Socioeconomic health differences in old age : unravelling the role of biomedical, behavioural, and psychosocial factors

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**SOCIOECONOMIC HEALTH DIFFERENCES IN OLD AGE:
UNRAVELING THE ROLE OF BIOMEDICAL, BEHAVIORAL,
AND PSYCHOSOCIAL FACTORS**

Annemarie Koster

The studies presented in this thesis were performed at the Care and Public Health Research Institute (CAPHRI) of the Universiteit Maastricht. CAPHRI participates in the Netherlands School of Primary Care Research (CaRe), re-acknowledged by the Royal Dutch Academy of Science (KNAW) in 2000.

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SOCIOECONOMIC HEALTH DIFFERENCES IN OLD AGE: UNRAVELING THE ROLE OF BIOMEDICAL, BEHAVIORAL, AND PSYCHOSOCIAL FACTORS

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aan de Universiteit Maastricht,
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volgens het besluit van het College van Decanen,
in het openbaar te verdedigen
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Contents

1	General introduction	1
2	Socioeconomic inequalities in mobility decline in chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, low back pain): only a minor role for disease severity and comorbidity	13
3	The role of psychosocial factors in explaining socioeconomic differences in mobility decline in a chronically ill population: results from the GLOBE study	29
4	Explanations of socioeconomic differences in changes in physical function in older adults	47
5	Association of inflammatory markers with socioeconomic status	65
6	Is there a biomedical explanation for socioeconomic differences in incident mobility limitation?	81
7	Socioeconomic differences in cognitive decline and the role of biomedical factors	97
8	Explaining socioeconomic differences in incident depression in older adults	113
9	General discussion	131
	Summary	147
	Samenvatting	153
	List of publications	159
	Dankwoord (Acknowledgement)	161
	About the author	163
	De auteur	165

1

General introduction

SOCIOECONOMIC DIFFERENCES IN HEALTH

We live in an ageing society. In the Netherlands in 2002, 14% of the population was 65 years or older and this is expected to increase to 23% in 2040 (1). Between 1950 and 2003, life expectancy has increased from 70.4 to 76.2 years for men and from 72.7 to 80.9 years for women. Life expectancy is estimated to further increase to 79.2 years for men and 82.6 years for women in 2050 (1). Socioeconomic status (SES) is an important predictor of life expectancy. SES refers to a person's position on the social hierarchy. Income, education, and occupation are the most frequently used indicators of SES. Low SES is associated with a higher risk of mortality. Also among older persons, SES differences in mortality are found (2-5). In the Netherlands, for example, the difference in life expectancy between lower and higher educated people is 4.9 years for men and 2.6 years for women (1). In addition, SES differences are also present in *healthy* life expectancy. People with a low SES do not only live shorter than their better-off counterparts; they also live a greater proportion of their lives in poor health (6, 7). In the Netherlands, highly educated men and women live respectively 9.9 and 8.6 years longer without physical limitations than lower educated persons (1). A study in the United States showed that these SES differences in healthy life expectancy have increased over the last decades (8).

SES differences have been found in various health outcomes and across many countries (9-11). Although SES differences in health occur in different age groups, these health differences have been less examined in older age groups than in younger age groups. Some studies report that SES differences in health attenuate in old age (12, 13), although evidence is contradictory (14). In an older population, function is an important health outcome. Different types of function, such as physical, cognitive, and mental function, cover larger dimensions of a person's health status. Overall, functional status measures are strong predictors of hospital outcomes (for example length of stay), nursing home admission, and mortality (15-18). Poor physical function can lead to disability (difficulty doing activities of daily living) which is related to many diseases and, especially in older adults, physical disability has been shown to be an important predictor of mortality (19). Cognitive impairment is a major problem among older persons and maintaining good cognitive function is an essential part of successful aging (20). Depression (mental function) also has negative consequences for well-being and daily functioning and is associated with higher mortality rates (21-23).

Low SES has been shown to be an important predictor of various functional outcomes. Particularly the association between SES and physical function has been well described in the literature (24-27). Low SES is a predictor of declines in physical

function which has been found in both younger and older adults (28, 29). The association between SES and cognitive function is relatively understudied. There is some evidence that low SES is also related to poor cognitive function; longitudinal studies are, however, limited at present (30, 31). Furthermore, SES differences have been found in mental disorders, including depression (25, 32, 33). A higher prevalence and incidence of depression has been observed among low SES groups compared to people with a higher SES. In general, relatively few studies have investigated the association between SES and functional outcomes in older persons and longitudinal studies are limited.

EXPLAINING SOCIOECONOMIC DIFFERENCES IN HEALTH

In order to prevent the negative consequences of poor function and especially the SES differences therein and to develop intervention strategies, understanding the pathways underlying these associations is important. In 1980, the Black Report provided theoretical explanations for SES differences in health (34). Two main mechanisms have been proposed: the selection mechanism and the causation mechanism. The selection mechanism assumes that health affects social mobility; healthy people may move up in the social hierarchy whereas unhealthy people may move down. The causation mechanism is believed to be more important in the explanation for SES differences in health (34). Social causation assumes that SES has an effect on health through an unequal distribution of determinants of health across SES groups. Based on the causation mechanism, there are several pathways in which SES may affect health outcomes. In this thesis, three important groups of factors will be examined in the explanation of SES differences in functional outcomes: biomedical, behavioral, and psychosocial factors. These groups of factors will be discussed below.

Biomedical factors

Biomedical factors, such as diseases and biological risk factors, are likely to be important in the mechanism through which low SES leads to poor functional outcomes. Low SES is related to a higher prevalence of many diseases, such as cardiovascular disease, stroke, and diabetes (35-37). Biological risk factors, such as hypertension, glucose intolerance, and reduced lung function are also more prevalent among low SES groups (38-40). Many diseases and their risk factors are directly related to physical function (26, 41). Biomedical factors are also related to poor cognitive function and increased symptoms of depression (42-44). Accumulating

evidence shows that inflammation, being a biological response of the immune system, might also be important. Recent research shows that high serum levels of inflammatory markers are inversely related to SES (45-47). High serum levels of inflammatory markers are related to poor physical and cognitive function and a higher prevalence of depression (48-50). Despite the knowledge that diseases and their risk factors are associated with both low SES and poor functional outcomes, the extent to which biomedical factors can explain SES differences in function has not been studied extensively.

Behavioral factors

The behavioral explanation of SES differences in health implies that people with low SES suffer from poor health due to higher rates of smoking, excessive alcohol consumption, physical inactivity, and obesity. Compared to high SES people, persons with a low SES generally have an unhealthier lifestyle. They smoke more often, drink more alcohol, consume unhealthier diets, and are less physically active (51-53). These adverse behavioral factors are negatively associated with functional outcomes (41, 54-57). Previous studies have found that behavioral factors contribute to SES differences in physical function, self-reported health, and mortality (25, 58, 59).

Psychosocial factors

Psychosocial factors may also moderate the impact of SES on function (60). Generally, people with low SES have fewer psychosocial resources than their better-off counterparts. Low SES is, for example, related to low control beliefs, negative coping styles, poor social support, and more stressful life events (60-62). Psychosocial factors influence the perception of events and the degree to which they are experienced as stressful. How people cope with these stressful situations affects various health outcomes, for example physical function (41, 63-65). The association between psychosocial factors and depression has been studied extensively, especially the positive effect of social support on mental health (66-69). The contribution of psychosocial factors in the explanation of SES differences in function has not been studied elaborately.

AIM AND OUTLINE OF THIS THESIS

The first objective of this thesis is to describe the associations between SES and three different aspects of function: physical, cognitive, and mental function. The

second objective of this thesis is to explore the role of biomedical, behavioral, and psychosocial factors in the explanation of SES differences in the three functional outcomes. This will contribute to a better understanding of the mechanisms underlying SES health differences. Figure 1 shows the research model in which the numbers refer to the different chapters in this dissertation.

Figure 1. Research model of this thesis. Numbers indicate the chapters in this thesis

The studies that are presented in this thesis have important strengths. First, the associations between SES and *three different types of function*, that represent different aspects of a person's health status, are studied. Second, the role of *three different groups of explanatory factors* is studied. Most studies in this thesis will focus on the contribution of one group of explanatory variables in the association between SES and physical, cognitive, or mental function. The contribution of biomedical, behavioral, and psychosocial factors, however, will also be studied simultaneously in order to determine the relative contribution of these factors in the explanation of SES differences in function. Third, the associations between SES and function are studied *longitudinally*. Especially longitudinal studies between SES and cognitive function are limited at present. Fourth, it will be determined whether SES differences in function still exist in *old age*. Finally, *different indicators of SES*, including education and income, that represent different dimensions of SES, will be used. For example, education is the socio-cultural dimension of SES and represents social class in an early stage of the life course compared to income representing a later life stage.

Data from three different longitudinal cohort studies were used. The first study is the GLOBE (Dutch acronym for Health and Living conditions of the population of

the city of Eindhoven and surroundings in the Netherlands) study with six years of follow-up. The particular sub-study consists of 2,867 men and women, aged 15 to 74 years, in which chronically ill patients were over-represented. Using these data, the association between SES and physical function is studied in both younger and older adults. The second study is the Longitudinal Aging Study Amsterdam (LASA), consisting of 3,107 men and women between 55 and 85 years old in different parts of the Netherlands. Nine years of follow-up were included. The third study is the Health, Aging and Body Composition (Health ABC) study, consisting of 3,075 black and white men and women, aged 70 to 79 years, from Memphis, Tennessee and Pittsburg, Pennsylvania in the US. Four and a half years of follow-up could be used.

In *Chapter 2* the association between SES and mobility decline is examined in four chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, low back pain) and it was examined whether this association was explained by disease severity and comorbidity. Data from the GLOBE study were used.

Chapter 3 elaborates on the previous chapter by examining the role of psychosocial factors in the explanation of SES differences in mobility decline, independent of disease severity, comorbidity, and health-related behavior, in the same population (GLOBE study).

The association between SES and changes in physical function in younger-old (aged 55 to 70 years) and older-old (aged 70 to 85 years) adults is studied in *Chapter 4*, using data from LASA. Additionally, this study seeks to simultaneously examine the relative contribution of diseases, behavioral, and psychosocial factors in explaining this association.

Whether SES differences exist at a pathophysiological level is examined in *Chapter 5*. Therefore, the association between SES and inflammatory markers is studied more in detail in well-functioning older adults and it will be determined whether any association remains after adjusting for diseases and behavioral factors typically related to elevated serum levels of inflammatory markers. Data from the Health ABC study were used.

Chapter 6 reports on the association between SES and incident mobility limitation in well-functioning older adults and the role of biomedical factors, including inflammatory markers, in explaining this link in the Health ABC study.

