conditions in early life may have long-term implications for health, but more importantly, that these effects may not be changed in any dramatic way by later experience (Kuh et al., 2003). Contrary to the critical period hypothesis which suggests partially irreversible effects somewhat deterministically, the sensitive period model acknowledges that the health implications of early exposures may be altered or moderated by conditions later in life (Mishra, Cooper & Kuh, 2010). It is thus usually more appropriate to assume sensitive rather than critical periods when focusing on the long-term health implications of social factors (Halfon et al., 2018).

![Figure 4](image)

**Figure 4.** Sensitive period life course model indicating that exposure to social determinants in adolescence may have implications for health and health inequalities in midlife that act at least partially independent of exposures during subsequent life periods.

Under the broader notion of risk accumulation follows the belief that the more hardship people face the worse their health will be (Kuh et al., 2003). Specifically, the *cumulative risk model* propose that the likelihood of experiencing poor health increases with the duration and/or amount of negative exposures or experiences (Lynch & Smith, 2005). As illustrated in Figure 5, this model somewhat crudely assumes a relationship in which the effect of one detrimental experience simply
adds to the effect of another, without considering if the exposures themselves are related (Bartley, 2017). If interested in the accumulation of a single exposure, the cumulative risk model is equivalent to the conventional epidemiological ‘dose-response model’. This is the reason why accumulation of risk was conceived as the ‘incremental burden of different exposures with age’ when the life course approach was first introduced (Ben-Shlomo et al., 2016, p. 977).

Figure 5. Cumulative risk life course model indicating simplistically the health effect of one detrimental experience adds to the effect of another and that the sum of exposures are essential.

To provide a more detailed account of the cumulative process, the social chain of risk model has evolved. As illustrated in Figure 6, this model assumes that one hardship is often followed by another in a series of unfavourable conditions over the life course (Kuh et al., 2003). It also partially builds on the sensitive period model by acknowledging that negative exposures during certain stages may be the starting point of what develops into a continuity of unfavourable conditions over the life course. These probabilistic chains of disadvantage may then yield poor health in different ways. When each exposure in the risk chain contributes to the health problem at least partially independently of each other, the chain of risk model will be merely a specific and sophisticated version of the cumulative risk model (Ben-Shlomo & Kuh, 2002); particularly, a cumulative risk model in which the relationship and sequence of the factors are taken into consideration. In contrast, when only the last ‘link’ in the chain has a meaningful impact on health, the chain of risk model is instead distinct from the cumulative risk model. This latter case has been labelled a chain of risk with a ‘triggering effect’ (Mishra et al., 2010).

Figure 6. Social chain of risk life course model indicating social determinants may be sequentially related and that there may be health implications of such sequences.
In comparison with the sensitive period, cumulative risk and social chain of risk hypotheses, the social mobility model has received less conceptual attention. It is, for example, not mentioned in any of the more seminal life course texts (see Ben-Shlomo et al., 2016; Ben-Shlomo & Kuh, 2002; Kuh et al., 2003). Social mobility has however been acknowledged by certain authors as an important life course life course process in itself (Cable, 2014; Hallqvist, Lynch, Bartley, Lang & Blane, 2004). This follows from fact that with e.g., acquired wealth, educational achievements, stagnating wages or occupational experiences and skills, people can move up or down in the socio-economic order (Atherton, 2016). As illustrated in Figure 7, social mobility refers to movements upward or downward between some hierarchical states, typically socio-economic position. These can be vertical transitions that occur in relation to older family members (inter-generational), but also changes taking place over one’s own adult life span (intra-generational). With regard to health, social mobility can be a process that constrains social inequalities in health when the health of mobile people fall in-between the socio-economic strata that they leave and the one that they join (Blane et al., 2007; Boyle, Norman & Popham, 2009; Claussen, Smits, Naess & Davey Smith, 2005).

![Figure 7. Social mobility life course model indicating that upward and downward movements in the socio-economic hierarchy may have implications for both health and health inequalities.](image)

Besides yielding insights to health disparities (Mackenbach, 2012), the movement between socio-economic strata can also possibly have direct health implications – with effects that can neither be ascribed to the old position nor alleviated by new one. This suspicion is based on the idea that the transition from one socio-economic position to another may be a stressful experience in and by itself. With origins in the work by Sorokin (1959) and also recognised more recently (Destin & Debrosse, 2017; Destin, Rheinschmidt-Same & Richeson, 2017; Newman, 1999; Simandad, 2018), this stress has been understood as a possible consequence from uncertainties that emerge during socio-economic transitions. Such uncertainties may for example include feelings of exclusion, loneliness and isolation arising specifically from having been ‘forced’ out of a context where one feels comfortable while simultaneously facing problems of integration in a new context (Simandad, 2018). It has therefore been suggested that negative implications for health may
appear both for upward and downward movements; both, i.e., to more favourable and to more disadvantaged socio-economic positions (Destin & Debrosse, 2017; Destin et al., 2017; Newman, 1999; Sorokin, 1959).

While being conceptually separate, the life course models can be basically seen as sub-sets of a more general accumulation process (Ben-Shlomo et al., 2016). This builds on the fact that negative (or positive) exposures at sensitive periods may be part of a continued accumulation of disadvantage (or advantage) over the life course; and such cumulative or sequential trajectories could, in turn, increase the likelihood of downward (or upward) mobility (Hallqvist et al., 2004). Along the same vein, for there to be differential degrees of accumulation, some mobility has to occur; and the intensity and/or duration of accumulation will likely depend on the sensitivity of the period when the process was initiated (Ben-Shlomo et al., 2016). Empirically separating the different life course processes from each other therefore is inherently difficult (Hallqvist et al., 2004), but also means that rather than viewing them as mutually exclusive, the life course models should be seen interrelated and complementary (Ben-Shlomo et al., 2016).

The above conceptual overview illustrates that adopting a life course approach in social epidemiology can shed light on complex ways through which social factors over successive life stages act independently, cumulatively or jointly to influence adult disease risks (Lynch & Smith, 2005). Since it also could provide insights into social groups’ potentially different life trajectories (Øversveen et al., 2017), this framework been seen as central for understanding the development of adult ill-health, but also for health inequalities (Arcaya et al., 2015; Bartley, 2017; Braveman, 2014b; Øversveen et al., 2017). This echoes the point of departure of the present thesis.
Previous research in social and life course epidemiology

Focus has thus far been directed towards the thesis’s conceptual underpinnings. The theoretical review outlined to this point has highlighted how the social determinants of health, social inequalities in health and social determinants of health inequality concepts complement one another with their slightly different foci. It has also illustrated how the frameworks unite in their emphasis on the importance of considering socio-economic, material and psychosocial conditions to understand the population patterns of health. When adding a life course approach to this discussion, the multifaceted, complex and long-term processes whereby social determinants may partially contribute to ill-health and health inequalities have furthermore been acknowledged.

Building on this conceptual foundation, the next section provides an empirical overview of studies at the intersection of the social and life course epidemiological literature. The first sub-section focuses on areas that have been of special concern for life course scholars. Areas that are especially relevant for this thesis are then outlined, with an emphasis on studies on self-reported health outcomes and the life course influences of i) socio-economic position, ii) psychosocial and material factors and iii) contextual inequalities in health. These last sections function as a basis for identifying gaps in knowledge that the present thesis may contribute to.

Life course processes – contributing to morbidity and mortality?

At the outset, investigations by Barker and colleagues (1995; 1993) on the long-term health implications of exposures in utero and during infancy, or biological programming and the foetal origins of adult disease (Kuh & Ben-Shlomo, 2004) should be seen as a catalyst for the integration of life course perspectives in social epidemiology (Ben-Shlomo et al., 2016; Cable, 2014). This research showed that cardiovascular ill-health and disease was not only a product of current or recent conditions in adulthood, such as lifestyle, but could be rooted in living conditions and life circumstances decades old.

Based on these initial and pioneering studies (Barker, 1995; Barker et al., 1993), early life course research was at least partially driven by an interest in evaluating the relative importance of socio-economic circumstances in childhood versus adulthood for later health. At this time, a central problem was therefore whether the past or the present – as a binary of two distinct and disconnected periods – mattered the most for adult health (e.g., as illustrated in the title ‘Past or present?’
(Rahkonen, Lahelma & Huuhka, 1997). Building on and inspired by this idea, the life course field expanded rapidly, albeit largely within the scope of consequences of socio-economic conditions in childhood and adulthood for later cardiovascular disease as well as all-cause, cause-specific and cardiovascular mortality (Cohen, Janicki-Deverts, Chen & Matthews, 2010). Against this background, Galobardes and colleagues (2004, 2008) concluded in two systematic reviews that people from socio-economically disadvantaged families have a higher risk of premature mortality overall and also from specific causes such as stomach cancer and stroke later in life. Similar results were found in systematic reviews by Pollitt, Rose and Kaufman (2005) and Galobardes et al. (2006) which showed that socio-economic circumstances in childhood also were related to adult cardiovascular disease and related risk factors, at least partly independent of adult socio-economic position. When examining the implications of socio-economic position in childhood and adulthood for mortality due to circulatory diseases in a representative Swedish birth cohort, Mishra, Chiesa, Goodman, De Stavola and Koupil (2013) found strongest support for idea that childhood may be a sensitive period to health impact of socio-economic disadvantage.

In accordance with the sensitive period model, the above results indicate that the consequences of early socio-economic disadvantage for different adult outcomes may be partially direct, i.e., that they may only be altered or modified to a certain degree by exposures over the subsequent life course. Apart from these findings, many studies have suggested that the risks of disease and death in later life arise from the joint contribution of socio-economic circumstances in childhood and adulthood. In this regard, studies have commonly used one of two approaches depending on whether the cumulative risk or the chain or risk hypotheses were of interest.

When assessing the implications of cumulative processes for adult health, the common strategy has been to summarise the amount of times people have been in a disadvantaged socio-economic situation. Here, studies generally point to a graded association between the number of times in a low socio-economic position and later cardiovascular morbidity and mortality (Davey Smith & Hart, 2002; Davey Smith, Hart, Blane, Gillis & Hawthorne, 1997; Rosvall, Chaix, Lynch, Lindström & Merlo, 2006). With the other approach corresponding more closely with the chain of risk model, socio-economic position in childhood and adulthood have typically been added separately and successively (i.e., first from childhood and then adulthood). Using this strategy, the long-term association between early socio-economic disadvantage and later cardiovascular outcomes often is partially to largely attenuated by the addition of adult indicators – thus indicating that the relationship may be explained both by social chains of risk and a sensitive period (Claussen, Davey Smith & Thelle, 2003; Falkstedt, Lundberg & Hemmingsson, 2011; Kilpi, Silventoinen, Konttinen & Martikainen, 2017; Strand & Kunst, 2007).
Considering that socio-economic position may not be constant over the life course (Galobardes, Shaw, Lawlor, Lynch & Davey Smith, 2006a), transitions within the socio-economic hierarchy could possibly have implications for health (Simandan, 2018) and for health inequalities (Blane et al., 2007). The issue of social mobility has therefore received some attention in the life course literature, although to a lesser extent than the previously outlined life course models. By comparing health outcomes between groups by combinations of their socio-economic position of origin and destination or socio-economic trajectories in adulthood, studies have suggested for example that mobile people have a higher risk of all-cause mortality than those who remain stable in the highest strata (Nilsson, Nilsson, Östergren & Berglund, 2005; Rosvall et al., 2006). This illustrates how experiences of different socio-economic strata may be more important for health than being immobile, but not whether the movement itself has implications for health.

The social life course epidemiological literature to date suggests that there may be implications of early socio-economic disadvantage for health that adult socio-economic attainment cannot fully account for; and also some health implications of socio-economic transitions. Moreover, although one’s socio-economic position may matter for adult health at different life periods, the cumulative burden of socio-economic disadvantage throughout the life span seem to nevertheless have greater health consequences than either childhood or adulthood socio-economic conditions would have alone. Finally, the importance of social determinants for self-rated health outcomes over the life course has received comparatively little attention as the majority of research has focused on cardiovascular morbidity and mortality. As such and since self-reported health is the focus of the present thesis, pertinent life course research will be presented in the following section.

**Life course socio-economic position – influencing self-reported health?**

When it comes to research examining the role of socio-economic position at the individual-level over the life course for adult self-reported health outcomes, the number of studies are fewer than those concerned with cardiovascular outcomes, albeit not absent.

In their systematic review, Niedzwiedz, Katikireddi, Pell and Mitchell (2012) found 12 studies. These focused specifically on the long-term influence of socio-economic position according to one or more of the life course models for different outcomes all capturing facets of quality of life in adulthood. With regard to the implications of socio-economic position in childhood and adulthood for adult health consistent with the sensitive period and chain of risk models, the results were mixed. For example, two studies in the systematic review (Niedzwiedz et al., 2012) pointed in opposite directions while using the same dataset, albeit different
methods. Mäkinen, Laaksonen, Lahelma and Rahkonen (2006) found that low socio-economic position in childhood was related to poorer mental functioning in adulthood at least partially independent of adult socio-economic position, thus offering support for a sensitive period as well as a possible social chain of risk. By contrast, Laaksonen et al. (2007) found no such association, but rather that mental functioning was poorer among those with higher socio-economic position in adulthood. When examining the implications of socio-economic position in childhood and adulthood for later self-rated health in four European contexts, Hyde, Jakub, Melchior, Van Oort and Weyers (2006) also found mixed results. In women, for example, poor self-rated health was associated with low socio-economic position in childhood and adulthood in England and the Netherlands (sensitive period and social chains of risk), while only with low socio-economic position in childhood in Germany (sensitive period) and neither in childhood nor adulthood socio-economic position in France.

The evidence underlying the cumulative risk hypothesis was more consistent in the same systematic review (Niedzwiedz et al., 2012). Two studies indicated that an increased exposure to low socio-economic position over the life course was related to a decrease in health-related quality of life in a graded manner; for both men and women (Otero-Rodríguez et al., 2011), or only in men (Singh-Manoux, Ferrie, Chandola & Marmot, 2004). Similar findings have appeared in other studies, suggesting that accumulation of socio-economic disadvantage may relate to functional somatic symptoms only in men (Gustafsson, Hammarström & San Sebastian, 2015) and to poor psychological health (Lindström, Fridh & Rosvall, 2014) as well as general self-rated health (Lindström, Hansen & Rosvall, 2012) in both men and women.

Support for intra-generational mobility was also inconsistent in the mentioned review (Niedzwiedz et al., 2012). When using the same approach of comparing health outcomes between groups by combinations of their origin and destination socio-economic position, people moving upward generally reported better health than those who remain stable (Breeze et al., 2001). In terms of assessing if this was due to the movement itself, or whether the health consequences of mobility should be seen as a result of the alternating socio-economic contexts (Simandan, 2018), only one study have examined this idea for self-reported outcomes (Houle, 2011). Based on a method that made it possible to disentangle the health impact of moving between two strata from those of belonging to them over time (van der Waal, Daenekindt & de Koster, 2017), the study found no support for mobility having implications for health in itself. The health of mobile people seemed to instead be a result of an adaptation and re-socialisation into the socio-economic context in the strata they joined (Houle, 2011).
Although the body of research generally points in the direction of a relationship between socio-economic positions over the life course and later self-reported health outcomes, the findings are generally mixed (Hyde et al., 2006; Niedzwiedz et al., 2012). The evidence base seems to mostly support the cumulative risk and chain of risk models, be inconsistent for the notion that childhood is a sensitive period and weak (albeit also less studied) for a health impact of social mobility.

Thus far, attention has been directed toward the health implications of structural determinants – i.e., socio-economic position – in childhood and adulthood. Adult socio-economic position is, however, not the only pathway through which early socio-economic disadvantage may influence later self-reported health outcomes. Instead, as outlined in the initial conceptual section, structural determinants also have the potential to impact on health through various material and psychosocial factors (Solar & Irwin, 2010). Accordingly, in the long-term relationship between early socio-economic conditions and later health, it is possible that material and psychosocial conditions also can act as mediators (Adler & Newman, 2002; Kuh, Power, Blane & Bartley, 2004). Moreover, psychosocial determinants in general (Martikainen et al., 2002) and social capital in particular have been seen as key factors with implications for health in their own right (Kawachi & Subramanian, 2018). The following section therefore focuses on the life course implications of material and psychosocial factors for self-reported health outcomes in adulthood.

**Life course psychosocial and material factors – affecting self-reported health?**

The life course role of psychosocial and material conditions for adult self-reported health can be studied from different perspectives; either by focusing on the health implications of these determinants in their own right, e.g., based on the sensitive period or cumulative risk models, or with the determinants acting as intervening variables according to the social chain of risk model.

As described in the previous sections, examining plausible chains of risk typically involves the inclusion of adult socio-economic position when examining the long-term association between childhood socio-economic position and self-reported and cardiovascular outcomes in adulthood. Similar to this strategy, the mediating role of material and/or psychosocial conditions is usually captured by adjusting for these factors in the association between early disadvantage and later health, and at times with childhood factors being retrospectively collected (Gustafsson et al., 2015; McKenzie, Carter, Blakely & Ivory, 2011; Nicholson, Bobak, Murphy, Rose & Marmot, 2005). If the association becomes wholly or partially attenuated by such an inclusion, this has been typically seen as evidence supporting the idea of social chains of risks. Studies using other techniques to get an estimate of the indirect effects such as, e.g., path analysis, usually draw similar conclusions. For
example, Kendig, Loh, O’Loughlin, Byles and Nazroo (2016) discovered that the association between socio-economic position of the parents and adult quality of life was mediated by one’s own household income in adulthood. Apart from this research, which has also been based mainly on a retrospective recall of childhood factors, few studies have estimated if early socio-economic disadvantage may act as a starting-point of what grows to a continuity of poor material and psychosocial conditions over the life course.

Building further on the relevance of psychosocial conditions for health (Marmot, 2004; Marmot & Wilkinson, 2001; Szreter & Woolcock, 2004; Wilkinson & Pickett, 2009), a number of studies have appeared in the last decades, indicating that the complex construct of social capital may be important for health (Moore & Kawachi, 2017). Defined as a psychosocial determinant at the individual level (Solar & Irwin, 2010), it has been hypothesised that higher levels of social capital can create opportunities for health-relevant resources such as support, influence and engagement. Following this line of thought, research has indicated that more social capital may improve health and/or buffer against ill-health (Almedom & Glandon, 2008; Ehsan & De Silva, 2015; Gilbert, Quinn, Goodman, Butler & Wallace, 2013; McPherson et al., 2014; Nieminen et al., 2010).

Most studies to date have focused on the health benefits of social capital, although it may also have downsides (Moore & Kawachi, 2017). The negative consequences could reflect a lack of or an exclusion from health-promoting resources and/or a health damaging process in itself as arising from demands, pressure or sociability in an undesirable direction (Portes, 1998). The above findings typically apply to children and adolescents (McPherson et al., 2014) as well as adults (Nieminen et al., 2010). The life course relationship between poor social capital earlier in life periods and later self-reported health outcomes has, however, only sparsely been studied (Kawachi & Berkman, 2014). The exception being the study by Landstedt, Almquist, Eriksson and Hammarström (2016) which revealed that youth civic engagement (as an indicator of social capital) was negatively related to depressive symptoms in adulthood, albeit weakly and in men only.

When it comes to research focusing on the life course implications of material and psychosocial factors, little attention has so far been paid to the intermediate role of these circumstances in the long-term association between early or adolescent socio-economic position and adult self-assessed health. Furthermore, research concerned with psychosocial conditions more specifically have mainly assumed a contemporaneous relationship between social capital and health; and although prospective studies do exist, they have been largely directed towards the adult life course (Eriksson & Ng, 2015; Verhaeghe & Tampubolon, 2012).
Life course determinants – explaining contextual inequalities in health?

Building on the literature presented thus far, recent or concurrent determinants in adulthood as well as ones from the earlier life course may contribute to the development of adult ill-health and disease. When assessing the extent to which social determinants of health can also be considered social determinants of health inequalities, research has been more limited (Dahlgren & Whitehead, 2006). This absence has occurred even though the magnitude, ubiquity and population trends of social inequalities in health have received much empirical attention. In this field, however, there has been a predominant focus on health disparities at the individual-level, with attempts to explain them being mostly contemporaneous (Hosseinpoor et al., 2006; McGrail, van Doorslaer, Ross & Sanmartín, 2009; Morasae et al., 2012; Sortso, Lauridsen, Emneus, Green & Jensen, 2017). Based on this gap in knowledge, Mosquera, San Sebastian, Ivarsson, Weinehall and Gustafsson (2017) used a decomposition method to examine the contribution of socio-economic factors in both childhood and adulthood for income inequalities in midlife metabolic syndrome. With a life course approach to health disparities, the authors concluded that about 10% of the disparity was attributed to social conditions in childhood.

Socio-economic inequalities in health exist at contextual levels, such as between neighbourhoods in Sweden (Gustafsson & San Sebastian, 2014; Sundquist, Malmström & Johansson, 2004) and elsewhere (Astell-Burt & Feng, 2015; Green, 2013; Rocha, Ribeiro, Severo, Barros & Fraga, 2017). This has been acknowledged by a large and growing body of ‘neighbourhood effects and health’ research (Diez Roux & Mair, 2010; Oakes, Andrade, Biyoow & Cowan, 2015). Instead of drawing upon a life course perspective to explain the disparities (Næss & Leyland, 2010), however, this field has mainly used cross-sectional data to assess whether the inequality could be attributed to the context of the area, independently from, or in combination with, concurrent individual characteristics (Arcaya et al., 2016). Efforts to explain neighbourhood socio-economic inequalities in adult health are thus lacking. In particular, there is a scarcity of research applying the differential exposure and indirect selection hypotheses to identify life course circumstances that influence people's health and simultaneously relate to their area of residence (Jokela, 2015). In this regard, socio-economic, material and psychosocial factors have been seen as related to adult ill-health and disease (Glymour, Avendano & Kawachi, 2014; Martikainen et al., 2002) and also to neighbourhood of residence (Hedman & van Ham, 2012; Hedman, van Ham & Manley, 2011; Popay et al., 2003). As such, while these conditions could be potentially important to explain contextual health disparities, hardly any studies have assessed their contribution to neighbourhood deprivation inequalities in health.
Rationale and objective

In the social and life course epidemiological literature, there has been a vast focus on cardiovascular disease and mortality, largely at the expense of mental and self-reported health outcomes. In addition, while researchers have been very much focused on and concerned with the long-term implications of negative conditions in childhood for adult ill-health, less attention has been paid to life periods in-between childhood and middle-age. Interested nevertheless in the independent, cumulative and/or sequential relationship between early life circumstances and later health, a common strategy has been the application of a retrospective recall of earlier exposures and not a prospective retrieval. Notwithstanding that our understanding about processes and pathways through which social factors may contribute to ill-health and health inequalities over the life course has markedly grown in the last few decades, much remains empirically unknown and unclear. Building on the above synthesis of previous research, the present thesis therefore seeks to address the following knowledge gaps that have been identified in the social and life course epidemiological fields;

Firstly, conceptual models and empirical research have proposed that the long-term health impact of early socio-economic disadvantage may at least partially be direct, but also that the effect may run indirectly through different intermediate conditions. Insofar as studies have outlined the complex pathways, stretching over decades, by which early disadvantage can become expressed in adult ill-health, this has so far been only sparsely considered.

Secondly, the relationship between social capital and health has gained much empirical attention. Overwhelming evidence has thus emerged, indicating that social capital in the present does shape our contemporaneous health. The extent to which this aspect of social life also has the persistence and power to shape health decades later is nevertheless in need of more attention.

Thirdly, studies have focused on the consequences of socio-economic transitions in adulthood or intra-generational mobility for health, indicating that perhaps the direction of change could be important. Although it is believed that upward and downward movements in the socio-economic hierarchy may be experiences with implications for health in their own right, little is known about whether the health impact of mobility is due to the movement itself.

Fourthly, as indicated by the presence of residential variations in ill-health, it has been generally recognised that health disparities exist not only at the individual level but also across neighbourhoods. So far, attempts to explain this inequality has been mainly contemporaneous and with little attention being paid to the life
course perspective. The extent to which *adult socio-economic inequalities in ill-health at the contextual level* are shaped by *social determinants in the previous life course* and at both *individual- and contextual-levels*, is thus yet to be studied.

Building on these gaps in knowledge, the overall purpose of this thesis was to contribute to our understanding about how multiple types of social determinants of health throughout the life course may contribute to adult ill-health and health inequalities. This work was guided by two thematic areas referred to as ‘life course determinants and adult health’ (A) and ‘life course determinants and adult health inequalities’ (B) throughout the thesis and these were further specified through four research questions (1-4):

A. To assess the importance of social determinants from across the life course for *ill-health* in midlife, specifically:

1) What is the intermediate role of socio-economic, material and psychosocial conditions in young adulthood, in the long-term association between adolescent socio-economic disadvantage and midlife ill-health?

2) What are the implications of poor social capital in adolescence and accumulated over the life course for midlife ill-health?

3) What are the health consequences of intra-generational social mobility for midlife ill-health?