Chapter 7 describes the association between SES and cognitive decline in well-functioning older adults participating in the Health ABC study. Furthermore, the role of biomedical factors is examined in explaining this association.

Using data from LASA, the association between SES and the onset of depression in older adults is presented in *Chapter 8*. Moreover, the role of psychosocial factors, physical health status, and behavioral factors is studied in the explanation of this link.

Finally, the findings of the previous chapters are summarized and discussed in *Chapter 9*.

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2

Socioeconomic inequalities in mobility decline in chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, low back pain): only a minor role for disease severity and comorbidity

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ABSTRACT

Objective. In chronically ill patients, mobility develops more unfavorable in persons with a low socioeconomic status than in their better-off counterparts. It is not clear, whether the adverse course of physical functioning in persons with a low socioeconomic status can be explained by a higher prevalence of more severe disease or comorbidity in these persons. Using longitudinal data from the Dutch GLOBE study, the present study examined the association between socioeconomic status and mobility decline and whether this could be explained by disease severity and comorbidity in four different chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, and low back pain).

Methods. Data were from 1,384 men and woman, aged 15-74 years, suffering from at least one of the four chronic diseases. Three indicators of socioeconomic status were used: education, occupational class, and income. Change in physical mobility between 1991 and 1997 was measured with the Nottingham Health Profile (NHP).

Results. Odds ratios of mobility decline between 1991 and 1997, adjusted for age, sex, marital status, and baseline mobility, were significantly higher in low socioeconomic groups in comparison to high socioeconomic groups. Only very little of this association could be explained by the higher disease severity and comorbidity in these patients. Findings were similar in patients with asthma/COPD, heart disease, diabetes mellitus, and chronic low back pain.

Conclusions. Our findings indicate that in order to reduce physical disabilities and particularly the socioeconomic differences therein, it may not be sufficient to solely intervene upon the risks of severe disease and comorbidities.

INTRODUCTION

There is substantial evidence that physical functioning, such as mobility, develops more unfavorably in persons with a low socioeconomic status (SES) (1-4). The same effects are also found in chronically ill persons (5-8). Chronically ill persons, who have a low level of education, belong to a low occupational class, or have a low household income, develop more disabilities than chronically ill persons with a high SES (7). Less clear, however, is whether the adverse course of physical functioning in persons with a low SES can be explained by a higher prevalence of more severe disease or comorbidity in these persons. A recent study showed that disease severity is associated with physical disabilities (7). Another study shows that people with a low socioeconomic position report a greater disease severity than people with a high socioeconomic position (9, 10). Low SES is also positively related to comorbidity which is also likely to increase disabilities (11). It is not clear whether previous studies in chronic disease patients have sufficiently controlled for a potentially higher disease severity and comorbidity in low SES groups. This is relevant for determining the extent to which any low SES – restricted mobility association is confounded by disease severity and/or comorbidity. Furthermore, in order to reduce physical disabilities and particularly the SES differences therein, it is important to know whether it is sufficient to reduce severe disease and comorbidities in chronically ill persons with a low SES or whether other socially patterned factors should be tackled as well.

Using longitudinal data this study examines the association between SES and mobility decline and whether this could be explained by disease severity and comorbidity in four different chronic disease groups (asthma/chronic obstructive pulmonary disease (COPD), heart disease, diabetes mellitus, and low back pain). These four diseases were chosen because they are fairly common, cover a wide range of possible consequences of disease for daily functioning (12), and represent varying body systems, i.e. respiratory (asthma/COPD), cardiovascular (heart disease), metabolic (diabetes mellitus), and muscular-skeletal (low back pain).

METHODS

Study population

Data were obtained from the GLOBE (a Dutch acronym for Health and Living Conditions of the Population of Eindhoven and surroundings) study, a longitudinal study that started in 1991 in the south east of the Netherlands, aimed at explaining

SES inequalities in health (13). It is based on a cohort of 15 to 74 year old, non-institutionalized people of Dutch nationality. In 1991 the study started with a baseline measurement consisting of a postal survey and an oral interview. A sample of 27,070 inhabitants of 18 municipalities received a postal questionnaire; the sample was drawn from population registers stratified by age and postal code (45-74 years old and people from the highest and lowest SES groups were overrepresented). The response rate of the postal survey was 70.1% resulting in a study population of 18,973 respondents. A sub-sample of 3,968 persons drawn from respondents to the postal survey was approached for an oral interview. Participants completed an interview and a self-administered questionnaire; the response was 72.2% (n=2,867). In this sub-sample, people who reported one or more of the following chronic diseases were overrepresented: asthma/COPD, heart disease, diabetes mellitus, and severe low back pain. For the analyses presented in this paper, respondents suffering from at least one of the four chronic diseases were selected, resulting in 2,015 respondents in 1991. Response at follow-up in 1997 was 69.8% of the 2,015 respondents in 1991, resulting in 1,407 respondents. For 23 participants outcome data were missing, leaving 1,384 participants for the present analyses. The number of respondents in each group was: asthma/COPD; n=465, heart disease; n=788, diabetes mellitus; n=137, and low back pain; n=707. There were 580 respondents who suffered from more than one condition.

Measures

Socioeconomic status. We used three indicators of SES: level of education, equivalent income, and occupational class. Three categories of level of education were created: high (university, higher vocational training); medium (higher secondary, intermediate vocational training); and low (lower secondary, lower vocational training). Occupational class was measured according to Erikson, Goldthorpe, and Portocarero (EGP) classification (14) that originally consisted of 11 categories. In this study four categories were created: (1) (higher) grade professionals; (2) routine non-manual workers and high skilled manual workers; (3) self-employed; and (4) low skilled and unskilled manual workers. Net equivalent income per month was measured by adding all income components of all household members adjusted for household size. Three categories of equivalent income, in euros, were distinguished: high (>947); medium (658-947); and low (<658). Education data were missing for 28 participants; this was 171 for occupation and 215 for income.

Physical mobility. Physical mobility was measured with the Nottingham Health Profile (NHP). The NHP is a self-administered questionnaire designed to measure perceived health problems (15). The part that measures physical mobility consists of

8 statements that reflect problems with mobility (see appendix). Respondents had to answer 'yes' or 'no' to each of these statements. Because the statements vary in severity, they have been weighted by means of Thurstone's Method of Paired Comparisons (16). The minimum score is 0 and the maximum score is 100, which represents a situation where the respondent answers affirmatively to all the statements. The higher the score, the greater the mobility problems are. To determine whether there was a decline or not between 1991 and 1997, a reliable change index was computed using Edwards-Nunnally method of determining improvement rates. This method takes into account the measurement reliability of the NHP mobility scale (17). Decline conform the Edwards-Nunnally method was defined as a 1991 - 1997 difference score greater than 6.

Disease severity. Disease-specific questionnaires on asthma/COPD (18), heart disease (19, 20), diabetes mellitus (21), and low back pain (22, 23) were used to determine the severity of the chronic disease (7, 8). Four categories for asthma/COPD were distinguished: (1,2,3) three categories indicating increasing severity of asthma/COPD (grade 1 to grade 3); and (4) other forms of asthma (18). For heart disease, five categories were distinguished: (1) non-specific symptoms; (2) angina pectoris without heart failure; (3) heart failure without angina pectoris; (4) angina pectoris with heart failure; and (5) other forms of heart disease (19, 20). Two categories of diabetes mellitus were distinguished: (1) diabetes without complications; and (2) diabetes with at least one complication (21). Finally, five categories of low back pain were distinguished: (1) low back pain, no radiation, less than three months; (2) low back pain, with radiation, less than three months; (3) low back pain, no radiation, at least three months; (4) low back pain, with radiation, at least three months; and (5) other forms of back complaints (22, 23).

Comorbidity. Comorbidity was defined as the number of other diseases that respondents reported. Comorbidity was measured with two variables. The first variable reflects the number of additional severe comorbid diseases that people had (asthma/COPD, coronary, stroke, stomach ulcer, kidney disease, diabetes mellitus, arthritis, rheumatoid arthritis, disease on nervous system, cancer). The second variable indicates the number of less severe comorbid diseases (intestinal disorders, high blood pressure, gallstones, kidney stones, prostate complaints, back pain, joint problems, migraine, depression, chronic skin disease, prolapse, varicose veins, and accidental injury).

Statistical analyses

All analyses, using SPSS version 10.1, were carried out separately for each chronic disease group and every SES indicator. To determine if there were

differences in baseline mobility between SES groups, a Kruskal-Wallis test was used. A non-parametric test was used because the NHP-score did not have a normal distribution; many respondents were free of mobility problems. The chi-square test was used to determine whether there were differences in the percent of people with a decline in mobility between SES groups. Logistic regression models were fitted to study the association between SES and disease severity and comorbidity. Disease severity and comorbidity were the dependent variables in these models and were therefore dichotomized. Disease severity was dichotomized as the most severe category compared with the other ones and comorbidity as any comorbid diseases compared with no comorbid disease. These analyses were adjusted for age (continuous variable), sex, and marital status (never married, married, divorced, widowed). Logistic regression models were also fitted to study the association between disease severity and comorbidity on the one hand and mobility decline, as the dependent variable, on the other hand, adjusting for age sex, marital status, and baseline mobility (continuous variable). Finally, the main logistic regression analyses were carried out where the dependent variable was the decline in mobility between 1991 and 1997. The determinant of interest was SES, where the highest SES group was always the reference category. Two logistic regression models were fitted, the first model adjusted for baseline mobility, age, sex, and marital status. Model 2 contained all variables of the first model, as well as severity of the chronic disease and comorbidity. The goodness of fit of the logistic regression models was tested using the Hosmer and Lemeshow test, which tells us something about predicted versus observed classifications. The significance of all logistic regression models was greater than 0.05, which means that the model's estimates fit the data at an acceptable level. After removing outliers from the data set for the logistic regression analyses, using the studentized residuals and Cook's distance, results were very similar or the same.

RESULTS

Table 1 presents the number of people at baseline, the median mobility score at baseline, and the percentage of people with a decline in mobility for every SES indicator and for every disease group. At baseline, people in a low SES group had more mobility problems than people in the high SES groups. This was statistically significant for every SES indicator in the asthma/COPD group, the heart disease group, and the group with low back pain. In the diabetes group, there were no statistically significant differences between SES groups. The percentage of people

with a decline in mobility problems generally was also highest in the low SES groups. For example, in the asthma/COPD group 42.3% of the people with a low educational level had a decline in mobility between 1991 and 1997 in comparison to 22.7% with a high educational level. These higher percentages were not significant for the diabetes group and the low back pain group.

Table 2 presents the results of the association between SES on the one hand and disease severity and severe comorbid diseases on the other hand. Risks of having a severe chronic disease and more severe comorbid diseases generally were higher for lower SES groups, although the heightened risks were not always statistically significant. For example, odds ratios of disease severity in low education groups were 1.92 (95% confidence interval (CI): 1.04-3.55) for asthma/COPD, 2.00 (95% CI: 0.87-4.60) for heart disease, 2.63 (95% CI: 0.61-11.26) for diabetes, and 1.10 (95% CI: 0.64-1.88) for low back pain. Odds ratios of severe comorbid diseases in low education groups were 1.94 (95% CI: 1.05-3.57) for asthma/COPD, 1.74 (95% CI: 1.09-2.78) for heart disease, 1.47 (95% CI: 0.41-5.35) for diabetes, and 1.80 (95% CI: 1.11-2.92) for low back pain. The associations between SES and less severe comorbid diseases were highly similar (not tabulated).

The association between disease severity, comorbidity and mobility decline is shown in table 3. A more severe disease increased the risk of getting more mobility problems. The strongest association was found in the most severe category for each disease; asthma/COPD grade 3 (odds ratio (OR): 1.72; 95% CI: 0.97-3.08), angina pectoris and heart failure (OR: 1.62; 95% CI: 1.00-2.62), diabetes with complications (OR: 4.98; 95% CI: 2.02-12.29), low back pain with radiation and at least 3 months (OR: 1.54; 95% CI: 0.97-2.46). Having additional severe comorbid diseases also increased the risk of getting more mobility problems. These results were statistically significant in all disease groups except in the diabetes group. There was only an association with less severe comorbid diseases in the heart disease group (OR: 1.25; 95% CI: 1.10-1.42).

Generally, odds ratios of mobility decline, adjusted for age, sex, marital status, and baseline mobility, were significantly higher in low SES groups in comparison to high SES groups (Table 4, Model 1). Low income showed particularly heightened risks of mobility decline; odds ratios were 2.83 (95% CI: 1.58-5.09), 1.97 (95% CI: 1.29-3.02), 2.28 (95% CI: 0.70-7.37), and 1.81 (95% CI: 1.16-2.83) for asthma/COPD, heart disease, diabetes, and low back pain, respectively. In the diabetes group, odds of mobility decline for low SES were not significantly different from the odds for high SES. Furthermore, low educational level did not affect mobility decline in the group with low back pain. Controlling for comorbidity and disease

Table 1. Baseline mobility (median, first and third quartile) and mobility decline, for different socioeconomic status indicators and for every disease group

Education ^a	n	Baseline mobility median 1 st - 3 rd quartile	Mobility decline %	Occupation ^b	n	Baseline mobility median 1 st - 3 rd quartile	Mobility decline %	Income ^c	n	Baseline mobility median 1 st - 3 rd quartile	Mobility decline %
Asthma/COPD											
380											
High	66	0.00	0.00-10.63	1	134	0.00	0.00-11.20	High	128	0.00	0.00-11.20
Medium	163	0.00	0.00-11.20	2	105	0.00	0.00-11.20	Medium	132	0.00	0.00-11.20
Low	227	10.79	0.00-21.99	3	14	15.85	0.00-31.44	Low	129	11.20	0.00-26.91
p-value	<0.01		<0.01	4	127	11.20	0.00-21.99	p-value	<0.01		<0.01
Heart disease											
687											
High	99	0.00	0.00-9.30	1	229	0.00	0.00-11.20	High	220	0.00	0.00-11.20
Medium	256	0.00	0.00-11.20	2	189	0.00	0.00-11.20	Medium	221	0.00	0.00-20.50
Low	415	10.57	0.00-23.89	3	28	20.50	0.00-31.07	Low	229	11.20	0.00-31.07
p-value	<0.01		<0.01	4	241	10.57	0.00-31.07	p-value	<0.01		<0.01
Diabetes											
123											
High	13	0.00	0.00-5.60	1	34	0.00	0.00-0.00	High	105	0.00	0.00-10.89
Medium	41	0.00	0.00-11.20	2	38	0.00	0.00-20.41	Medium	33	0.00	0.00-11.20
Low	76	0.00	0.00-20.40	3	5	20.50	0.00-38.23	Low	36	0.00	0.00-23.26
p-value	0.72		0.12	4	46	0.00	0.00-25.57	p-value	0.50		0.09
Low back pain											
631											
High	93	10.57	0.00-11.20	1	204	11.20	0.00-21.77	High	602	11.20	0.00-21.77
Medium	245	11.20	0.00-21.77	2	178	11.20	0.00-21.77	Medium	210	11.20	0.00-21.99
Low	358	11.20	0.00-32.56	3	23	21.99	11.20-32.56	Low	207	11.20	0.00-35.23
p-value	<0.01		0.65	4	226	11.42	0.00-32.56	p-value	<0.01		0.17

^a Education: high (university, higher vocational); medium (higher secondary, intermediate vocational); low (lower secondary, lower vocational)^b Occupation: 1 (higher) grade professionals; 2 routine non-manual workers or high skilled manual workers; 3 self-employed; 4 low skilled and unskilled manual workers^c Equivalent income in euros: high (>947.04); medium (657.53-947.04); low (<657.53)

Table 2. Odds ratios (OR) and 95% confidence interval (CI) for severity and comorbidity by socioeconomic status; separately for different chronic disease groups, adjusted for age, sex and marital status *

Education ^a	Severe disease †			Severe comorbidity ‡			Severe disease †			Severe comorbidity ‡		
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Asthma/COPD												
High	1.00		1.00		1.00		1.00		1.00		1.00	
Medium	1.45	0.78-2.69	1.06	0.58-1.94			1.72	1.00-2.96	1.16	0.67-2.00	1.35	0.81-2.24
Low	1.92	1.04-3.55	1.94	1.05-3.57			2.85	0.91-8.90	1.49	0.46-4.87	1.78	1.07-2.99
					4		2.79	1.65-4.73	2.37	1.35-4.15		
Heart disease												
High	1.00		1.00				1.00		1.00		1.00	
Medium	1.69	0.71-4.02	1.13	0.69-1.83			1.51	0.82-2.78	0.98	0.66-1.46	1.30	0.73-2.33
Low	2.00	0.87-4.60	1.74	1.09-2.78			1.61	0.50-5.18	1.61	0.72-3.59	1.49	0.84-2.65
					4		1.86	1.06-3.27	1.85	1.27-2.70		
Diabetes												
High	1.00		1.00				1.00		1.00		1.00	
Medium	1.86	0.42-8.23	0.79	0.21-2.98			1.22	0.47-3.19	0.99	0.38-2.57	2.29	0.77-6.79
Low	2.63	0.61-11.26	1.47	0.41-5.35			5.98	0.59-60.56	1.31	0.19-9.29	3.94	1.30-11.92
					4		0.99	0.37-2.65	0.81	0.31-2.13		
Low back pain												
High	1.00		1.00				1.00		1.00		1.00	
Medium	1.07	0.61-1.87	1.11	0.67-1.82			1.06	0.66-1.68	1.15	0.75-1.77	1.16	0.75-1.82
Low	1.10	0.64-1.88	1.80	1.11-2.92			1.86	0.72-4.77	0.81	0.33-2.01	1.36	0.86-2.14
					4		1.12	0.72-1.73	1.26	0.85-1.89		

* All p-values of the Hosmer and Lemeshow for goodness of fit of logistic regression are >0.05, indicating that the model's estimates fit the data at an acceptable level. All analyses were carried out with the same number of respondents in each group: asthma/COPD 465, heart disease 788, diabetes mellitus 137, and low back pain 707

^a See notes in Table 1

† Severe disease: 1: most severe category; and 0: less severe categories

‡ Severe comorbidity: 1: any severe comorbid condition; and 0: no severe comorbid condition

Table 3. Odds ratios (OR) and 95% confidence interval (CI) for decline in mobility by disease severity and comorbidity; separately for different chronic disease groups, adjusted for age, sex, marital status, and baseline mobility *

		Mobility decline	
		OR	95% CI
Asthma/COPD			
Severity	grade 1	1.00	
	grade 2	1.01	0.51-2.01
	grade 3 †	1.72	0.97-3.08
	other forms of asthma/COPD	0.78	0.38-1.59
Severe comorbid diseases		1.66	1.24-2.22
Less severe comorbid diseases		1.10	0.93-1.30
Heart disease			
Severity	non-specific symptoms	1.00	
	angina pectoris without heart failure	1.29	0.88-1.88
	heart failure without angina pectoris	0.81	0.47-1.41
	angina pectoris with heart failure †	1.62	1.00-2.62
	other forms of heart disease	0.77	0.44-1.36
Severe comorbid diseases		1.38	1.10-1.72
Less severe comorbid diseases		1.25	1.10-1.42
Diabetes			
Severity	without complications	1.00	
	with complications †	4.98	2.02-12.29
Severe comorbid diseases		0.95	0.56-1.63
Less severe comorbid diseases		1.01	0.71-1.44
Low back pain			
Severity	no radiation, less than 3 months	1.00	
	with radiation, less than 3 months	0.95	0.60-1.50
	no radiation, at least 3 months	1.02	0.61-1.72
	with radiation, at least 3 months †	1.54	0.97-2.46
	other forms of back complaints	0.91	0.36-2.31
Severe comorbid diseases		1.27	1.06-1.52
Less severe comorbid diseases		0.98	0.97-0.99

* See corresponding note in Table 2

† Most severe disease category

severity had only little effect on the odds ratios of mobility decline for low SES (Table 4, Model 2). Almost all significant odds ratios of model 1 remained statistically significant in model 2. In model 1, 12 odds ratios were statistically significant. After controlling for disease severity and comorbidity, 10 odds ratios were still statistically significant. Odds ratios also decreased only little between model 1 and 2. Interaction terms between SES and age, and SES and sex were not statistically significant, which confirmed that the association between SES and mobility decline was similar for all ages and men and women.