B. To assess the significance of social determinants from across the life course for contextual *health inequalities* in midlife, specifically:

4) What is the contribution of socio-economic, material and psychosocial factors in adolescence, young adulthood and middle-age to midlife neighbourhood deprivation inequalities in ill-health?
The setting

The following section outlines the historical, geographical and cultural contexts in which the population of present thesis is situated.

Sweden from the early 1980s until the late 2000s

The present thesis is positioned in Sweden across almost three decades, from the early 1980s and until the mid-2010s. As indicated by Sweden’s historical focus on, for example, redistribution, free education, unionisation, income protection, generous unemployment and social security benefits as well as universal health care, this is a country that has been generally regarded as a strong welfare state. Since well-developed welfare systems may reduce the number of people living in severe disadvantage, health inequalities can be expected to be smaller here than in other settings (Mackenbach, 2012).

Despite these comparatively favourable preconditions, Sweden is not a country free of challenges. In the early 1990s, a severe economic crisis struck the nation thereby increasing unemployment for a few years (Palme et al., 2002). The same period in the 1980-90s was also marked by increasing income inequalities which has become especially manifest during the 2000s (OECD, 2011). In combination with a contemporaneously intensified re-commodification process, where social security has become gradually more contingent on labour market participation (Papadopoulos, 2005), the welfare system in Sweden has been seriously eroded (Fritzell et al., 2014). The present thesis is thus situated in what Raphael (2013) has described as a ‘declining welfare state’.

Northern Sweden

In particular, this thesis follows a group of adolescents who grew up in a northern Swedish municipality in the early 1980s throughout young adulthood in the late 1980s and until middle-age in 2007 (Hammarström & Janlert, 2012). Northern Sweden, called Norrland in popular speech, covers more than half of the country, but comprises only about 12% of the total population (Statistics Sweden, 2010). With about 4.8 residents per square kilometre, this is a sparsely populated area where people live either in rural areas/small villages in the north-western inland or in slightly larger cities along the south-eastern coast. Geographically, Norrland is divided into the four counties of Norrbotten, Västerbotten, Västernorrland and Jämtland, which each encompass of a number of smaller municipalities. This area is known for its rich nature, the forests, mountains and rivers, which are used not only for fishing and hunting, but also in the larger industries of forestry, energy production and mining.
The initial setting of the thesis is the municipality of Luleå, located along the east coast in the northernmost county of Norrbotten. In 1980, about 66,800 people lived in Luleå municipality, a number that increased to about 73,000 in 2008. The majority of the municipal inhabitants reside in Luleå City, a town founded in 1621 and which became an important commercial centre in the beginning of the twentieth century, focusing on marine and shipyard businesses. In more modern times, Luleå City has been described as an industrial town where metallurgic and steel industries, the IT sector as well as research and higher education have been driving the development. In addition to having a history of higher unemployment levels in the 1980s, Luleå is fairly comparable to Sweden overall on, e.g., labour market, housing and socio-economic structure (Hammarström, 1986). Although the study participants initially lived in northern Sweden, many of them relocated over time. Most of them moved to other municipalities nearby, but some also to other parts of the country.
The health outcome

As a reflection of the interest in exposures within the social epidemiological field (Berkman & Kawachi, 2014), the terms ‘health’ and ‘ill-health’ have thus far been used loosely and without further specification. More specifically this thesis focus on the general and wide ill-health concept which concern self-reported physical or mental symptoms that can sometimes include an underlying disease but not necessarily (Wikman, Marklund & Alexanderson, 2005). To assess the long-term health influence of social determinants, functional somatic symptoms (FSS) are therefore applied as an indicator of ill-health throughout this thesis.

Functional somatic symptoms

Perceived usually as a change towards the worse in terms of health status and/or function, FSS typically presents themselves in the shape of bodily sensations or experiences (Zijlema et al., 2013). More specifically, these are subjective reports of physical complaints such as e.g., musculoskeletal pain, headache, palpitations, nausea and fatigue (Eikelboom, Tak, Roest & Rosmalen, 2016) that can be related to depression and anxiety, but not always (Creed, 2016; Eikelboom et al., 2016).

At the general level, FSS appears in young people (Campo, 2012) as well as adults (Creed, 2016) and in Sweden the increase of somatic symptoms over the last few decades – especially among youth – has been a cause of great concern (Hagquist, 2009; Petersen et al., 2010; Public Health Agency of Sweden, 2018a, 2018b; The National Board of Health and Welfare, 2017). Although the complaints will most often be transient, for those who have many they can potentially persist over the course of life (Campo, 2012; olde Hartman et al., 2009; Steinhausen & Winkler Metzke, 2007). When it comes to adults, FSS tends to be more common in those with a low socio-economic position, while similar patterns are less clear in youth (Halldórsson, Kunst, Köhler & Mackenbach, 2000; Huurre, Rahkonen, Komulainen & Aro, 2005; Ladwig, Marten-Mittag, Formanek & Dammann, 2000; San Sebastian, Hammarström & Gustafsson, 2015).

These are common complaints that occur at short intervals and with low intensity for most people (Ihlebæk, Eriksen & Ursin, 2002; McAteer, Elliott & Hannaford, 2011; Ursin & Eriksen, 2010). When facing intolerable symptoms, however, there seems to be no clear breaking point in the FSS continuum where ‘normal’ levels of tolerable pain can be clearly separated from ‘pathological’ ones requiring medical assistance (Ihlebæk et al., 2002; Ursin & Eriksen, 2010). Rising numbers and/or severity of symptoms nevertheless commonly contributes to functional limitations as well as to decreased health status and health-related quality of life (Bruusgaard, Tschudi-Madsen, Ihlebæk, Kamaleri & Natvig, 2012; Creed, 2016;
Additionally, the occurrence of many symptoms has not only been related to extended utilizations of health care services (Tomenson et al., 2013), it is also a common reason for sickness-related absences (Poulsen et al., 2013; Roelen, Koopmans & Groothoff, 2010).

When attempting to explain FSS, research has acknowledged how the symptoms arise from a complex interplay between the brain, body and environment (Löwe & Gerloff, 2018) as indicated, for example, by a hypothesised disturbance and dysregulation of the stress systems (Kozlowska, 2013). In particular, studies have suggested that FSS may be both a concurrent and long-term response to various social conditions acting at individual and neighbourhood levels (Chapman, 2005; Creed, 2016; Gustafsson et al., 2015; San Sebastian et al., 2015; Tak, Kingma, van Ockenburg, Ormel & Rosmalen, 2015; van Gils, Janssens & Rosmalen, 2014). This suggests that FSS may be an appropriate example of ill-health that is relevant to study across the life course.

Due to its resemblance to and connection with the term ‘medically unexplained symptoms’ (Creed, 2016), the question of whether FSS should be conceptualised as being without or having an unknown pathological origin and thus preferably measured by controlling for possible diagnosis, remains indefinite (Eikelboom et al., 2016). In spite of this ambiguity, jointly with the related concept of ‘subjective health complaints’ (Eriksen & Ihlebæk, 2002), FSS should be seen as a critique towards applications of medically unexplained symptoms since this latter term implies that explanations that cannot be attributed to organic pathology are not part of medicine (Henningsen, 2016; Ursin & Eriksen, 2010). As such, according to Zijlema et al. (2013), FSS should concern complaints in general, regardless of whether they are medically explained or not. Following this line of thought, the extent to which FSS may be manifestations of underlying organic disease is not central to this thesis and therefore not specifically explored.
Methods

In this thesis, different quantitative methods are employed on one common study population to approach the two themes and four research questions. Within this section, key concepts are first presented according to the research questions in which they are addressed (Table 1). Thereafter, the material including both survey and register data are introduced, followed by a description of the measures and analytical methods.

Table 1. Overview of key social determinants, life course models, data source and analytical methods according to the four research questions 1-4.

<table>
<thead>
<tr>
<th>Social determinants</th>
<th>Research question 1</th>
<th>Research question 2</th>
<th>Research question 3</th>
<th>Research question 4</th>
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<tbody>
<tr>
<td>Socio-economic, material and psychosocial factors</td>
<td>Poor social capital</td>
<td>Upward and downward socio-economic transitions</td>
<td>Socio-economic, material and psychosocial factors</td>
<td></td>
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<tr>
<td>Life course models</td>
<td>Sensitive period</td>
<td>Sensitive period</td>
<td>Social mobility</td>
<td>Sensitive period</td>
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<td></td>
<td>Chain of risk</td>
<td>Cumulative risk</td>
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<td>Chain of risk</td>
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<tr>
<td>Data type</td>
<td>Survey</td>
<td>Survey</td>
<td>Survey</td>
<td>Survey and register</td>
</tr>
<tr>
<td>Analysis method</td>
<td>Path analysis</td>
<td>Multiple linear regression</td>
<td>Diagonal Reference Models</td>
<td>Decomposition analysis</td>
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Participants and procedures

The Northern Swedish Cohort (NoSCo) is a prospective longitudinal study that provided information for this thesis and the four consecutive research questions. NoSCo includes all 9\textsuperscript{th} grade school-leavers in the middle-sized municipality of Luleå located in northern Sweden (Hammarström & Janlert, 2012). This means that the study population consists of 1,083 pupils (506 girls and 577 boys) who in 1981 attended, or should have attended, their last year of compulsory school. The cohort has been followed up five times – in 1981 (age 16 years), 1983 (age 18 years), 1986 (age 21 years), 1995 (age 30 years) and 2007 (age 42 years). Over nearly three decades throughout adolescence, young adulthood and middle-age, the attrition rate was extremely low. Out of the 1,083 students who were initially invited, 1,080 chose to participate in 1981. From the original cohort, 1,071 pupils were alive over the whole period and of which 1,010 continued to participate in all follow-ups thereby giving a response rate of 94.3% (Hammarström & Janlert, 2012). The high retention rate was achieved by substantial work directed towards tracing all participants across the years, a task that was facilitated by the Swedish
personal identity numbers. The willingness to participate was also supported by presenting the results to the participants after each data collection.

**Data collection**

The cohort was initiated in the early 1980s by Professor Anne Hammarström within the field of social medicine and she has since then been the PI of all follow-ups. When introduced, the study was a response to the current and comparably high levels of youth unemployment in northern Sweden. Due to a dearth of research on the topic at this time, early work within the cohort focused specifically on the health consequences of youth unemployment (Hammarström & Janlert, 2012).

**Figure 8.** Design of the Northern Swedish Cohort, presenting the time point when and source through which the data has been collected: **Q** = questionnaire, **B** = biological measures, **R** = registers and **NH** = neighbourhood information (also from registers). Data sources in bold are those used in the thesis.

The cohort design is illustrated in Figure 8 which indicates that the data collection has extended across 26 years, with information gathered from different sources at various time points. For the first two waves, information was collected at the participants’ schools, while the later ones were carried out through class reunions in what then was their former schools. Data sources in bold (Figure 8), specifically questionnaires and neighbourhood data (from the Swedish registers), were used in the thesis and is described in more detail below. Apart from this information, biological (B) measures on, for example, blood pressure (ages 16, 21 and 42) and cholesterol levels (age 42) were collected. Register data (R) has also been added at different times, e.g., information from health records at birth and school (age 16) and also various indicators related to labour market participation (annually from 1992 to 2007). Based on class rosters from the schools, sex was measured based on the division of pupils as either boy or girl. More information on the data collection can be found elsewhere (see Hammarström & Janlert, 2012).
Survey
Self-reported information was collected through questionnaires at all five follow-ups in 1981, 1983, 1986, 1995 and 2007. During these years, the participants in the cohort answered an extensive questionnaire consisting of approximately 90 items on e.g., labour market experiences, socio-economic circumstances, health, health behaviours, family and social situation and leisure time (Hammarström & Janlert, 2012). In an effort to include those who could not join the class reunions, questionnaires were sent by mail, followed by a reminder. If they did not reply to either one, an opportunity to answer by telephone was provided. For the present thesis, information from the survey in 1983 is not used.

Neighbourhood register data
In addition to information retrieved through the surveys on health complaints and individual-level living conditions, neighbourhood register data has also been added to the cohort. Specifically, Associate Professor Per E. Gustafsson collected information on the neighbourhoods in which all participants of the last follow-up (n=1,010) resided on 31 December in 1980, 1986, 1995 and 2007 as well as socio-demographic information of all other residents in these areas from Statistics Sweden (Gustafsson, Janlert, Theorell, Westerlund & Hammarström, 2011). When leaving compulsory school in 1981, many members of the cohort moved away from their parental home. To better capture the neighbourhood context of the family, area of residence in 1980 was therefore chosen instead of 1981 as the baseline measurement point (Gustafsson & San Sebastian, 2014).