Table 4. Odds ratios (OR) and 95% confidence interval (CI) for decline in physical functioning by socioeconomic status; separately for different chronic disease groups *

		Asthma/COPD		Heart disease		Diabetes		Low back pain	
		OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Education^a									
Model 1 †	High	1.00		1.00		1.00		1.00	
	Medium	1.18	0.58-2.38	1.88	1.06-3.32	0.98	0.19-4.99	1.04	0.62-1.76
	Low	1.90	0.95-3.79	2.40	1.38-4.16	1.77	0.38-8.33	1.03	0.62-1.72
Model 2 ‡	High	1.00		1.00		1.00		1.00	
	Medium	1.07	0.52-2.20	1.75	0.98-3.12	0.76	0.14-4.26	1.02	0.60-1.74
	Low	1.73	0.85-3.53	2.37	1.35-4.14	1.18	0.87-1.02	1.01	0.60-1.70
Occupation^b									
Model 1 †	1	1.00		1.00		1.00		1.00	
	2	1.21	0.67-2.17	1.73	1.13-2.65	1.65	0.55-4.95	1.34	0.87-2.08
	3	2.37	0.75-7.50	2.30	1.00-5.28	0.97	0.11-8.61	1.58	0.61-4.07
	4	2.21	1.27-3.86	1.91	1.28-2.86	1.15	0.38-3.44	1.60	1.05-2.43
Model 2 ‡	1	1.00		1.00		1.00		1.00	
	2	1.11	0.60-2.03	1.67	1.09-2.57	1.77	0.52-5.96	1.35	0.86-2.10
	3	2.02	0.62-6.65	2.21	0.95-5.16	0.65	0.07-6.42	1.61	0.62-4.19
	4	1.79	1.00-3.20	1.80	1.19-2.71	1.45	0.42-5.04	1.59	1.04-2.45
Income^c									
Model 1 †	High	1.00		1.00		1.00		1.00	
	Medium	1.81	1.02-3.20	1.72	1.13-2.61	1.53	0.48-4.81	1.26	0.82-1.92
	Low	2.83	1.58-5.09	1.97	1.29-3.02	2.28	0.70-7.37	1.81	1.16-2.83
Model 2 ‡	High	1.00		1.00		1.00		1.00	
	Medium	1.82	1.01-3.26	1.69	1.10-2.59	1.22	0.35-4.18	1.23	0.80-1.89
	Low	2.60	1.43-4.74	1.94	1.25-2.99	1.76	0.51-6.06	1.69	1.07-2.67

* See corresponding note in Table 2

^{a,b,c} See notes in Table 1

† Model 1: Adjusted for sex, age, marital status, and baseline mobility

‡ Model 2: Adjusted for sex, age, marital status, baseline mobility, comorbidity, and baseline severity

DISCUSSION

In different chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, low back pain), people with a low SES generally had an increased risk of mobility decline in comparison to people with a high SES. Low SES was also associated with more severe chronic diseases and more severe comorbid diseases. Both disease severity and comorbidity were also related to mobility. In spite of these associations disease severity and comorbidity contributed only very little to the SES differentials in mobility decline. This was found across all disease groups and all three SES indicators. Strongest associations were found with equivalent income as SES indicator. Odds ratios in the lowest income groups ranged from 1.69 to 2.60 in the fully adjusted model (Table 4, Model 2). Strongest associations between SES and mobility decline were found in the heart disease group. The contribution of

disease severity and comorbidity in the heart disease group was not different from the other chronic diseases. Effects were less pronounced in the diabetes group, but this was probably partly due to the small number of people in the diabetes group. These findings imply that in chronically ill patients, low SES is related to decreasing mobility and that the higher prevalence of severe disease and comorbidities in lower SES groups can hardly explain this association.

Because of the longitudinal character of our study, we may conclude that there probably is a causal relation between SES and mobility decline. However, because only baseline data on severity and comorbidity were analyzed, the association between SES on the one hand and disease severity and comorbidity on the other hand might have resulted from reverse causation or health-related mobility (24-26). For example, people having a more severe disease might have ended up in a lower SES group (26). It is likely that this selection effect has a larger effect on income and perhaps occupation than on educational level, as the latter indicator is less sensitive to change during adulthood. In this study, same results were found across all SES indicators, including education. Furthermore, there is substantial evidence that low SES predicts the incidence of all kinds of diseases (27), more severe diseases (9, 10), and higher risks of comorbidity (11). Whether based on causation or selection, our findings indicate that the higher prevalence of severe disease and comorbidities in lower social classes can hardly explain the heightened odds of mobility restrictions in these groups.

Our findings imply that there are other factors that explain the association between low SES and mobility decline. These factors may be related to behavioral factors or psychosocial factors. Low SES is related to many adverse behavioral factors, such as smoking, excessive alcohol consumption, and decreasing physical activity that on their turn are related to poor health outcomes (3, 28, 29). There is also evidence that psychosocial factors, such as control belief and stress, play an important role in functional outcomes (30), and also in the association between SES and poor functional outcomes (31, 32). Income, being the strongest predictor of mobility, suggests that the direct availability of financial resources might also be important (33). A lack of material resources held by individuals and the availability of public resources might be important in explaining SES differences in health. Life course factors such as lifetime exposures to adverse occupational and social circumstances may also play a role.

Some limitations of the study have to be considered. First, this study was entirely based on self-reports. Despite this limitation, we were able to control for disease severity, using disease-specific questionnaires and comorbidity. However, we only adjusted for disease severity and comorbidity at baseline. Assuming that

disease severity and comorbidity increase more in low SES groups than in high SES groups over time, the contribution of severity and comorbidity may have been underestimated. Second, our study consists of persons younger than 75 years only. For the present research question, this should be considered a limitation, because generalizability is thus restricted to persons younger than 75 years. SES differences in mobility decline and the role of medical factors therein may be different in persons older than 74 years. Third, we did not have information about the time of onset of disease. It remains unknown whether the time of onset of disease differs between SES groups and the extent to which this affects mobility decline. Fourth, baseline NHP-data were collected during an oral interview, while NHP-data in 1997 with a postal questionnaire. It is possible that people were more likely to admit health problems in the postal survey, therefore we could have overestimated the number of people with a mobility decline. However, it remains unclear whether this affected the association between SES and mobility decline. Fifth, baseline response was 72.2%; and was lower in low SES groups. Attrition due to mortality and non-response was also higher in low SES groups (8). Response at follow up in 1997 was 69.8%. Participants who were lost to follow-up also had statistically significantly worse NHP-scores at baseline ($p < 0.01$) in comparison to persons who remained in the study. Furthermore, these persons had more severe chronic diseases and more severe comorbid diseases ($p < 0.01$). The association between SES and mobility decline might therefore be underestimated in this study. Whether the relative contribution of disease severity and comorbidity is equally underestimated remains unknown. Finally, occupation data and income data were missing for 171 and 215, respectively. These persons more often had a lower educational level compared with persons with full data. This may also have led to an underestimation of the effect of occupation and income on mobility decline.

In summary, there is a strong relation between SES and mobility decline among chronically ill persons. Only little of this relation could be explained by disease severity and comorbidity. Findings were similar in patients with asthma/COPD, heart disease, diabetes mellitus, and chronic low back pain. Further research is necessary to determine whether behavioral, material, and/or psychosocial factors additionally contribute to the association between SES and mobility decline among the chronically ill. Our findings are also important for policies aimed at reducing functional limitations in people with chronic diseases, particularly in people from lower SES group. They imply that reducing the risk of severe disease and comorbidities may not be sufficient to reduce physical disabilities and particularly the SES differences therein.

APPENDIX

Mobility part of the Nottingham Health Profile

1. I can only walk indoors
2. I find it hard to bend
3. I'm unable to walk at all
4. I have trouble getting up and down stairs or steps
5. I find it hard to reach for things
6. I find it hard to dress myself
7. I find it hard to stand for long
8. I need help to walk outside

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3

The role of psychosocial factors in explaining socioeconomic differences in mobility decline in a chronically ill population: results from the GLOBE study

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ABSTRACT

Objective. In chronically ill patients, mobility develops more unfavorably in persons with low socioeconomic status than in their better-off counterparts. Using longitudinal data from the Dutch GLOBE study (a study of health and living condition of Eindhoven and its surroundings), the present study examines whether psychosocial factors can explain socioeconomic differences in mobility decline, independent of disease severity, comorbidity, and health-related behavior, in a chronically ill population.

Methods. Data were from 1,384 men and woman, aged 15-74 years, suffering from at least one of the four chronic diseases: asthma/COPD, heart disease, diabetes mellitus, and low back pain. Three indicators of socioeconomic status were used: education, occupational class, and income. Change in physical mobility between 1991 and 1997 was measured with the Nottingham Health Profile (NHP). Psychosocial factors were measured extensively and included life events, long term difficulties, coping styles, social support, and personality characteristics.

Results. Low occupational level and low income predicted an increased risk of mobility decline in comparison to people with a higher occupational level and income, even after controlling for sociodemographics, disease severity, comorbidity, and health-related behavior. Additional adjustment for psychosocial factors hardly reduced the socioeconomic status-related odds ratios of mobility decline.

Conclusions. Psychosocial factors (and also health-related behaviors, disease severity, and comorbidity) cannot explain socioeconomic differences in mobility decline in a chronically ill population. Our findings will not give us new tools for prevention and intervention strategies in order to reduce physical disability and particularly the SES differences therein.

INTRODUCTION

The association between low socioeconomic status (SES) and poor physical functioning has been well described (1-3). The same associations are also found in chronically ill persons. Chronically ill persons with a low level of education, who belong to a low occupational class, or who have a low income develop more disabilities than their better-off counterparts (4-7). Less clear, however, is how these differences can be explained. Our previous work shows that only very little of the SES differentials in mobility decline could be explained by a higher disease severity and comorbidity in these patients (8). These findings imply that there are other factors important in explaining the association between low SES and mobility decline.

In addition to disease severity and comorbidity, psychosocial factors may be important in explaining the association between low SES and mobility decline. There are studies that show that psychosocial factors explain part of the SES differentials in health (2, 9, 10). People with low SES generally have a more negative psychosocial profile than people with high SES. Low SES is related to low personal control beliefs, poor social support, and negative coping styles (10-13). Psychosocial resources influence the perception of events and the degree to which they are experienced as stressful and how people can cope with these stressful situations affects various health outcomes (10, 14). Psychosocial factors are linked to ill health and poor functional outcomes (15, 16). Aspects of close relationships and negative psychosocial work characteristics have been reported independent predictors of poor health (17). It has also been shown that powerlessness was associated with greater activity limits and greater health problems (18). A review about risk factors for functional decline in community-living elderly shows that there is good evidence that low level of social contacts and poor emotional social support are associated with poor functional outcomes (19). Another study shows that older persons with less psychosocial resources were particularly at risk in developing disabilities (15).

Using longitudinal data from the Dutch GLOBE study we examine the association between SES and mobility decline and whether this can be explained by psychosocial factors (life events, long term difficulties, coping styles, social support, and personality characteristics), independent of disease severity, comorbidity, and health-related behavior, among persons suffering from one or more of four chronic diseases (asthma/ chronic obstructive pulmonary disease (COPD), heart disease, diabetes mellitus, and low back pain).