In social epidemiological research, residential units are commonly characterised by some geographical and administrative demarcation. In the UK and US, often based on census tracts/blocks or wards (Diez Roux & Mair, 2010) and in Sweden, usually on small-area market statistics (SAMS) (Andersson & Musterd, 2010). In this thesis, neighbourhoods are thus defined and operationalised as a SAMS-unit intended to capture what has been generally perceived as a close neighbourhood. The SAMS areas are created as polygons, demarcated by roads or similar physical borders with the intention to group buildings of comparable type and appearance. This means that the areas average about 1,000 individuals (n=979 in 1980 and n=1,400 in 2007) (Gustafsson & San Sebastian, 2014) and due to the moving patterns of the cohort, the number of unique neighbourhoods increased from 72 in 1980 to 215 in 1986 and 374 in 2007.

For the present thesis, neighbourhood register data from 1980, 1986 and 2007 were used jointly with information from the surveys at these years to specifically answer research question 4.
Measures
This section focuses on the measures and their operationalisations, starting with the two primary outcomes, followed by the main exposures and the covariates.

Primary outcomes
Within this thesis, functional somatic symptoms at age 42 were used as the health outcome to address all research questions and with a measure of neighbourhood deprivation at age 42 applied as an inequality indicator also in research question 4. The operationalisations of these measurements are specified below.

Functional somatic symptoms
The main health outcome applied throughout this thesis was functional somatic symptoms (FSS) at age 42. This index was based on a sum of three items that together covered 10 symptoms occurring during last 12 months. The measure was operationalised by a panel of 25 clinicians in areas of child and adult psychiatry, psychology and paediatrics. They were more specifically asked if they considered each symptom in a list of 42 to be classified as FSS or not. Based on their verdicts, the following symptoms were included: 1) headache or migraine (80% of the panel members agreed); 2) stomach ache (other than heartburn, gastritis or gastric ulcer; 96% agreed); 3) nausea (68%); 4) backache, hip pain or sciatica (64%); 5) general tiredness (76%); 6) breathlessness (64%); 7) dizziness (72%); 8) overstrain (64%); 9) sleeping problems (68%) and 10) palpitations (72%) (Hammarström et al., 2016). Before creating the index, all items were recoded to range between no (=0), light (=1) and severe symptoms (=2).

Based on the above symptoms, an index of FSS was created for the age 42 with Cronbach’s alpha being 0.79 thus indicating that the measure displays acceptable to good internal consistency (Hammarström et al., 2016).

Neighbourhood deprivation
For the wave in 2007 when the participants were 42 years of age, a measure of neighbourhood deprivation was also applied as an outcome, or specifically as the socio-economic indicator in the inequality analysis. This variable was measured using register data and aimed at capturing the socio-economic context of the area (Gustafsson & San Sebastian, 2014; Gustafsson et al., 2013). Guided by previous reports (Diez Roux et al., 2001; Matheson, White, Minoeddin, Dunn & Glazier, 2010) and by availability in the registers, the variable was operationalised with the following 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) *unemployment* (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 standardised and the mean across the Z-scores used to get an estimate of neighbourhood socio-economic deprivation. Cronbach’s alpha was 0.86. Importantly, and in contrast to previous studies using the measure to assess ‘neighbourhood effects on health’ (Gustafsson, Bozorgmehr, Hammarström & San Sebastian, 2017; Gustafsson & San Sebastian, 2014), the variable was reverse-coded indicating less deprivation at higher values in this thesis. This was done in order for it to align with the theory and methodology of the inequality analysis (O’Donnell et al., 2008).

**Main exposures**

An overview of the main exposures – the social determinants – are presented in Tables 2 and 3. More generally, these were based on items originating from the Swedish Survey of Living Conditions (Thorslund & Wärneryd, 1985) and the Level of Living Surveys (Johansson, 1970). In order to make the questions age-relevant, different versions of the questionnaire were used at the separate life periods. This means that the indicators or items used differ somewhat between the measures presented below.

**Individual socio-economic position**

With regard to research question 1, socio-economic position was measured based on, and operationalised according to, parental (age 16) and each participant’s own occupation (age 21 and 30 years). The coding of own occupation was done in accordance with the socio-economic classification of Statistics Sweden (1984), with manual workers being defined as low occupation and all other non-manual employees and self-employed persons as high occupation (Gustafsson, Janlert, Theorell & Hammarström, 2010). For some participants at age 21 and 30, there was no current occupation (due to unemployment, studies or military service) and when previous occupation was not accessible, highest educational attainment was used as a proxy (at age 21, n = 206 and at age 30, n = 41). Here, university studies or a high school degree qualifying the person for university studies was considered representative of high occupation, while all other high school degrees or lower levels of education as low occupation. At age 16, the occupation of the parents was used as the indicator of socio-economic position. Based on an older version of the classification scheme (Johansson, 1970), parental occupation was coded into either one of three social groups. Having both parents belonging to ‘social group 3’ (corresponding to manual workers) was defined as low parental
occupation, contrasting to having at least one parent being in the higher social groups as high occupation (Gustafsson, Persson & Hammarström, 2012).

To answer research question 4, separate indicators of socio-economic position at the ages of 16, 21 and 42 years was added to explain neighbourhood-based health inequalities. These included: parental socio-economic position (age 16) and own socio-economic position (ages 21 and 42), all operationalised as above, as well as low education defined as having higher secondary education or less (age 42).

**Intra-generational socio-economic transitions (social mobility)**

Focusing on the health influence of intra-generational social mobility in research question 3, each participant’s self-reported occupation was used as proxies of their ‘prior’ and ‘current’ socio-economic position at the ages of 30 and 42 years. In line with the Erikson, Goldthorpe and Portocarero (2010) scheme (EGP) and based on the socio-economic classification (SEI) of Statistics Sweden (1984), two socio-economic measures with six strata was operationalised. Specifically, SEI category 56, 57 and 60 was classified into the highest strata of professionals (I + II in the EGP classification), SEI 46 in the second one of routine non-manual employees (III) while the third included SEI 79 and 89 or all ‘own account’ workers other than professionals (IVa-c). The fourth strata of assistant non-manual employees (V) was made up of SEI 33 and 36 while SEI 21 and 22 represented the fifth strata of skilled manual workers (VI) and SEI 11 and 12 the last of the unskilled manual workers (VIIa).

Based on these two measures, four mobility variables were then operationalised, aimed at capturing socio-economic transitions between ages 30 and 42. First, a binary variable contrasting between non-mobile (= 0) and mobile (= 1) people. To account for the direction of mobility, three dummy variables were generated. The first aimed at capturing upward mobility by separating upward transitions from those being downward or non-mobile (1 = if upwardly mobile; 0 = other). Similarly, the second dummy variable differentiated downwardly mobile people from upwardly and immobile (1 = if downwardly mobile; 0 = other). The third dummy variable contrasted between people who had not experienced mobility from those being upwardly and downwardly mobile (1 = if non-mobile; 0 = other).

**Material factors**

With reference to research question 1, two cumulative and age-specific material scores were developed at ages 21 and 30 by summarising the following indicators: low income defined as self-reported monthly income below the 20th percentile (age 21); own unemployment (age 21 and 30); low cash margin defined as the inability to raise 5,000 SEK (age 21) and 13,000 SEK within a week (age 30); financial strain as having been forced to withhold from 11 different activities due
to financial reasons over the last 12 months (age 30) and spousal unemployment (age 30).

Table 2. Overview of indicators used for the main exposures – the social determinants – by age (16 and 21) and research question.

<table>
<thead>
<tr>
<th>Age</th>
<th>Social determinants</th>
<th>Research question</th>
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<tbody>
<tr>
<td></td>
<td>Single indicator</td>
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<tr>
<td></td>
<td>Low parental</td>
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<tr>
<td></td>
<td>occupation</td>
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<td></td>
<td>Sum of indicators</td>
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<tr>
<td></td>
<td>1) low participation</td>
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<td></td>
<td>2) low influence</td>
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<td></td>
<td>3) low support</td>
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<th>Age</th>
<th>Social determinants</th>
<th>Research question</th>
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<td>16</td>
<td>Individual socio-economic</td>
<td>Single indicator</td>
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<tr>
<td></td>
<td>Low parental</td>
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<td></td>
<td>occupation</td>
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<tr>
<td></td>
<td>Material</td>
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<td></td>
<td>Psychosocial/</td>
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<td></td>
<td>social capital</td>
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<td>Neighbourhood</td>
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<td>socio-economic</td>
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<th>Age</th>
<th>Social determinants</th>
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<td>16</td>
<td>Individual socio-economic</td>
<td>Single indicator</td>
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<td>Low own</td>
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<td>Psychosocial/</td>
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<tr>
<th>Age</th>
<th>Social determinants</th>
<th>Research question</th>
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<tr>
<td>21</td>
<td>Individual socio-economic</td>
<td>Single indicator</td>
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<tr>
<td></td>
<td>Low own</td>
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<td></td>
<td>occupation</td>
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<td>Material</td>
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<td>Psychosocial/</td>
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<td>social capital</td>
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<td>Neighbourhood</td>
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<td>socio-economic</td>
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<th>Age</th>
<th>Social determinants</th>
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<td>Low own</td>
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<td></td>
<td>occupation</td>
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<td></td>
<td>Separate indicators</td>
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<tr>
<td></td>
<td>1) unemployment</td>
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<td></td>
<td>2) low cash margin</td>
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<th>Age</th>
<th>Social determinants</th>
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<td>Low own</td>
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<td>occupation</td>
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<td></td>
<td>Separate indicators</td>
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<td></td>
<td>1) unemployment</td>
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<td></td>
<td>2) low cash margin</td>
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</table>
To address research question 4, separate material indicators were also included at ages 16, 21 and 42 years to explain neighbourhood-based health inequalities. Specifically, parental unemployment (age 16), own unemployment (age 21 and 42), low cash margin operationalised as above (age 21) and as the inability to raise 15,000 SEK within a week (age 42), financial strain as having been forced to withhold from 11 different activities due to financial reasons during the last 12 months (age 42).

**Psychosocial factors and social capital**

Two cumulative and age-specific psychosocial measures were also developed at the ages of 21 and 30 years to address research question 1 by summarising the follow items in italics. Residential instability referring to the number of moves in the last three years above the 80th percentile (age 21). Illness and death defined as someone close suffering from serious or long-term illness or who had died in the last three years (age 21) and in the last 12 months (age 30). Separation during the last 12 months (age 30). Low decision latitude based on a summary below the 20th percentile of items about the degree to which the respondents work or other main activity 1) involving opportunity to learn new things; 2) requiring a high skill level; 3) requiring creativity; 4) involving repetitive work (reverse coded); 5) involving one’s own influence on how to carry out the work; 6) involving influence on what should be done (age 30). Social isolation based on a summary of the number of people below the 20th percentile: 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 (age 30). Exposure to threat and violence either 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 (age 30).

To answer research question 4, a number of psychosocial indicators were also added separately from the ages of 16, 21 and 42 years to assess their contribution to neighbourhood-based health inequalities. These included the following items in italics. Parental loss either via parental divorce/separation, death or parents never having lived together (age 16). Parental illness defined as having parent(s) with physical illness, mental or alcohol problems (age 16). Low social influence based on a summary of 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 (age 21 and 42). Social isolation being mostly or always alone during the days (age 21) and operationalised as above (age 42). Separation (age 42) and exposure to threat and violence operationalised as above (age 42).
Table 3. Overview of indicators used for the main exposures — the social determinants — by age (30 and 42) and research question.