METHODS

Study population

Data were obtained from the GLOBE (a Dutch acronym for Health and Living Conditions of the Population of the city of Eindhoven and surroundings) study, a longitudinal study that started in 1991 in the south east of the Netherlands, aimed at explaining SES inequalities in health (20). It is based on a cohort of 15 to 74 year old, non-institutionalized people of Dutch nationality. In 1991 the study started with a baseline measurement consisting of a postal survey and an oral interview. A sample of 27,070 inhabitants of 18 municipalities received a postal questionnaire; the sample was drawn from population registers stratified by age and postal code (45-74 years old and people from the highest and lowest SES groups were overrepresented). The response rate of the postal survey was 70.1% resulting in a study population of 18,973 respondents. A sub-sample of 3,968 persons drawn from respondents to the postal survey was approached for an oral interview. Participants completed an interview and a self-administered questionnaire; the response was 72.2% (n=2,867). In this sub-sample, people who reported one or more of the following chronic diseases were overrepresented: asthma/COPD, heart disease, diabetes mellitus, and severe low back pain. For the analyses presented in this paper, respondents suffering from at least one of the four chronic diseases were selected, resulting in 2,015 respondents in 1991. Response at follow-up in 1997 was 69.8% of the 2,015 respondents in 1991, resulting in 1,407 respondents. For 23 participants outcome data were missing, leaving 1,384 participants for the present analyses. The number of respondents in each group was: asthma/COPD; n=465, heart disease; n=788, diabetes mellitus; n=137, and low back pain; n=707. There were 580 respondents who suffered from more than one condition.

Measures

Socioeconomic status. We used three SES indicators: level of education, equivalent income, and occupational class. Three categories of level of education were created: high (university, higher vocational training); medium (higher secondary, intermediate vocational training); low (lower secondary, lower vocational training). Occupational class was measured according to Erikson, Goldthorpe, and Portocarero (EGP) classification (21) that originally consisted of 11 categories. In this study four categories were created: (1) (higher) grade professionals; (2) routine non-manual workers and high skilled manual workers; (3) self-employed; and (4) low skilled and unskilled manual workers. Net equivalent income per month was measured by adding all income components of all household members adjusted for household

size. Three categories of equivalent income, in euros, were distinguished: high (>947); medium (658-947); and low (<658). Education data were missing for 28 participants; this was 171 for occupation and 215 for income.

Physical mobility. Physical mobility was measured with the Nottingham Health Profile (NHP) (8). The NHP is a self-administered questionnaire designed to measure perceived health problems (22). The part that measures physical mobility consists of 8 statements that reflect problems with mobility (see appendix). Respondents had to answer 'yes' or 'no' to each of these statements. Because the statements vary in severity, they have been weighted by means of Thurstone's Method of Paired Comparisons (23). The minimum score is 0 and the maximum score is 100, which represents a situation where the respondent answers affirmatively to all the statements. The higher the score, the greater the mobility problems are. To determine whether there was a decline or not between 1991 and 1997, a reliable change index was computed using Edwards-Nunnally method of determining improvement rates. This method takes into account the measurement reliability of the NHP mobility scale (24). Decline conform the Edwards-Nunnally method was defined as a 1991 - 1997 difference score greater than 6.

Psychosocial factors. Psychosocial factors included life events, long term difficulties, coping styles, social support, and personality characteristics. Life events were measured by an nine-item questionnaire about negative events in the past year: moved to another house, substantial drop in income, victim of robbery/theft etc., becoming unemployed, serious illness of partner or other family member, serious illness of parents (or in-laws), child, brother, sister, or good friend, and divorce. Three categories were created; 0, 1, 2, or more life events. Long lasting difficulties were measured with an adapted version of the Dutch Long Standing Difficulties List (25). Three different types of long lasting difficulties were distinguished: difficulties with health problems of significant others, problems with relationships including social contacts with parents, partner, and neighbors, and situational problems including problems with housing and financial problems. For every subscale three categories were created; 0, 1, 2, or more problems. Seven coping styles were distinguished: inactive problem focusing, avoidance behavior, depressive reaction pattern, low social support seeking, palliative reaction pattern, low expression of emotions, and pessimism (26). Each coping variable was divided into tertiles. Two dimensions of social support were measured: emotional and instrumental support referring to three significant others; scores were divided into tertiles. Personality characteristics included neuroticism and locus of control. Neuroticism was measured with the 12-item Dutch version of the Eysenck Personality Questionnaire (27); the score was divided into tertiles. Finally, external locus of control was measured by means of the

11-item Dutch version of Rotter's locus of control scale (28). This variable was also classified into tertiles.

Disease severity and comorbidity. Disease-specific questionnaires on asthma/COPD (29), heart disease (30, 31), diabetes mellitus (32), and low back pain (33, 34) were used to determine the severity of the chronic disease. Comorbidity was defined as the number of other diseases that respondents reported. Comorbidity was measured with two variables. The first variable reflects the number of additional severe comorbid diseases that people had, the second variable indicates the number of less severe comorbid diseases. Fuller descriptions of these variables can be found elsewhere (6-8).

Behavioral factors. Behavioral factors included smoking (never, former, current), alcohol consumption (no, low, moderate, excessive), physical activity (no, light, moderate, frequent), and body mass index (BMI) ($<20 \text{ kg/m}^2$, $20\text{-}27 \text{ kg/m}^2$, $>27 \text{ kg/m}^2$). Behavioral factors are described in more detail elsewhere (35).

Sociodemographics. Sociodemographics included age (continuous), sex, and marital status (never married, married, divorced, widowed)

Statistical analyses

All analyses were performed using SPSS version 11.5. A chi-square test was used to determine whether there were differences in the percent of people with a decline in mobility between SES groups. To evaluate the association between SES and psychosocial factors, logistic regression models were fitted. Psychosocial factors were the dependent variables in these models and were therefore dichotomized as the worst category versus the other ones. The highest SES group was always the reference category. These analyses were adjusted for sociodemographics, disease severity, and comorbidity. Logistic regression models were also fitted to study the association between psychosocial factors on the one hand and a decline in mobility between 1991 and 1997, as the dependent variable, on the other hand, adjusting for sociodemographics, baseline mobility, disease severity, and comorbidity. Finally, the main logistic regression analyses were carried out to study the association between SES and mobility decline. The model adjusted for sociodemographics, baseline mobility, disease severity, comorbidity, and behavioral factors. To examine the contribution of psychosocial factors, they were separately introduced into that model.

RESULTS

The mean age of the study sample ($n=1,384$) was 53.7 years (standard deviation (SD)=12.5); 50.1% were women. Table 1 shows the percentage of people in each SES group and the percentage of people with a decline in mobility according to SES. More people with low education, low occupational level or low income had a decline in mobility than those in higher SES groups. For example, 37.2% of the people with low education had a decline in mobility between 1991 and 1997 in comparison to 25.4% with high education.

Table 1. Percentage of mobility decline according to socioeconomic status

Socioeconomic status	n=1,384	Mobility decline	
	%	%	p-value
Education ^a			
High	14.5	25.4	<0.01
Medium	35.0	31.3	
Low	48.4	37.2	
Occupation ^b			
1	30.1	27.3	0.01
2	24.9	33.7	
3	3.4	38.3	
4	29.3	37.0	
Income ^c			
High	30.1	25.5	<0.01
Medium	28.2	34.6	
Low	26.2	39.1	

^a Education: high (university, higher vocational); medium (higher secondary, intermediate vocational); low (lower secondary, lower vocational)

^b Occupation: 1 (higher) grade professionals; 2 routine non-manual workers or high skilled manual workers; 3 self-employed; 4 low skilled and unskilled manual workers

^c Equivalent income in euros: high (>947.04); medium (657.53-947.04); low (<657.53)

Generally, people with low SES have a more negative psychosocial profile than people in high SES groups (Table 2). Inactive problem focusing, an avoiding coping style, and low locus of control were statistically significantly associated with low education, low occupational level and low income. Low SES showed a particularly increased risk of low locus of control: odds ratios (OR) were 7.56 (95% confidence interval (CI): 4.57-12.47) in the lowest education group, 4.72 (95% CI: 3.35-6.66) in the group with low skilled and unskilled manual workers, and 3.33 (95%

Table 2. Odds ratios (OR) and 95% confidence interval (CI) of adverse psychosocial factors by socioeconomic status *

Psychosocial factors	Education ^a		Occupation ^b		Income ^c		
	Medium	Low	2	3	4	Medium	Low
Life events (>=2)	1.42 (0.91-2.21)	1.45 (0.94-2.25)	1.39 (0.97-2.00)	1.61 (0.77-3.37)	1.34 (0.94-1.91)	1.03 (0.72-1.49)	1.52 (1.07-2.17)
Long term difficulties							
Health (>=2)	1.06 (0.64-1.75)	1.21 (0.74-1.97)	1.29 (0.86-1.96)	2.06 (0.96-4.41)	1.21 (0.81-1.81)	0.75 (0.50-1.14)	1.32 (0.89-1.95)
Relationships (>=2)	0.96 (0.66-1.39)	0.77 (0.53-1.12)	0.94 (0.68-1.31)	0.75 (0.36-1.55)	0.83 (0.60-1.13)	0.74 (0.54-1.02)	0.74 (0.53-1.02)
Situation (>=2)	1.60 (0.99-2.58)	1.46 (0.91-2.34)	1.46 (0.98-2.15)	0.76 (0.30-1.94)	1.24 (0.85-1.81)	0.91 (0.61-1.36)	1.84 (1.27-2.67)
Coping							
Inactive problem focusing	1.21 (0.80-1.84)	3.30 (2.23-4.90)	1.53 (1.10-2.12)	2.61 (1.38-4.95)	3.06 (2.25-4.95)	1.63 (1.20-2.22)	2.18 (1.60-2.99)
Depressive reaction	1.01 (0.68-1.52)	1.28 (0.87-1.89)	1.06 (0.76-1.49)	0.83 (0.40-1.73)	0.99 (0.72-1.37)	0.96 (0.69-1.33)	1.47 (1.06-2.03)
Avoidance	1.36 (0.88-2.10)	2.37 (1.56-3.60)	1.46 (1.04-2.06)	2.17 (1.12-4.18)	1.77 (1.28-2.46)	1.31 (0.95-1.82)	1.83 (1.32-2.54)
Low social support seeking	1.33 (0.90-1.99)	1.73 (1.17-2.54)	0.99 (0.71-1.37)	1.44 (0.76-2.72)	1.11 (0.81-1.52)	1.15 (0.84-1.57)	1.08 (0.78-1.51)
Palliative reaction	1.63 (1.12-2.38)	1.47 (1.01-2.13)	1.27 (0.93-1.72)	1.19 (0.62-2.28)	1.17 (0.87-1.58)	1.15 (0.85-1.55)	1.62 (1.19-2.20)
Low expression of emotions	1.03 (0.69-1.54)	1.64 (1.12-2.41)	0.84 (0.60-1.16)	1.47 (0.77-2.80)	1.14 (0.84-1.55)	1.50 (1.10-2.04)	1.12 (0.86-1.66)
Pessimism	0.79 (0.55-1.12)	0.93 (0.66-1.33)	1.05 (0.78-1.43)	1.58 (0.79-3.17)	1.08 (0.80-1.45)	1.20 (0.89-1.61)	1.23 (0.90-1.67)
Poor social support							
Emotional	1.21 (0.82-1.77)	1.49 (1.03-2.16)	1.03 (0.74-1.44)	0.98 (0.49-1.96)	1.26 (0.92-1.73)	1.21 (0.88-1.66)	1.34 (0.97-1.85)
Instrumental	0.88 (0.62-1.26)	0.79 (0.55-1.11)	1.06 (0.77-1.44)	0.47 (0.22-0.99)	1.01 (0.75-1.37)	1.24 (0.92-1.68)	1.03 (0.75-1.41)
Personality							
High neuroticism	1.77 (1.16-2.71)	1.90 (1.26-2.88)	1.71 (1.22-2.39)	1.25 (0.60-2.60)	1.58 (1.15-2.19)	0.98 (0.71-1.37)	1.33 (0.96-1.84)
Low locus of control	2.53 (1.50-4.24)	7.56 (4.57-12.47)	2.46 (1.72-3.52)	4.01 (2.07-7.79)	4.72 (3.35-6.66)	2.42 (1.73-3.38)	3.33 (2.36-4.69)