<table>
<thead>
<tr>
<th>Age</th>
<th>Social determinants</th>
<th>Research question</th>
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</thead>
<tbody>
<tr>
<td>30</td>
<td>Individual socio-economic</td>
<td>Single indicator</td>
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<tr>
<td></td>
<td>Material</td>
<td>Low own occupation</td>
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<tr>
<td></td>
<td></td>
<td>Sum of indicators</td>
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<tr>
<td></td>
<td></td>
<td>1) low cash margin</td>
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<tr>
<td></td>
<td></td>
<td>2) unemployment</td>
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<td></td>
<td></td>
<td>3) financial strain</td>
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<td>4) spousal</td>
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<tr>
<td></td>
<td></td>
<td>unemployment</td>
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<td></td>
<td>Psychosocial/social capital</td>
<td>Sum of indicators</td>
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<tr>
<td></td>
<td></td>
<td>1) illness/death of a</td>
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<td>close one</td>
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<td></td>
<td></td>
<td>2) separation</td>
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<td></td>
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<td>3) low decision latitude</td>
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<td></td>
<td></td>
<td>4) social isolation</td>
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<td></td>
<td></td>
<td>5) exposure to threat and violence</td>
</tr>
<tr>
<td>42</td>
<td>Individual socio-economic</td>
<td>Single indicator</td>
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<tr>
<td></td>
<td>Material</td>
<td>Own occupation</td>
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<td></td>
<td>Psychosocial/social capital</td>
<td>Sum of indicators</td>
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<td></td>
<td></td>
<td>1) low participation</td>
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<td>2) low influence</td>
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<td>3) low support</td>
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<tr>
<td></td>
<td>Neighbourhood socio-economic</td>
<td>Sum of indicators</td>
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<tr>
<td></td>
<td></td>
<td>As at age 16 and 21, but with 7) low education</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8) high education (rev) instead of 7) low occupation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8) high occupation (rev)</td>
</tr>
</tbody>
</table>
To answer research question 2, four age-specific scores referring to poor social capital at the individual level were operationalised at the ages of 16, 21, 30 and 42 years, which were also summarised into one cumulative measure of poor social capital. Based on the data at hand, the items that follow in italics were chosen to represent the concept’s different dimensions. Low social participation defined as seldom being involved in associational activities (ages 16, 21, 30 and 42) and leisure activities (age 16). Low social influence little ability to decide in school and home (age 16) and a summary of items concerning whether the participant could 1) 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 (ages 21, 30 and 42). Low social support defined as having poor contact with mother and father (age 16), mostly or always alone during the day (age 16 and 21) and a summary of items referring 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; 4) the participant can speak openly to; as well as 5) having a special person as supports and is close; 6) persons around to ask for favours and 7) persons that are close outside the family (ages 30 and 42).

The measures for each life period (ages 16, 21, 30 and 42) were summarised based on responses given on a four- to six-level Likert scale and coded from 1-6 with the highest value (6) given to the option mostly related to poor social capital. Each age-specific measure was then standardised before being summarised into a cumulative measure of poor social capital over the life course.

**Neighbourhood deprivation**

To answer research question 4, measures of neighbourhood deprivation at the ages of 16, 21 and 42 years were also added separately to assess their contribution to the neighbourhood health inequality after having been divided into quintiles ranging from high (= 1) to low (= 5) levels of deprivation. The operationalisations of measures at ages 16 and 21 years were similar to the one at the age of 42 (as described above) but included low occupation and high occupation (highest occupation in the household being unskilled manual worker and professional or self-employed, respectively) instead of high and low education because these latter indicators were not available in the registers at the corresponding years. Cronbach’s Alphas were 0.88 (age 21) and 0.93 (age 16).

**Covariates**

The covariates added to the analyses are presented by age and according to the research question in which they are included in Table 4. Specifically, sex was adjusted for in all analyses. Two different measures of previous health status, FSS (age 16 and 21) in analyses corresponding to research questions 1 and 4 and self-rated health (age 30) in to address research question 3. The operationalisation of
FSS was identically to the one at age 42, but with $\alpha = 0.70$ at both age 16 and 21 (Hammarström et al., 2016)

Table 4. Overview of the covariates according to age and the research questions 1-4.

<table>
<thead>
<tr>
<th>Age</th>
<th>Covariates</th>
<th>Research Question</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>16</td>
<td>Sex</td>
<td>X</td>
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<tr>
<td></td>
<td>Previous health status(^a)</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Parental socio-economic position</td>
<td></td>
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<tr>
<td></td>
<td>Material factors cumulative measure</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>Previous health status(^a)</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>Previous health status(^b)</td>
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<tr>
<td></td>
<td>Civil status</td>
<td></td>
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<tr>
<td>42</td>
<td>Children</td>
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<tr>
<td></td>
<td>Civil status</td>
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</tbody>
</table>

\(^a\) Functional somatic symptoms
\(^b\) Self-rated health

Analytical strategy

This thesis applies path analysis, linear regression and diagonal reference models to examine the role of social determinants from across the life course for midlife ill-health (theme ‘life course determinants and adult health’, research questions 1-3). To assess the contribution of social determinants from across the life course for midlife health inequalities (theme ‘life course determinants and adult health inequalities’, research question 4), a decomposition analysis is used. The sections that follow provide a brief overview of these statistical methods.

Path analysis

To assess research question 1, path analysis in Mplus 7 was used to examine the intermediate role of socio-economic, material and psychosocial circumstances in the long-term association between adolescent socio-economic disadvantage and adult ill-health. Specifically, a multiple mediator model (Figure 9) linking socio-economic position at age 16 to FSS at age 42 through socio-economic position, cumulative measures of material and psychosocial factors at ages 21 and 30 were estimated. The model was estimated while controlling for sex and FSS at age 16. Since contemporary measures may share common omitted causes, residuals of variables at ages 21 and 30 were allowed to co-vary (Preacher & Hayes, 2008).
The analysis was performed with percentile bootstrap, a nonparametric re-
sampling technique that generated 95% bootstrapped confidence intervals (CI) and bootstrapped standard errors for the indirect effects (5,000 samples were requested). This approach was adopted to examine the significance of mediation while not imposing the assumption of multivariate normality (Preacher & Hayes, 2008). Using this approach, bootstrap confidence intervals of the indirect effects can be asymmetrical since they are based on an empirical estimation of the sampling distribution, rather than on the notion that the sampling distribution is normal.

![Diagram](attachment:image.png)

**Figure 9.** The multiple mediator model examining the relation between socio-economic position in adolescence (age 16) and functional somatic symptoms in midlife (age 42), via socio-economic position, cumulative material factors and cumulative psychosocial factors in young adulthood (age 21 and 30)

**Multiple linear regression**

Related to research question 2, the analysis consisted of a series of multiple linear regression models that were estimated in SPSS 20. The life course implications of poor social capital in adolescence for midlife ill-health was specifically assessed by examining the association between social capital at age 16 and FSS at age 42 by successively introducing measures of social capital from the ages of 21, 30 and 42. The implications of accumulated social capital over the life course for midlife ill-health were further assessed by estimating the association between cumulative social capital and FSS at age 42. All analyses included sex as a covariate and also socio-economic position, cumulative material factors and FSS at age 16.

**Diagonal reference models**

To examine the health implications of intra-generational social mobility or adult socio-economic transitions in research question 3, diagonal reference models
(DRMs) (Sobel, 1985) were estimated with the *Dref* sub-command of the *gnm* package in R (Turner & Firth, 2008). This method was applied to capture the health consequences of mobility in itself, something that requires that the effect of *moving between* two strata can be separated from those of *belonging to* them over time. This criterion is essential because at a given point in time, a particular strata will logically incorporate both those that have been permanently residing there as well as those that have entered it through mobility. The fact you may be located at a certain position in the socio-economic hierarchy, but either have or have not experienced mobility, is the issue that needs to be solved to capture an effect of change. In this regard, the DRM method is suitable because it allows for a proper adjustment of prior and current socio-economic positions. To do this, the hierarchical socio-economic position variables are re-parameterised as the *estimated* mean FSS levels, not the sample mean (Sobel, 1981), for people who have remained in the same strata between 1995 and 2007. Including these estimations will thus be seen as a control for ‘prior’ and ‘current’ socio-economic positions (van der Waal et al., 2017).

**Decomposition analysis**

With regard to research question 4, the analysis was performed in two steps to assess the contribution of socio-economic, material and psychosocial factors from three life stages to adult neighbourhood deprivation inequalities in health. First, using neighbourhood deprivation at age 42 as the socio-economic indicator and FSS at age 42 as the health outcome, the inequality was estimated with the concentration curve and the corresponding concentration index (Wagstaff, Paci & van Doorslaer, 1991). The concentration curve (CC) is obtained by plotting the cumulative proportion of ill-health (FSS at age 42) against the cumulative proportion of the study population ranked from the most disadvantaged to the least according to the inequality indicator (neighbourhood deprivation at age 42). In terms of interpretation, CC coincides with the diagonal 45-degree line if FSS is equally distributed across neighbourhoods (i.e., there is no inequality). If the CC lies above the diagonal, this means that FSS is higher in deprived neighbourhoods and if the CC lies below this means that FSS is higher in more advantaged areas. 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, Bilger, Sajaia & Lokshin, 2011). Accordingly, C will be zero if CC coincides with the diagonal, positive if CC lies below and negative if CC lies above.

To explain the disparity, the decomposition analysis developed by Wagstaff, van Doorslaer and Watanabe (2003) was used to examine the contribution of socio-economic, material and psychosocial factors in adolescence, young adulthood and midlife to the inequality. More specifically, decomposition analyses were run, in which the concentration index C from the first analysis constituted the dependent
variable (O'Donnell, van Doorslaer, Wagstaff & Lindelow, 2008). Through this method, the inequality C can be attributed to a number of determinants. The influence of each factor is then 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 (i.e., the distribution of the determinant with regard to neighbourhood deprivation at age 42). This method estimates the contribution of the determinants to the inequality independently from each other. The influence can be expressed on the absolute scale, but also (as done in this thesis) on the relative scale as a percentage contribution in the direction of the inequality.

In the decomposition analysis used in this thesis, it is standard procedure to add the inequality indicator (neighbourhood deprivation at age 42) as a determinant when explaining the disparity. This may appear illogical at first, but is important in order to prevent other determinants from picking up its effect. In other words, if this variable was excluded from the decomposition analysis, the contribution of factors that are highly correlated with neighbourhood deprivation in this study could be overestimated or the residuals may be larger (Wagstaff et al., 2011).

**Ethical and legal considerations**

The present thesis was performed in accordance with ethical principles of medical research involving human subjects (World Medical Association, 2013). It was conducted under the auspices of Umeå University (forskningshuvudman) and adhered to the Public Access to Information and Secrecy Act (SFS 2009:400) and the Personal Data Act (SFS 1998:204). Ethical approval was granted for the follow-ups of the NoSCo (Dnr 07-057M) as well as to the complementary neighbourhood measures from Statistics Sweden (Dnr 2011-326-32M) by the Ethical Committee of Uppsala and Umeå University as well as by the Regional Ethical Review Board in Umeå, Sweden.

Measures were taken to protect the rights, privacy and integrity of the study participants. They were ensured confidentiality and informed about the study, its implications, that taking part was voluntary and that they could opt out at any time without having to give a reason. Based on this, the cohort members gave their consent for the data to be used in research. The information provided by the participants further covered topics that can be considered personal and sensitive (e.g., health and family conditions) (Codex, 2018). In the processing, safekeeping and storing of data, the information was therefore handled so it could not be tied directly to any one individual. The personal integrity of the cohort participants was ensured by keeping the questionnaires in locked areas, by working with encrypted datasets and by performing all analyses on coded data, i.e., no individual identifiers (Swedish Data Protection Authority, 2008).
Results

This section presents the main findings where results corresponding to the two themes of the thesis are presented in separate sub-sections. First, with regard to theme ‘life course determinants and adult health’ focusing on research questions 1-3. Then for theme ‘life course determinants and adult health inequalities’ and research question 4.

The implications of life course social determinants for midlife ill-health

The main findings relevant for theme ‘life course determinants and adult health’ are presented in Figure 10. Jointly, these suggest that multiple types of social determinants can have implications for midlife ill-health and that this influence may be partially explained by underlying life course processes.

Corresponding to the first research question, the results from the path analysis indicated that neither family socio-economic conditions in adolescence nor one’s own socio-economic position in young adulthood were independently and directly associated with midlife ill-health. Rather, a significant total indirect effect ($\beta = 0.036$, 95% bootstrapped CI = 0.005, 0.068) was revealed. This suggested that socio-economic, material and psychosocial conditions over the life course to some extent acted intermediate in the long-term association between adolescent socio-economic disadvantage and adult ill-health. Related to the fact that recent material and psychosocial conditions at age 30 were strongly and independently associated with FSS at age 42, the subsequent specific indirect effects pointed to two plausible pathways. The first one through socio-economic position at age 21 and further via material conditions at age 30 ($\beta = 0.009$, 95% bootstrapped CI = 0.001, 0.018). The second one also through socio-economic position at age 21 but then through psychosocial factors at age 30 ($\beta = 0.010$, 95% bootstrapped CI = 0.003, 0.017). As illustrated in Figure 10 under ‘question 1’, the results showed that the long-term relationship between adolescent socio-economic disadvantage and midlife FSS may be mediated by socio-economic position in young adulthood and material and psychosocial factors in middle-age.