* Adjusted for age, sex, marital status, disease severity, comorbidity

a, b, c See notes in Table 1, the highest SES group is always the reference group (not shown)

CI: 2.36-4.69) in the lowest income group. Long term difficulties with respect to health and relationships, and pessimistic coping style were not significantly associated with any of the SES indicators.

Table 3 shows the results of the association between psychosocial characteristics and mobility decline. Only low expressions of emotions (OR: 1.36, 95% CI: 1.00-1.84) and high neuroticism (OR: 1.68, 95% CI: 1.23-2.31) were statistically significant predictors of mobility decline. Results were similar without adjustment for disease severity and comorbidity (data not shown).

Generally, odds ratios of mobility decline, adjusted for sociodemographics, baseline mobility, disease severity, comorbidity, and health-related behavior were higher in low SES groups in comparison to high SES groups (Table 4). Highest risks were found for low income: odds ratios were 1.79 (95% CI: 1.24-2.57) for lowest income group and 1.34 (95% CI: 0.94-1.90) for the medium income group. Odds ratios of mobility decline for low education were not statistically significant; odds ratios were 1.04 (95% CI: 0.68-1.59) for the medium education group and 1.15 (95% CI: 0.75-1.75) in the lowest education group. Life events, long term difficulties, coping styles, social support, and personality characteristics were separately introduced into the first model. Psychosocial factors hardly reduced the odds ratios of the first model. Adjusting for the seven different coping styles even slightly increased the odds ratios. The odds ratio of mobility decline in the lowest income group remains statistically significant even after adjusting for the different psychosocial factors.

In additional analyses (data not shown) the psychosocial factors were added to the final model as continuous variables and we also determined the role of all psychosocial items individually. This did, however, not change our general findings that psychosocial factors hardly reduced to odds ratios of mobility decline. Furthermore, the role of psychosocial factors in explaining the association between SES and mobility decline was not different between the four chronic disease groups. Interactions between life events and long term difficulties on the one hand and coping, social support, and personality on the other hand were not statistically significant and did not contribute to the SES/ mobility decline relationship either.

Table 3. Odds ratios (OR) and 95% confidence interval (CI) for mobility decline by psychosocial characteristics *

Psychosocial factors		Mobility decline 97-91	
		OR	95% CI
Life events	lowest tertile	1.00	
	middle tertile	1.07	0.81-1.41
	highest tertile	1.11	0.81-1.51
Long term difficulties	Health		
	lowest tertile	1.00	
	middle tertile	0.89	0.66-1.14
	highest tertile	1.35	0.96-1.89
	Relationships		
	lowest tertile	1.00	
	middle tertile	1.15	0.82-1.61
	highest tertile	1.02	0.77-1.35
	Situation		
	lowest tertile	1.00	
	middle tertile	1.25	0.91-1.71
	highest tertile	1.21	0.87-1.66
Coping	Inactive problem focusing		
	lowest tertile	1.00	
	middle tertile	0.94	0.70-1.25
	highest tertile	0.86	0.64-1.16
	Depressive reaction		
	lowest tertile	1.00	
	middle tertile	0.87	0.65-1.16
	highest tertile	1.07	0.78-1.47
	Avoidance		
	lowest tertile	1.00	
	middle tertile	0.98	0.74-1.30
	highest tertile	1.14	0.84-1.54
	Low social support seeking		
	lowest tertile	1.00	
	middle tertile	1.11	0.82-1.50
	highest tertile	1.15	0.86-1.54
	Palliative reaction		
	lowest tertile	1.00	
	middle tertile	0.91	0.66-1.24
	highest tertile	1.05	0.79-1.40
	Low expression of emotions		
	lowest tertile	1.00	
	middle tertile	1.07	0.78-1.51
	highest tertile	1.36	1.00-1.84
	Pessimism		
	lowest tertile	1.00	
	middle tertile	1.14	0.84-1.53
	highest tertile	1.09	0.81-1.47
Poor social support	Emotional		
	lowest tertile	1.00	
	middle tertile	1.05	0.78-1.42
	highest tertile	0.95	0.71-1.28
	Instrumental		
	lowest tertile	1.00	
Personality	middle tertile	0.93	0.70-1.23
	highest tertile	0.90	0.67-1.22
	High neuroticism		
	lowest tertile	1.00	
	middle tertile	1.13	0.84-1.54
	highest tertile	1.68	1.23-2.31
	Low locus of control		
	lowest tertile	1.00	
	middle tertile	1.08	0.80-1.46
	highest tertile	1.25	0.91-1.70

* Adjusted for age, sex, marital status, baseline mobility, disease severity, comorbidity

Table 4. Odds ratios (OR) and 95% confidence interval (CI) for mobility decline by socioeconomic status

	Adjusted for confounders only *		Adjusted for confounders and following variable									
			Life events		Long term difficulties		Coping		Social support		Personality	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Education ^a												
High	1.00		1.00		1.00		1.00		1.00		1.00	
Medium	1.04	0.68-1.59	1.04	0.68-1.58	1.03	0.67-1.58	1.08	0.70-1.66	1.04	0.68-1.59	0.99	0.64-1.52
Low	1.15	0.75-1.75	1.15	0.75-1.75	1.14	0.75-1.74	1.21	0.78-1.88	1.15	0.75-1.75	1.08	0.70-1.69
Occupation ^b												
1	1.00		1.00		1.00		1.00		1.00		1.00	
2	1.33	0.93-1.89	1.32	0.93-1.88	1.31	0.92-1.87	1.35	0.95-1.93	1.32	0.93-1.89	1.29	0.90-1.84
3	1.50	0.72-3.15	1.50	0.71-3.15	1.48	0.70-3.14	1.74	0.82-3.69	1.50	0.71-3.16	1.46	0.69-1.08
4	1.43	1.01-2.03	1.43	1.01-2.03	1.42	1.00-2.02	1.51	1.05-2.17	1.43	1.01-2.03	1.41	0.98-2.02
Income ^c												
High	1.00		1.00		1.00		1.00		1.00		1.00	
Medium	1.34	0.94-1.90	1.34	0.95-1.90	1.38	0.97-1.96	1.38	0.97-1.97	1.33	0.94-1.90	1.35	0.95-1.93
Low	1.79	1.24-2.57	1.78	1.24-2.57	1.76	1.22-2.54	1.85	1.27-2.70	1.78	1.24-2.57	1.76	1.21-2.55

* Age, sex, marital status, baseline mobility, disease severity, comorbidity, smoking, alcohol consumption, physical activity, BMI

^{a, b, c} See notes in Table 1

DISCUSSION

In a chronically ill population, low occupational level and low income predicted an increased risk of mobility decline during six years of follow-up in comparison to people with a higher occupational level and income, even after controlling for sociodemographics, disease severity, comorbidity, and health-related behavior. Low SES was also associated with a more negative psychosocial profile. There was a weak association between psychosocial factors and a decline in mobility; only low expressions of emotions and high neuroticism were significant predictors of a decline in mobility between 1991 and 1997. Psychosocial factors could not explain the SES differentials in mobility decline because additional adjustment for psychosocial factors hardly reduced the SES-related odds ratios of mobility decline.

Psychosocial factors were measured extensively allowing a comprehensive assessment of the role of these factors in the association between SES and mobility decline. However, there could be other psychosocial factors, which we did not measure, involved in the association between SES and mobility decline. For example, job related stressors that are known to be related to low SES and poor health outcomes were not included (13, 17, 36, 37). These stressors were not included because this would substantially reduce the sample size (only the employed). Furthermore, we only adjusted for psychosocial factors at baseline, information about change in psychosocial factors during the life course was not available. Assuming that psychosocial characteristics develop more unfavorably over time in low SES groups than in high SES groups, the contribution of psychosocial factors may have been underestimated. In addition, the role of psychosocial factors in the association between low SES and mobility decline was determined after control for disease severity, comorbidity, and health-related behavior. There is some evidence that psychosocial factors affect health via behavioral factors (13, 38). Therefore, we might have underestimated the role of psychosocial variables in the SES/mobility decline relationship. However, in additional analyses, where we did not control for behavioral factors, psychosocial factors still explained only little of the SES differentials in mobility decline (not tabulated).

In this study we found only a weak association between psychosocial factors and mobility decline. This is in contrast with other studies that show much stronger associations between psychosocial factors and functional outcomes (2, 10, 15, 17, 19). Most of these studies were carried out in middle-aged and older populations, our study has a rather wide age range. However, in additional analyses where we conducted the same analyses in a group over 55 years old, we found similar results (data not shown). Similar results between psychosocial factors and mobility decline

were also found in additional analyses with mobility decline as a continuous variable. Stronger associations between psychosocial factors and baseline mobility were found in additional cross-sectional analyses (not tabulated). This is consistent with a study by Mackenbach et al. (2001) where the authors find significant cross-sectional associations between various psychosocial characteristics and physical disability but no significant longitudinal associations. A reason for not finding these longitudinal associations could be the relatively short period of follow-up. In addition, the possibility of reversed causation between psychosocial factors and mobility decline cannot be excluded in the cross-sectional analyses.