With reference to the second research question, the multiple regression analysis suggested that poor social capital in adolescence may have negative, long-term implications for health in midlife. Specifically, social capital at age 16 was related to FSS at age 42, even after social capital at later life periods (ages 21, 30 and 42) had been accounted for ($\beta = 0.107$, 95% CI = 0.044, 0.170). While the further addition of covariates did not alter the findings substantially, the inclusion of baseline FSS at age 16 moderately attenuated the association between adolescent
social capital and midlife FSS, which became borderline significant (β = 0.060, 95% CI = -0.003, 0.123). Throughout these series of analyses, social capital at age 42 was found to be the main independent predictor (β = 0.310, 95% CI =0.242, 0.379 in the fully adjusted model). With regard to the accumulation of poor social capital over the life course for ill-health in midlife, the results indicated that cumulative poor social capital was related to higher FSS levels at age 42, before (β = 0.331, 95% CI = 0.271, 0.392) and after (β = 0.287, 95% CI = 0.226, 0.349) adjusting for sex, socio-economic position, cumulative material factors and FSS at age 16. Jointly, the results (presented under ‘question 2’, Figure 10), indicated that poor social capital in midlife as well as the accumulation of poor social capital over the life course was strongly related to midlife FSS.

**LIFE COURSE DETERMINANTS AND ADULT HEALTH**

**QUESTION 1: SOCIAL CHAIN OF RISK**

- Socio-economic position
- Socio-economic position
- Material
- Psychosocial

β = 0.009*
β = 0.010*

**QUESTION 2: CUMULATIVE RISK & SENSITIVE PERIOD**

- Poor social capital

β = 0.287**
β = 0.060
β = 0.060
β = -0.031
β = -0.031
β = 0.310**

**QUESTION 3: SOCIAL MOBILITY**

- Socio-economic position
- Socio-economic position

B = -0.08*
B = 0.09

**Figure 10.** Presenting the main results corresponding to the theme ‘life course determinants and adult health’ and research questions 1-3 focusing on 1) the social chain of risk life course model, 2) the cumulative risk and adolescence as a sensitive period and 3) social mobility.

* p < 0.05 and ** p < 0.01

When examining the health consequences of adult socio-economic transitions or intra-generational social mobility for FSS in midlife related to research question
three, the results were inconsistent. Compared with being immobile, an upward socio-economic move between ages 30 and 42 was associated with lower levels of FSS in midlife \((B = -0.11, 95\% CI = -0.18, -0.03)\) after adjusting for prior and current socio-economic position. Downward mobility, in turn, although the parameter estimate \((B = 0.08)\) was in the opposite direction thus indicating that a downward move would be associated with higher FSS levels at age 42, this was insignificant \((95\% CI = -0.008, 0.16)\). After adding the covariates, the association between upward mobility and FSS at age 42 was slightly attenuated but remained significant \((B = -0.08, 95\% CI = -0.16, -0.008)\) while the association between downward mobility and FSS stayed borderline significant \((B = 0.09, 95\% CI = -0.002, 0.18)\). As summarised under ‘question 3’ (Figure 10), the results indicated that there was no direct consequences of downward intra-generational social mobility with regard to FSS at age 42, while movements upward seemed to have a small positive effect on FSS.

**The implications of life course social determinants for midlife health inequalities**

Corresponding to the theme ‘life course determinants and health inequalities’ and the fourth research question attempting to explain midlife neighbourhood deprivation inequalities in ill-health, the analysis was performed in two steps.

![Concentration Curve for FSS age 42](image)

**Figure 11.** Concentration curve for cumulative FSS at age 42 by cumulative percentage of the population, ranked by concurrent neighbourhood deprivation with the dashed line above the diagonal line of equity indicating that FSS is concentrated in more socio-economically deprived neighbourhoods.
The results from the initial inequality analysis are presented in Figure 11 where the concentration curve (dashed line) is above the diagonal 45-degree line and the corresponding concentration index small but negative and significant $C = -0.041$ (95% CI: -0.071, -0.011). The findings thus point to a disparity in favour of the privileged since there seem to be a concentration of higher FSS levels within more socio-economically deprived neighbourhoods at age 42.

In the next step, the inequality was decomposed by a stepwise introduction of socio-economic (neighbourhood as well as individual), material and psychosocial determinants at ages 16, 21 and 42. When separately estimating the contribution of adolescent factors, the results suggest that about 35% of the disparity in FSS by neighbourhood deprivation at age 42 was attributed to factors in during this life period. In particular, neighbourhood socio-economic conditions explained about 5%, individual socio-economic position (of the parents) 5.7%, material factors (unemployment of the parents) 1.4% and psychosocial factors (parental loss and illness) an additional 15%. When including determinants from young adulthood, which independently accounted for about 24%, the joint contribution of adolescent factors decreased from 35% to 18%. With regard to the contribution of specific indicators, own socio-economic position in young adulthood explained 3%, psychosocial factors (low social influence and social isolation) about 6.5%, while material factors did not explain anything. In adolescence, after accounting for determinants in young adulthood, neighbourhood socio-economic conditions explained 2% and with the largest contribution being attributed to individual socio-economic position with 8%, followed by parental illness with 4.5%.

As an overall assessment of explanatory strength of the decomposition analysis, the inclusion of factors at all ages and also demographic covariates increased the total proportion of inequality explained to 99%. This indicated that most of the FSS inequality to the disadvantage of people in less affluent neighbourhoods was explained by the determinants included in the study. More specifically, 88.5% out of these 99% was accounted for by conditions in middle-age. Adding these factors reduced importance of adolescent determinants and determinants from young adulthood to about 6% and 4%, respectively. In turn, material factors at age 42 jointly explained the largest share of the inequality with about 28.5% where low cash margin and financial strain contributed with 12.2% and 16.3%, respectively, while there was no influence attributed to one’s own unemployment. Moreover, psychosocial determinants jointly explained 28.3% and of which 13.2% was independently accounted for by low social influence, 6% to harassments and 2.4% to social isolation. Individual socio-economic position, in turn, contributed with 17.1% whereas neighbourhood socio-economic conditions explained 14.6%.

Figure 12 summarises the main results and in which it can be seen that midlife neighbourhood deprivation inequalities in ill-health seem to be mostly explained
by concurrent determinants in midlife – neighbourhood socio-economic (14.6%), individual socio-economic (17.1%), material (28.5%) and psychosocial (28.3%). Adolescent determinants initially explained a fairly large share of the inequality (35%), but contributed only with 5.9% when factors from all life period has been introduced and of which 1.8% was attributed to individual socio-economic factors and 4.1% to psychosocial indicators. The independent and joint contribution of conditions in young adulthood was, in this regard, minor with only 3.5% being explained by psychosocial factors.

**Figure 12.** Presenting the main results corresponding to the ‘life course determinants and health inequalities’ theme and research question 4, focusing on adolescence as a sensitive period and social chains of risk. Specifically, the independent and mutually adjusted contribution of neighbourhood socio-economic, individual socio-economic, material and psychosocial determinants in adolescence (age 16), young adulthood (age 21) and midlife (age 42) to midlife neighbourhood deprivation inequalities in FSS is outlined.
Discussion

Due to methodological limitations when it comes to availability of suitable data and the utilisation of appropriate analytical methods, earlier social and life course epidemiological research have generally considered only two time points, in childhood and adulthood, with little attention paid to adolescence and other life periods in between. This has contributed to a crude and incomplete picture of the health influences of what plays out on the journey from adolescence to adulthood. At the intersection between the social and life course epidemiological literature, the overall purpose of this thesis was therefore to increase our awareness about how multiple types of social determinants of health over the life course contribute to ill-health and health inequalities in midlife.

To do this, certain specific gaps in knowledge served as a basis for two broader a priori themes focusing on the life course implications of social determinants for midlife ill-health and the contribution of these conditions over the life course to contextual health inequalities in midlife, respectively. The results jointly pointed to interplay among different determinants, concurrently and over time, as well as to an interrelation between the life course processes, both with regard to ill-health and health inequalities. The following synthesis and discussion of the findings therefore is structured by patterns cross-cutting the original themes and research questions.

Social determinants are jointly and independently important

At the outset the results broadly suggest that both ill-health and health disparities in midlife may be explained by socio-economic, material and psychosocial factors – contextual health inequalities almost completely and ill-health to a smaller but significant extent. Related to discussions within the literature about the relative importance of structural (socio-economic) and intermediary (psychosocial and material) determinants (Lynch et al., 2000; Marmot & Wilkinson, 2001), the findings from this thesis indicated that they may all be meaningful, together and independently of each other. In other words, these life and living conditions were found to be largely complementary and it seemed as if they would act on health and contribute to health inequalities chiefly in parallel.

One illustrative example of this independent and joint importance comes from the explanation of health inequalities in the fourth research question. Here, the share of neighbourhood-based disparity in health that was separately accounted for by different types of determinants in, e.g., adulthood, was comparatively even (neighbourhood socio-economic, 14.6%; individual socio-economic, 17.1%; material, 28.5%; and psychosocial, 28.3%). When looking more closely at the
independent influence of particular factors, however, a large contribution to the disparity did not follow solely from a strong association with health. For example, adult social isolation was significantly related to ill-health while explaining 2.4% the inequality and low cash margin in midlife which was less strongly associated with ill-health came to explain 12.2% of the inequality. In line with the differential exposure explanation as outlined in the background (Solar & Irwin, 2010), these specific findings appeared because low cash margin – as compared to social isolation – related only weakly to ill-health while still being heavily concentrated to deprived neighbourhoods. The impression that a strong determinant of health will matter for a corresponding inequality in health to the extent it is also related to people’s socio-economic position (in this case the deprivation of one’s area of residence), was therefore supported in this thesis (Dahlgren & Whitehead, 2006). In itself, this result thus reinforces the initial distinction made in the background section between social determinants of health, and of health inequalities.

Apart from highlighting that strong social determinants of health are not always and necessarily strong determinants of health inequalities, the results also shed light on the complementary nature of the different factors. For example, when the joint contributions of determinants in adolescence, young adulthood and middle-age were assessed, the inequality was almost completely explained (99%). This shows that these different factors together largely constitute the underpinnings of the disparity, even when other aspects such as, e.g., health-related behaviours that could possibly partially contribute further downstream, have not been accounted for. The results thus demonstrate how powerful the collection of these different determinants is when it comes to explaining health inequalities.

Another example of this independent and joint importance comes from assessing the long-term association between adolescent socio-economic disadvantage and midlife ill-health in the first research question. Here, the results drew attention to two plausible life course pathways indicating that material and psychosocial factors in midlife – jointly with socio-economic position in young adulthood – may act indirectly en route from early socio-economic circumstances to later ill-health. This finding pointed to two sets of intermediary determinants that have been seen as central in the social epidemiological literature (Solar & Irwin, 2010). By indicating that these pathways may run independently of and in parallel with each other, however, the results contrast partly with the notion that psychosocial conditions are predominantly mediators in the concurrent or long-term relation between socio-economic or material circumstance and health, which is a common claim in the literature (Martikainen et al., 2002; Moor et al., 2017). Nevertheless, while psychosocial determinants are thought to act on health via psychological mechanisms whilst the material ones do not, Marmot and Wilkinson (2001) have stressed that both could bear implications for health through physiological effects of their emotional and social meanings. The results from this thesis thus further
support the impression of materialist and psychosocial explanations not being mutually exclusive, but complementary (Adler, 2006).

As a possible exception to this general picture illustrating the importance of social determinants for ill-health, inconsistent findings appeared when examining the health consequences of adult socio-economic transitions with regard to the third research question. In accordance with previous studies (Niedzwiedz et al., 2012), the results from this thesis highlighted the complexity of intra-generational social mobility and its implications for health. Specifically, the results suggested that upward mobility in the socio-economic hierarchy over the adult life period could be potentially beneficial for health, while downward mobility seemed to be of less importance for midlife health complaints. Rather than unanimously supporting the ideas that upward and downward mobility would have consistently negative consequences for health (Destin & Debrosse, 2017; Destin et al., 2017; Newman, 1999; Sorokin, 1959), the results displayed how movements in opposite directions may constitute separate experiences. The positive influence of upward mobility found in this thesis may, for example, be rooted in psychosocial processes related to a reinforced self-image (Goldthorpe, 1980). In addition, although downward mobility could still be a disruptive and stressful event, the weak association found in this thesis may be a reflection of the Swedish context in which generous social security systems partially buffer against a negative impact of such transitions (Hout & DiPrete, 2006).

Social determinants of the present appear most important

To add more complexity, the results also indicated that socio-economic, material and psychosocial determinants could have both contemporaneous and long-term implications for midlife ill-health and for health disparities. In the background, it was suggested that middle-age can be a life period when people may be sensitive to unexpected life changes due to a shouldering of multiple roles while at the same time facing a variety of demands and expectations (Willis et al., 2010). Consistent with this line of reasoning, throughout the series of analyses where factors from multiple life periods were simultaneously considered, the impact of concurrent or recent conditions in middle-age was dominant for both ill-health and health disparities. Based on the findings from this thesis, on the surface, the present thus appear most important for midlife ill-health and health inequalities.

As an example illustrating the contemporaneous importance of adult factors for ill-health and health disparities, exposure to poor social capital in midlife seemed to play a particularly influential role for concurrent ill-health. In comparison, the independent contribution of poor social capital throughout the earlier life course appeared limited once factors from all life periods and confounders had been considered. Along the same line, the results also indicated that about 90% of the
neighbourhood inequality in health was jointly and independently explained by recent or concurrent socio-economic, material and psychosocial determinants in midlife. Only a minor independent contribution could therefore be attributed to life and living conditions during adolescence and young adulthood in the fully adjusted model. Moreover, when focusing on complex pathways by which early disadvantage could become expressed in adult ill-health, neither socio-economic conditions in adolescence nor young adulthood seemed to have direct and long-term implications for midlife ill-health. Instead, recent material and psychosocial conditions in middle-age was found to make the largest independent contribution to adult health. By drawing attention to a strong contemporaneous role of social determinants in midlife and at the same time a fairly small independent influence of factors in adolescence and young adulthood, the findings from this thesis paint a rather nuanced picture the potential significance of the past life course.

In the background, the binary question shaping early life course epidemiology of whether it is the present or the past that matters for health, was briefly mentioned (Rahkonen et al., 1997). Based on the preceding findings, one could infer from the current thesis that the answer to this issue would definitely be ‘the present’. This would be an erroneous, premature and simplified conclusion, however, one that neglects essential nuances that will be exemplified in the next section.

The past is present and the present is the past

At the general level, the findings from this thesis have demonstrated how socio-economic, psychosocial and material determinants may contribute to midlife ill-health and health inequalities over the life course; but more specifically, that such processes may be primarily explained through an accumulation and/or chains of unfavourable factors over the course of life (Lynch & Smith, 2005).

As an exemplary finding following in line with the cumulative risk model (Kuh et al., 2003), the added burden of poor social capital throughout adolescence, young adulthood and middle-age appeared as an aspect of social life with the persistence and power to yield worse health in midlife. In the same vein, when assessing the influence of socio-economic, material and psychosocial factors in adolescence, young adulthood and middle-age for the neighbourhood-based health inequality in a stepwise manner, the proportion of total inequality explained grew from 35% to 42% and 99%. This result not only stresses the joint role of these determinants when it comes to explaining the disparity, but also suggested that the contribution gradually increased with the successive addition of factors from one period to the next. In addition, the results also indicated that one’s socio-economic position in young adulthood and then material and psychosocial conditions in middle-age may be indirect pathways in the long-term association between adolescent socio-economic circumstances and midlife ill-health. In line with the social chain of risk
model (Kuh et al., 2003), this demonstrated how socio-economic circumstances in adolescence could develop into a continuity of disadvantage throughout young- and mid-adulthood, ultimately shaping health in midlife.

In relation to the conceptual point of departures as laid out in the background, the above notion requires that further attention be paid to nuances in the results. Specifically, throughout the series of different analyses, the association between adolescent social determinants (e.g., poor social capital) and midlife ill-health was generally fairly strong. When social determinants over the subsequent life course and covariates had been accounted for, however, the association generally weakened substantially. Similar results appeared when the neighbourhood-based health inequality was assessed. Specifically, when the independent contributions of adolescent determinants were considered, these factors jointly explained about 35% of the disparity. When determinants in young adulthood and middle-age had been accounted for, however, the remaining contribution was minimal. This can be interpreted as lack of support for the perception of adolescent social conditions resulting in permanent or resilient health effects that endure over the life course as posited by the sensitive period model.

Considering the plasticity inherent in human development and in many specific bodily structures, systems and functions, the absence of permanent or resilient health implications in adolescence may not be all that surprising (Halfon et al., 2018). The fact that adolescence may not be a particularly sensitive period to the health impact of social determinants in this thesis, however, should not be seen as an indication of this life phase being irrelevant. To the contrary, the findings does indeed suggest that conditions at this life stage contribute to ill-health and health disparities decades later, but with the nuance that this long-term influence may mainly reflect a social process rather than an early impact on health.

The presence of the past as illustrated in this thesis seems to be a conditional and not all-encompassing one. As an extension of the social determinants of health framework that does not usually emphasise the life course view, the results from this thesis indicate that the implications of recent or concurrent determinants in adulthood for midlife ill-health and health disparities could be rooted in related conditions throughout the earlier life course. The results thus correspond with and further strengthen the general belief that the lives of young people may shape their health and life chances in the future (Patton et al., 2016; Sawyer et al., 2012; Viner et al., 2012). Rather than having direct and irreversible implications for health in themselves, however, the socio-economic, material and psychosocial challenges that they face seem to represent pieces in a larger puzzle of cumulative and/or chains of unfavourable conditions from adolescence and onwards. This illustrates how the key phenomena for adult ill-health and health inequalities may
be a continuum of life conditions rather than representations of bodily processes or disaggregated life periods that can be considered in opposition to each other.

**Methodological considerations**

The frameworks, concepts, data, measurements and methods that have been applied and adopted throughout this thesis are all subject to both advantages and shortcomings. The sections that follow thus include a discussion about some of the main methodological analytical and conceptual issues, thereby highlighting both strengths and limitations of the thesis.

**Issues of design, population and sample**

An overall strength of the thesis is the combination of prospective, longitudinal questionnaire and register data that have been collected at several points of the life course over almost three decades with low attrition. With information drawn specifically from the NoSCo (Hammarström & Janlert, 2012), a particularly rich dataset was used. Considering that all 9th grade school-leavers in the municipality of Luleå were included in the study and due to the exceptionally high response rate where 94.3% of those alive in the original cohort participated in all follow-ups, the risk of selection bias has been significantly reduced (Porta, 2014). This low level of study drop-out was achieved by a number of concentrated efforts to reach all the participants. For example, the data collection was focused to class reunions and for ones that did not attend, structured telephone interviews were offered as an alternative to facilitate participation.

When it comes to assessing the thesis external validity, the study participants can be seen as fairly similar to the same age cohort in Sweden as a whole on a number of demographic, socio-economic and health-related measures (Hammarström & Janlert, 2012). In addition, the middle-sized industrial municipality and town of Luleå share some features, e.g., the size and proportion of rural/urban population and industries with a typical Swedish town/municipality (Novo, 2000). In other aspects, however, neither can the sample nor the area be seen as representative. For example, the cohort was closed and initiated in a time when immigration to Sweden was low. The study population is thus more ethnically homogeneous than the contemporary one in Sweden today. In addition, the participants grew up in a time of occupational instability, faced rather high levels of youth unemployment at the time of labour-market entry in the mid to late 1980s and then a subsequent economic recession in the early 1990s. All in all, while the mechanisms revealed in this thesis may be relevant in other situations, the aspects just outlined should be considered when attempting to generalise the findings to other geographical or historical contexts.
**Issues of measurements**

This thesis uses longitudinal data based on information that was prospectively and similarly collected through questionnaires at four different time points. Over the years since the cohort was initiated, some items of the questionnaires have been kept intact while others have been adjusted slightly to ensure age-relevance. Importantly, information from the surveys was not collected specifically for the purpose of addressing the questions in this thesis. As such, although the concepts have been grounded in theory and measured based on previous research to the extent that appropriate items have been available, the operationalisations have been based on ‘retro-fitting’ the concepts to existing data, something that offers only limited protection against information bias.

Other than the neighbourhood measures that were constructed with register data, self-reported information has been applied for most operationalisations and with many items including a retrospective recall extending over the last 12 months. This approach has the benefit of providing insights into the participants’ own experiences while at the same time – as in the case of FSS – reducing the risk of capturing short-term fluctuations that are not central for symptom measurement (Zijlema et al., 2013). It can, however, also be a source of bias considering that the participants may have problems remembering things that happened earlier in life (recall bias) and/or report them inaccurately because they feel the need to present themselves in a certain way (social desirability bias). In addition, since both measurements of exposures (social determinants) and health outcome (FSS) were based on self-reported data, their association could have been inaccurately estimated. It may, for example, have been overestimated because the participants reported poorer health to justify hardship (Black, Johnston & Suziedelyte, 2017) and underestimated since those that experience disadvantage may have adjusted their expectations of health to their current situation (Burgard & Chen, 2014). As discussed by Althubaiti (2016), bias due to self-reports are not easily addressed, but something that usually requires efforts of validation, mainly in the design and implementation phase of the study. By protecting the rights, privacy and integrity of the participants at the time of data collection, the risk of social-desirability bias could have been potentially reduced within this thesis (Althubaiti, 2016). When it comes to assessing the other forms of bias, however, these have unfortunately been difficult to assess.

The issue of whether or not measures should be age-specific or kept intact over time is a particular challenge for life course research. For example, the application of age-specific measures may limit comparability, while the utilisation of identical ones could mean that they have different meanings at separate life periods. With regard to the application of FSS, this constitutes neither an established term nor a validated construct as illustrated by debates about its nature, conceptualisation and impact (Voigt et al., 2010). Due to the availability items in the questionnaires,
the health outcome FSS at age 42 was operationalised identically as measures of FSS included as covariates at the ages of 16 and 21. Without disregarding the complexity of the construct as outlined in the background, the operationalisation of FSS in this thesis was similar to other population-based studies (Zijlema et al., 2013). Considering that young people may inhabit different social spaces as well as develop and exploit forms of social connections than are not identical to those of adults (McPherson et al., 2014; Morgan, 2011), psychosocial determinants were operationalised as age-specific concepts in this thesis. Along the same line and since certain indicators of e.g., socio-economic position may be more relevant at one life period than another (Galobardes, Shaw, Lawlor, Lynch & Davey Smith, 2006b), material and socio-economic measures were also treated as age-specific. Items regarding contact with parents, for example, were therefore included in the adolescent social capital measure while not in the ones from adulthood. Similarly, based on data availability, the social groups (Johansson, 1970) of the parents was used as a proxy for adolescent socio-economic position. Related to the discussion about finding suitable, appropriate and agreed-upon indicators at this life stage (West & Sweeting, 2004), it is acknowledged that this represents a crude and inadequate approach.

When measuring determinants that were operationalised as summary indices of multiple questionnaire items (material and psychosocial factors more generally and social capital in particular) internal consistency was not reported e.g., by Cronbach’s Alpha. This was motivated by these measures not being perceived as unidimensional, but as holistic constructs considered to be both theoretically and empirically heterogeneous. Regardless of the clustering of different indicators as indicated by a high internal consistency, together they may still have cumulative implications for health. For example, with regard to psychosocial measures, even if the death of a close one and having low decision latitude are causally unrelated, their co-occurrence would most likely bring about more stress than would either of them singly. Similarly, the theoretical dimensions of social capital may co-vary, but not necessarily. The level of social capital should for example be expected to increase with involvement in associational activities, irrespective of change in the other domains. In accordance with Streiner (2003), although the indicators may jointly contribute to, rather than be manifestations of, the latent construct of social capital, the dimensions should nevertheless act on health through common pathways over the life course.

As debated in the area effects on health literature, defining neighbourhoods using administrative or geographical boundaries (e.g., SAMS-units) may not constitute appropriate demarcations to capture ‘collective bodies’ (Merlo, Ohlsson, Lynch, Chaix & Subramanian, 2009). The results concerning research question four thus are relevant to the extent that SAMS-areas actually represent valid boundaries for the concentration of ill-health by deprived neighbourhoods. With reference to the
relatively small inequality found in this thesis, this could possibly be a reflection of unsuitable demarcations. Furthermore, the definition of areas as SAMS-units was fixed across the years. This facilitates for comparisons but means that the validity of the boundaries may differ over time since, e.g., physical changes in the neighbourhoods have not taken into account. On a similar note, the measures of neighbourhood deprivation were of similar content to other operationalisations (see e.g., Diez Roux et al., 2001; Matheson et al., 2010) but the selection of indicators was nevertheless partially limited by availability in the registers.

**Issues of analysis and confounding**

To assess the role of social determinants over the life course for midlife ill-health, the analytical methods applied included path analysis, multiple linear regression and diagonal reference models, while the contribution of social determinants over the life course to midlife health inequalities was examined with a decomposition analysis.

On the one hand, the current analytical approach where different methods have been used limits the comparability of the results across the thesis. On the other hand, it means that methods suitable to answer the research questions have been applied and also that the analyses could possibly be partially complementary. For example, the independent and mutually adjusted influence of the determinants, concurrently and over time, was only assessed in research questions 2, 3 and 4, while their interrelationship was at least partially examined in research question 1. Similarly, the inclusion of potential confounders was limited to items available in questionnaires which means that the risk of residual confounding is high. In the analysis corresponding to the first research question, this issue was at least partially recognised by allowing residuals of contemporary measures to co-vary (Preacher & Hayes, 2008). In addition, a study population of over 1000 people was considered a high number in the early 1980s. Compared with more recent cohort studies, however, and for some specific methods in this thesis (e.g., the DRM analysis) the size of the sample appear rather small.