Factors that we did not include, which could also be important in explaining the SES differentials in mobility decline, are material factors. Income, being the strongest predictor of mobility decline in our study, suggests that the direct availability of financial resources might be important (2, 39). A lack of material resources held by individuals and the differential availability of public resources, such as schooling, health care, social welfare, and working conditions, might be important in explaining SES differences in health. On the other hand, long term situational difficulties, which included financial problems, were not strongly associated with SES and mobility decline in this study. In additional analyses with income, where we also adjusted for difficulties with paying bills for food, rent or electricity (or related aspects) in the past year, low income remained a significant predictor of mobility decline (not tabulated).

This study has some limitations. First, we did not have an objective measure of physical function, our outcome is rather subjective because it was entirely based on self-reports. People with a negative psychosocial profile may report greater problems concerning their physical function, without objective evidence for their reported worse physical performance. This could have led to an overestimation of the association between psychosocial factors and mobility decline. In this study, however, we found only a weak association between psychosocial factors and mobility decline. In addition, our study has a longitudinal character and therefore the potential for this effect is minimized because this would be present at baseline as well as follow-up. Second, baseline NHP-data were collected during an oral interview, while NHP-data in 1997 with a postal questionnaire. It is possible that people were more likely to admit health problems in the postal survey, therefore we could have overestimated the number of people with a mobility decline. However, it remains unclear whether this affected the association between SES and mobility decline and the minor role of psychosocial factors therein. Third, baseline response was 72.2%; and was lower in low SES groups. Attrition due to mortality and non-response was also higher in low SES groups ($p < 0.001$) (7). Response at follow up in 1997 was 69.8%. Participants without follow-up data also had statistically significantly

worse NHP-scores at baseline ($p < 0.01$) in comparison to persons who remained in the study. The association between SES and mobility decline might therefore be underestimated in this study. Finally, occupation data and income data were missing for 171 and 215, respectively. These persons more often had a lower educational level compared with persons with full data. This may also have led to an underestimation of the effect of occupation and income on mobility decline.

In summary, psychosocial factors (and also health-related behaviors, disease severity, and comorbidity) cannot explain SES differentials in mobility decline in a chronically ill population. Our findings will not give us new tools for prevention and intervention strategies in order to reduce physical disability and particularly the SES differences therein.

APPENDIX

Mobility part of the Nottingham Health Profile

1. I can only walk indoors
2. I find it hard to bend
3. I'm unable to walk at all
4. I have trouble getting up and down stairs or steps
5. I find it hard to reach for things
6. I find it hard to dress myself
7. I find it hard to stand for long
8. I need help to walk outside

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4

Explanations of socioeconomic differences in changes in physical function in older adults

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ABSTRACT

Objective. This study examines the association between socioeconomic status and changes in physical function in younger- (aged 55-70 years) and older-old (aged 70-85 years) adults and seeks to determine the relative contribution of diseases, behavioral, and psychosocial factors in explaining this association.

Methods. Data were from 2,366 men and women, aged 55-85 years, participating in the Longitudinal Aging Study Amsterdam (LASA). Two indicators of socioeconomic status were used: education and income. Physical function was measured by self-reported physical ability over nine years of follow-up.

Results. In older adults, low socioeconomic status was related to a poorer level of physical function during nine years of follow-up. In subjects who were between 55 and 70 years old, there was an additional significant socioeconomic-differential decline in physical function, while socioeconomic differentials did not further widen in subjects 70 years and older. Behavioral factors, mainly BMI and physical activity, largely explained the socioeconomic differences in physical function in the youngest age group, while psychosocial factors reduced socioeconomic status differences most in the oldest age group.

Conclusions. The findings indicate age-specificity of both the pattern of socioeconomic status differences in function in older persons and the mechanisms underlying these associations.

INTRODUCTION

Low socioeconomic status (SES) is related to poor physical function and the development of physical disabilities in older adults (1-6). Disability, usually defined as the inability to carry out the usual tasks of daily living, is established as a powerful measure of health status in old age (7). In addition, physical disability in older persons is related to many diseases and has been shown to be a predictor of mortality in older adults. Therefore, it is important to understand what factors to intervene upon in order to reduce physical disabilities and the SES differences therein.

Several factors, including biomedical, behavioral, and psychosocial factors, may be important in explaining these SES differences in physical function. Biomedical factors, such as cardiovascular disease, stroke, and diabetes, are related to both low SES and adverse functional outcomes (8-12). A recent study shows that biomedical factors, including a wide range of diseases and biological risk factors, explained part of the SES differences in mobility limitation incidence in older adults (5). Low SES is also related to many adverse behavioral factors, such as smoking, excessive alcohol consumption, and decreasing physical activity, that in turn are related to poor functional outcomes (13-15). Finally, psychosocial factors may be important in explaining SES differences in poor functional outcomes because people with low SES generally have fewer psychosocial resources than people with high SES (16-18). Psychosocial factors, such as control beliefs and social support, are also linked to ill health and poor functional outcomes (19-21).

The present study examines the association between SES and longitudinal changes in physical function in a representative sample of Dutch older adults, who participated in the Longitudinal Aging Study Amsterdam. The contribution of a number of potential explanatory factors to explain SES differences in physical function has been studied before. In this study, however, we will integrate several explanatory factors, including diseases, behavioral, and psychosocial factors in order to determine the relative contribution of these factors to the explanation of SES inequalities in changes in physical function. Moreover, it is examined whether the effect of SES is different for younger- and older-old and for men and women.

METHODS

Study population

Data were from the Longitudinal Aging Study Amsterdam (LASA), an ongoing cohort study on predictors and consequences of changes in well-being and autonomy in older persons in the Netherlands (22). A random sample of persons, 55 to 85 years of age, was drawn from the population registers of 11 municipalities. This sample was originally recruited for the NESTOR study on Living Arrangements and Social Networks of older adults (LSN) in 1992 (23). The response rate was 62.3% ($n=3,805$). About eleven months after the LSN interview, 3,107 (81.7%) subjects were enrolled in the main baseline interview of LASA conducted between September 1992 and September 1993. At the first follow-up in 1995/1996, 2,545 participants took part in the study; 416 (13.4%) had died and 146 (4.7%) were lost to follow-up for other reasons (refusal, too frail, could not be contacted). At the second follow-up measurement in 1998/1999, 2,076 persons participated in the study; 343 (13.5%) had died and 126 (5.0%) were lost to follow-up for other reasons. At the third follow-up measurement in 2001/2002, 1,691 persons took part in the study; 289 (13.9%) had died and 96 (4.6%) were lost to follow-up for other reasons. For the present study, respondents were selected with data on physical function at at least two measurements, resulting in 2,366 participants.

Measures

Socioeconomic status. Two indicators of SES were used, education and household income. Categories for education were: high (university, college, higher vocational, general secondary, and intermediate vocational education) medium (general intermediate, and lower vocational education), and low (elementary education or less). For monthly net household income, three categories were distinguished: high (greater or equal to 1,035 euro), medium (between 625 and less than 1,035 euro), and low (less than 625 euro). For participants with a partner living in the household, household income was multiplied by 0.7 to make it comparable to the incomes in a one-person household. Income data at baseline were missing for 342 respondents. For 290 respondents imputations were made using income data collected at the LSN measurement in 1992 or follow-up measurements (3). For 52 participants income data were missing at all measurements; therefore they were excluded from the analyses with income, resulting in 2,314 participants for the analyses with income.

Physical function. Physical function was measured by self-report. At each measurement cycle questions were asked about the degree to which the respondent had difficulty performing six usual daily activities: walking up and down a 15-step staircase without resting, getting (un)dressed, getting up from and sitting down in a chair, cutting own toenails, walking 5 minutes outdoors without resting, and using own or public transport (24). The response categories were: (0) not able to do, (1) only with help, (2) with much difficulty, (3) with some difficulty, and (4) without difficulty. The sum score of the six items ranged from 0 to 24, with lower scores indicating more limitation in physical function.

Covariates. Sociodemographics included age and sex. The presence of diseases at baseline was determined by asking the participants whether they had any of the following chronic diseases: cardiac disease, peripheral arterial disease, stroke, diabetes mellitus, lung disease, cancer, and arthritis (25).

Lifestyle factors, measured at baseline, included smoking, alcohol use, physical activity, and body mass index (BMI). Four categories of smoking were created: current, former, never and missing. Categories of alcohol use were: not drinking, moderate drinking, excessive drinking (at least three glasses daily), and missing. Physical activity was defined as the total number of physical activities performed in the past two weeks including walking, cycling, gardening, performing light and heavy household, and a maximum of two sports. Four categories were created: 6-7, 4-5, 1-3, and missing. BMI (weight in kilograms divided by height in meters squared) was categorized as lower than 25 kg/m², between 25 and 30 kg/m², greater or equal to 30 kg/m², and missing.

Psychosocial factors, measured at baseline, included partner status (presence of a partner, yes or no), network size, instrumental and emotional social support, mastery, and self-efficacy. The size of the personal network was determined by asking the respondent to identify the persons, other than their partner, whom they had frequent contact with and who were important to them (range is 0-75) (26). For a maximum of nine network members with whom contact was most frequent, information was collected regarding the intensity of received instrumental and social emotional support (range is 0 (no social support)-36). Mastery was measured with a five-item version of the Pearlin Mastery Scale (range is 5 (lowest)-25) (27). Self-efficacy was measured with a 12-item version of the General Self-efficacy Scale of Sherer et al. (range is 12 (lowest)-60) (28, 29). Missing values for network size (n=112), instrumental support (n=118), emotional support (n=120), mastery (n=64), and self-efficacy (n=107) were replaced by group means in order to maintain an optimal sample size for the analyses.

Statistical analyses

Differences in baseline characteristics between SES groups were determined using chi-square test for categorical variables and t-test statistics for continuous variables. Multilevel analyses, using linear mixed models in SPSS, were used to examine the association between SES and the longitudinal change in physical function. A multilevel analysis has the advantage that it is a suitable technique for repeated measurement analyses. Five models were fitted, the first model adjusted for age, sex, and longitudinal time (in terms of the number of the observation). The model also included two interaction terms that were statistically significant; the interaction between age and longitudinal time and between sex and longitudinal time. In model 2, 3, and 4, diseases, behavioral factors, and psychosocial factors were introduced separately into the first model. The fifth model included all variables of the previous models. Analyses were performed using SPSS version 12.0.