With regard specifically to the issue of confounding, a comprehensive theoretical model was developed to examine the first research question. In this analysis, only two factors – baseline FSS and sex – were controlled for that could potentially confound the mediator-outcome association (VanderWeele, 2015). Similarly, for research question 2, it was possible to adjust for some potential confounders, e.g., socio-economic position (Harpham, Grant & Thomas, 2002), when focusing on the association between poor social capital over the life course for midlife ill-health. Other confounding factors that may exist in the shared environment of the household were not (Giordano, Merlo, Ohlsson, Rosvall & Lindström, 2013).
When examining the health implications of intra-generational social mobility, it was not possible to account for the time people spent in different socio-economic positions (Houle, 2011), nor to assess why people moved up or down in the socio-economic hierarchy, e.g., whether the transition was by choice or involuntary, or took place within or between organisations. Although a set of control variables related to mobility and FSS were included (e.g., parental socio-economic position, sex and education), selection mechanisms that could possibly act as a partial explanation were therefore likely overlooked. Furthermore, when examining the influence of neighbourhood deprivation for health, related determinants at the individual-level should be accounted for (Subramanian, Jones & Duncan, 2003). With regard to this issue, symmetric individual-level controls were not available for all neighbourhood indicators (e.g., income and wealth) while similar proxies that can be considered detailed markers of economic situation were included (e.g., financial strain and low cash margin).

Throughout the series of different analyses, efforts have been made to anchor the operationalisations in theory while adjusting for confounders to the extent that appropriate variables were available in the material. In spite of these attempts, the likelihood that important measures have been omitted and the fact that the latent features of some concepts have not been specifically considered, limits the study. In addition, while one strength of the thesis is the application of methods suitable to answer the different research questions, each area of analysis undergo continuous methodological developments. Aimed at improving causal inference new approaches have, for example, evolved within the fields of mediation (Imai, Keele & Tingley, 2010; VanderWeele, 2015; Wang, Nelson & Albert, 2013) and decomposition analysis (Heckley, Gerdtham & Kjellsson, 2016; Kessels & Erreygers, 2016). As a result, considering that none of these newer methods have been implemented and since the risk of residual confounding is high, based on the findings from this study causal interpretations should be done cautiously.
Concluding remarks

The results from this thesis focusing on how multiple types of social determinants of health contributed to ill-health and health inequalities in midlife over the life course paint a multifaceted picture. Specifically, the findings draw attention to a complex interplay across different determinants, contemporaneously as well as over time, and to an interrelation between life course processes of importance for both ill-health and health inequalities in adulthood. Despite this diversity, there are some broader conclusions that cut across the original themes and research questions by which the results can be summarised as follows:

Firstly, the results highlight the pervasive importance socio-economic, material and psychosocial determinants for ill-health and health disparities in midlife and shed light on the fact that these different factors may all be meaningful, together as well as independently of each other.

Secondly, the findings point to a long-term influence of socio-economic, material and psychosocial circumstances in adolescence and young adulthood. At the same time, the results indicate that the health and health inequality consequences of recent or concurrent factors in mid-adulthood might be stronger than of those in earlier life periods. On the surface, the present thus appears to be more important than the past for ill-health and health inequalities in midlife.

Thirdly, the results suggest that determinants of socio-economic, psychosocial and material nature in adolescence and young adulthood can still be considered important for midlife ill-health and health inequalities, but by other means than a direct impact. Specifically, rather than resulting in permanent or resilient health implications that endure over the life course, the long-term influence appears to reflect social processes that are conditional on recent life and living conditions in adulthood. The findings from this thesis thus indicate that the past becomes part of the present through an accumulation or chains of unfavourable conditions that contribute to midlife ill-health and health disparities by a formative influence on one’s adult life.

Taken together, the findings from this thesis illustrate how a continuum of life and living conditions, carried from the past into the present not so much in the body but by the life that we live, underpins adult ill-health and health disparities. The interesting question is therefore not about assessing the importance of the present or the past as outlined in the background, but rather how the past makes itself known through its formative influence on the present life; and conversely, how the present reflects and embodies a lifelong past. These are questions that the current thesis has provided some tentative answers to.
Implications for public health and paths for future research

The increase in mental ill-health and psychosomatic symptoms among northern European youth (Potrebny et al., 2017) has been recognised as an alarming issue by authorities in Sweden (Public Health Agency of Sweden, 2018b; The National Board of Health and Welfare, 2017). This follows from the fact that since the mid-1980s, the number of Swedish adolescents reporting repeated experiences of two or more somatic complaints over a longer time period has almost doubled (Public Health Agency of Sweden, 2018a). Based on the findings from this thesis, such health problems in youth may continue to be shaped for the rest of the life span, through social processes comprised of a continuum of life and living conditions. Since the results did not find strong support for persistent and irreversible health consequences rooted in adolescent social determinants, early preventive efforts may be the most effective strategy but not constitute the only solution. Instead, interventions in young adulthood and onwards could possibly reduce the risk of later ill-health and health disparities by breaking the links in a social chain of risk or by limiting a cumulative and added burden throughout the life course.

In this regard, initiatives aimed at improving the lives of young people may be the most appropriate and efficient response considering that early efforts could have both short- and long-term implications (Patton et al., 2016; Sawyer et al., 2012; Viner et al., 2012). In the case of Sweden, the Public Health Agency (2018b) has singled out the school environment and the labour market as crucial target areas in need of improvement for promoting the health and life chances of adolescents and young adults, contemporaneously as well as for the future. In the educational system, policies and interventions could for example strengthen school health, abolish school segregation as well as ensure professional development and reduce the administrative burden of staff (SOU 2017:35). In turn, strategies further along the life course aimed at employment and livelihood could focus on increasing the compensation of social security benefits, improving working conditions and removing barriers to enter and establish a position on the labour market (SOU 2017:47). Subsequently, it may be especially important to create opportunities for people without or with insufficient grades to complete secondary education later life (Dir 2017:21).

The results from this thesis further suggests that material and psychosocial factors are jointly and independently meaningful for ill-health and health disparities over the life course. This finding corresponds with the Swedish Commission for Equity in Health’s first report in which several target areas spanning from early life and over the life span via a range of policy domains such as education, work, housing, income, lifestyle, control and participation, was outlined (SOU 2016:55, p. 165). In addition, the life course importance of material and psychosocial conditions as illustrated in this thesis further aligns with the notion that efforts to address the
The dual goal of public health requires persistent and systematic intersectorial action (WHO, 2011a). Debates about the relative importance of different determinants thus seems redundant since many initiatives may be valuable. Rather than singling out either material or psychosocial influences (Marmot, 2017), unified strategies that cover both may be a way forward. To give an illustrative example, housing and neighbourhood programs in the US have been implemented with the aim of, or at least under the assumption that, by experiencing improvements in psychosocial and material living standards, the relocation of families from a high-poverty area to a low-poverty area will improve their health (Anderson et al., 2003; Orr et al., 2003). In their realist review of studies from one such intervention, Jackson et al. (2009) discovered that better physical conditions due to the move was a source for improved health in female youth since it increased their feelings of safety while barriers to social integration was a basis for ill-health in male youth. This shows how material or psychosocial circumstances may act in conjunction to influence health in one intervention, but also how complex and multifaceted such initiatives may be.

The findings from this thesis jointly with the broader evidence base on the social determinants of health has generated a lot of information on conditions over the life course that influence health overall and in different socio-economic groups. As indicated above, we thus have insight into and knowledge about target areas that need to be supported to achieve the two objectives of public health in Sweden (SOU 2017:47), globally (CSDH, 2008) and in Europe (Marmot et al., 2012). For the most part, however, these recommendations have been based upon a body of mainly descriptive, epidemiological research that does not offer concrete ways forward. Future studies should therefore focus on policies and interventions that address social determinants with the aim of evaluating what works (or not) to promote health and reduce health disparities, for whom, in what circumstances and how; as well as identify unexpected harms and benefits (Bambra et al., 2010; Macintyre & Petticrew, 2000). To provide further evidence that can be useful for public health policy and practice, research should also try to more systematically incorporate a causal framework. This is central for observational, epidemiological studies, but also offer a valuable perspective for evaluating program effectiveness since it forces us to think of interventions as (potential) causes (Glass, Goodman, Hernán & Samet, 2013).

Taking steps towards evaluative research using flexible methods that account for the context and other complex aspects that underlie interventions is a necessity to ensure policy relevance within the field of public health (Pettman et al., 2012) Still, there are questions in the social and life course epidemiological literature that deserves, and need more attention in parallel. With regard to issues covered in this thesis, it is known that the size of health disparities (Green, 2013; Merlo et al., 2003; San Sebastian et al., 2015) as well as the suitable choice of measures for
health and socio-economic position can vary between life periods (Galobardes, Shaw, Lawlor, et al., 2006a, 2006b; West & Sweeting, 2004). In Sweden, socio-economic disparities in health have thus far been monitored without considering these aspects – something that follows at least partially from the reporting being fragmented in terms of both approach and periodicity (Vågerö, 2017). Upcoming research in Sweden on social inequalities in health would therefore benefit from an integration of and reflection around the life course perspective. Building firstly on Vågerös (2017) call for more consistent and systematic reporting, this work should include but not be limited to the working aged population. To exhaustively monitor health inequalities, all life periods should preferably be represented. This would add complexity and require discussions about what constitutes as suitable measures for health and socio-economic position, especially in adolescence (West & Sweeting, 2004) and old age (Artazcoz & Rueda, 2007). Secondly, research is required also to outline the mechanisms that underlie and drive health disparities (SOU 2017:47). Decomposition analyses can in this regard be useful to identify social determinants that explain the inequalities (Heckley et al., 2016; Wagstaff et al., 2003). Over and above taking the differential exposure explanation as an initial line of thought (as in the decomposition approach), the complementary mechanisms of differential effect, vulnerability and susceptibility should also be explored – especially since suitable methods are now available (Diderichsen et al., 2018).

The operationalisations made in the present thesis were based on a ‘retro-fitting’ of theoretical concepts to data that had already been collected. Not only does this have methodological implications as discussed above, it is also an opportunity to suggest ways through which the research questions in this thesis could be better addressed. In depth examinations of and comparisons between different socio-economic transitions in adulthood and their consequences for health may be one step forward for the field of intra-generational social mobility. Using register data for example, this could involve more detailed analyses of time spent in different socio-economic strata, reasons for moving (e.g., was it by choice or involuntary and within or between organisations) or number of steps upward or downward in the socio-economic order. Similarly, future studies focusing on neighbourhood disparities in health should move beyond the idea that neighbourhoods are static (Oakes et al., 2015). One approach could be to recognise that the amount of time people spend outside in their area of residence and in other ‘activity spaces’ over the life course may be essential for the relationship between neighbourhood and health (Sharp, Denney & Kimbro, 2015). Another way could be to assess how this relationship is affected as neighbourhoods change over time when people move in and out, the residents grow older, have children or find a job (Hedman, 2011).

In this thesis as well as in social epidemiology more generally, the variables used often constitute proxies for underlying constructs. In relation to methodological
developments when it comes to statistical approaches that facilitate examinations of latent measures – such as e.g., structural equation modelling – the field would benefit from discussions about the characteristics of specific latent concepts. For example, in this thesis and in the related social epidemiological literature (see e.g. van der Gaag & Webber, 2008), social capital is treated as a latent construct that can be measured through a number of items that are directly measured. What is not clear and most certainly implicit, however, is if the concept should be seen as formative or reflective. In other words, are we dealing with manifest indicators that affect the latent variable (so-called causal-formative indicators) or ones that are affected by the latent variable (so-called effect-reflective indicators) (Bollen & Diamantopoulos, 2017). This ambiguity applies to many social epidemiological concepts and is basically a question of how they should be measured, interpreted and addressed (Bollen & Bauldry, 2011). Defining constructs as either formative or reflective need to follow from and be guided by our theoretical understanding of its underlying features (McCoach & Kenny, 2014). It thus seems reasonable to assume that discussions about these issues can be initiated.

To conclude, the findings from this thesis aligns with recommendations from the Swedish Commission for Equity (SOU 2017:47) in emphasising the need for broad intersectorial action with initiatives and policies spanning over a range of policy domains that successively and persistently cover people’s life span. Considering the descriptive, epidemiological nature of this thesis, however, it is only implicitly able to point towards possible interventions. In this last section, some potentially fruitful directions for future studies in the public health field therefore has been outlined. Building on the need to ensure relevance for policy and practice, more evaluation research and studies focusing on causality constitutes, in this regard, two complementary, and not mutually exclusive, ways forward.
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