RESULTS

Preliminary analyses indicated a significant interaction between SES and age in this study, therefore all analyses were performed in two age groups: younger than 70 years and 70 years and older. Interactions between SES and sex were not statistically significant. Table 1 shows baseline characteristics of the total study population by SES and age group. In both age groups there were more women in low SES groups and respondents were older in low SES groups. People with higher education or income had significantly higher baseline physical function scores, indicating better function, compared to low SES groups in both age groups ($p < 0.01$). There was no strong association between the prevalence of diseases and SES. In the lowest age group, only the prevalence of peripheral atherosclerosis and arthritis was significantly higher in both the lowest education and income groups. In the highest age group, the prevalence of arthritis was also significantly higher in people with a low level of education. In the lowest SES groups, there were more non-smokers and less people drank alcohol in both age groups ($p < 0.01$). Persons younger than 70 with high SES were significantly more physically active compared to the lowest SES groups ($p < 0.01$). In the highest age group the association was less clear. In both age groups, people with low SES had a significantly higher BMI compared to people with high SES ($p < 0.01$). Low SES was also associated with a smaller network, less emotional support, lower feelings of mastery (only in the lowest age group), and lower self-efficacy compared to people with high SES.

Mean physical function scores at the four measurements by age group according to SES are shown in table 2. Low SES groups had worse physical function scores at all measurements compared to high SES groups. The oldest age group had lower physical function scores at all measurements than the youngest age group.

Table 3 shows the results of the multilevel analyses for the group younger than 70 years and table 4 for those 70 years and older. The tables present the intercept and slope of the variable time for each model. The intercept is the parameter estimate (baseline score) of the middle and low SES group compared to the highest SES group. The slope is the coefficient of the SES*longitudinal time interaction term, indicating the effect of longitudinal time on physical function. The first model is graphically presented in figure 1. People younger than 70 years old in the lowest SES group not only had a significantly worse physical function score at baseline and follow-up, they were also declining significantly more over time compared to people in the highest SES group (Table 3: Model 1, Figure 1). Baseline differences between the lowest and the highest education group were larger in the oldest age group. SES differences did, however, not further widen over time in people of 70 years and older (Table 4: Model 1, Figure 1).

In model 2, 3, and 4 diseases, behavioral factors, and psychosocial factors were introduced separately into the first model. In the youngest age group (Table 3), the largest reduction in intercepts was found in model 3, due to behavioral factors, for both education and income. A percentage reduction in intercept from model 1 was computed by $(\text{intercept}_{\text{model 1}} - \text{intercept}_{\text{model 3}}) / (\text{intercept}_{\text{model 1}}) * 100\%$. The average reduction due to behavioral factors in the lowest SES groups was 65%. BMI and physical activity most reduced the regression coefficients; smoking and drinking did not further decrease the regression coefficients (not tabulated). Behavioral factors could not explain the SES-differential decline (slope). The SES differences in baseline physical function were completely explained when all factors of the previous models were added to the fifth model. The differences in slope, however, remained statistically significant.

In the oldest age group, the large differences in baseline physical function between the lowest and the highest SES group could not be fully explained by diseases, behavioral or psychosocial factors (Table 4). The largest reduction in intercept differences between the lowest and the highest SES group in this age group was found in model 4, due to psychosocial factors (on average 28%). In additional analyses, we determined which psychosocial factors were most important in reducing the regression coefficients. None of the psychosocial factors, however, had a large

Table 1. Baseline characteristics of the study population by socioeconomic status and age group

Baseline characteristics	<70 years n=1,262										>70 years n=1,104									
	Education					Income					Education					Income				
	High n=401	Medium n=419	Low n=442	p	High n=553	Medium n=445	Low n=248	p	High n=261	Medium n=311	Low n=532	p	High n=248	Medium n=339	Low n=481	p	High n=248	Medium n=339	Low n=481	p
Women, %	34.4	54.7	69.9	<0.01	41.0	55.7	75.8	<0.01	40.6	42.4	65.6	<0.01	39.1	41.9	68.2	<0.01	39.1	41.9	68.2	<0.01
Age, mean (SD)	62.1 (4.2)	62.4 (4.3)	63.2 (4.2)	<0.01	61.5 (4.0)	62.9 (4.2)	64.4 (4.2)	<0.01	77.4 (4.3)	76.7 (4.2)	78.0 (4.3)	<0.01	77.4 (4.2)	76.9 (4.3)	77.7 (4.3)	0.03	77.4 (4.2)	76.9 (4.3)	77.7 (4.3)	0.03
Physical function Baseline score (0-24), mean (SD)	23.4 (2.1)	23.1 (2.4)	22.4 (3.4)	<0.01	23.4 (1.8)	22.9 (2.7)	21.8 (4.0)	<0.01	21.3 (4.2)	20.8 (4.7)	19.1 (5.6)	<0.01	20.9 (4.3)	21.2 (4.3)	19.1 (5.8)	<0.01	20.9 (4.3)	21.2 (4.3)	19.1 (5.8)	<0.01
Diseases																				
Cardiac disease, %	13.5	15.3	12.2	0.42	13.4	14.0	14.1	0.95	23.1	25.2	23.8	0.83	24.3	24.9	23.9	0.95	24.3	24.9	23.9	0.95
Peripheral atherosclerosis, %	5.0	4.8	9.7	<0.01	3.1	9.2	10.1	<0.01	11.5	12.3	10.1	0.60	8.9	10.7	12.2	0.41	8.9	10.7	12.2	0.41
Stroke, %	1.0	2.6	2.5	0.19	1.3	2.0	4.0	0.04	5.8	6.8	6.8	0.84	5.7	6.5	6.7	0.86	5.7	6.5	6.7	0.86
Diabetes mellitus, %	3.5	3.8	4.8	0.63	3.4	5.4	3.2	0.22	5.8	6.8	8.6	0.33	6.5	6.2	8.4	0.44	6.5	6.2	8.4	0.44
Lung disease, %	7.5	7.2	10.6	0.13	7.2	9.2	10.5	0.27	13.1	13.2	12.5	0.95	14.6	11.9	12.8	0.63	14.6	11.9	12.8	0.63
Cancer, %	5.0	7.0	8.1	0.19	6.3	6.3	8.9	0.36	6.7	11.3	11.6	0.11	8.1	10.7	11.3	0.39	8.1	10.7	11.3	0.39
Arthritis, %	26.3	29.9	35.7	0.01	25.0	34.0	38.7	<0.01	33.8	31.3	40.3	0.02	34.4	32.6	40.0	0.07	34.4	32.6	40.0	0.07
Behavioral factors																				
Smoking, %																				
Non-smoker	15.5	26.5	32.4	<0.01	19.2	26.1	35.1	<0.01	23.4	26.7	39.3	<0.01	22.2	25.4	42.2	<0.01	22.2	25.4	42.2	<0.01
Former	49.9	41.5	33.3		46.5	40.2	32.3		45.2	45.0	28.9		50.0	42.5	28.5		50.0	42.5	28.5	
Smoker	28.4	23.2	25.3		28.2	24.9	21.4		18.8	19.0	15.4		15.3	20.4	16.0		15.3	20.4	16.0	
Missing	6.2	8.8	9.0		6.1	8.8	11.3		12.6	9.3	16.4		12.5	11.8	13.3		12.5	11.8	13.3	
Physical activity, %																				
6-7	29.9	28.6	20.1	<0.01	28.9	23.4	25.0	<0.01	11.5	11.9	9.2	<0.01	10.9	10.3	11.0	0.83	10.9	10.3	11.0	0.83
4-5	45.6	44.9	44.1		46.5	47.6	37.1		33.3	41.2	39.8		37.5	41.9	39.3		37.5	41.9	39.3	
0-3	22.2	19.6	29.6		21.9	22.0	31.5		48.3	42.1	39.7		46.0	42.5	42.4		46.0	42.5	42.4	
Missing	2.2	6.9	6.1		2.7	7.0	6.5		6.9	4.8	11.3		5.6	5.3	7.3		5.6	5.3	7.3	
BMI, %																				
<25	38.2	31.7	23.1	<0.01	35.6	27.6	26.2	<0.01	41.4	29.6	21.2	<0.01	34.3	31.0	23.3	<0.01	34.3	31.0	23.3	<0.01
25-<30	42.9	44.4	47.3		47.4	43.8	40.7		33.3	44.7	34.8		33.1	42.5	37.4		33.1	42.5	37.4	
≥30	10.7	14.1	18.6		8.9	18.0	21.0		10.0	14.8	24.2		16.9	12.4	23.5		16.9	12.4	23.5	
Missing	8.2	9.8	11.1		8.1	10.6	12.1		15.3	10.9	19.7		15.7	14.2	15.8		15.7	14.2	15.8	

* Score ranged from 0 to 24; higher scores indicate better physical ability

	1992/1993				1995/1996				<70 years				≥70 years				1998/1999				2001/2001			
	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)		
Education																								
High	400	23.4 (2.1)	389	23.0 (2.6)	363	22.7 (3.0)	329	22.3 (3.2)	261	21.3 (4.2)	257	19.9 (5.1)	173	18.9 (6.0)	113	18.8 (5.8)								
Medium	419	23.1 (2.4)	412	22.5 (3.3)	370	22.5 (3.2)	323	21.8 (3.7)	310	20.8 (4.7)	305	19.8 (5.6)	235	18.8 (5.5)	145	18.7 (5.6)								
Low	442	22.4 (3.4)	425	21.9 (3.7)	388	21.2 (4.4)	339	20.6 (4.6)	531	19.1 (5.6)	510	17.8 (6.1)	345	16.6 (6.5)	219	15.7 (6.4)								
Overall	1261	22.9 (2.7)	1226	22.5 (3.3)	1121	22.1 (3.6)	991	21.6 (3.9)	1102	20.1 (5.2)	1072	18.8 (5.8)	751	17.8 (6.2)	477	17.4 (6.2)								
Income																								
High	552	23.4 (1.8)	543	23.1 (2.4)	499	22.9 (2.6)	457	22.4 (2.9)	248	20.9 (4.3)	246	19.9 (5.1)	177	18.8 (5.8)	113	18.8 (5.9)								
Medium	445	22.9 (2.7)	432	22.4 (3.1)	394	21.9 (3.6)	355	21.3 (4.1)	339	21.2 (4.3)	326	19.6 (5.4)	247	18.2 (6.3)	157	18.0 (6.0)								
Low	248	21.8 (4.0)	236	21.3 (4.7)	218	20.6 (5.0)	176	19.8 (5.2)	479	19.1 (5.8)	465	18.0 (6.1)	318	17.2 (6.1)	204	16.2 (6.1)								
Overall	1245	22.9 (2.8)	1211	22.5 (3.3)	1111	22.1 (3.6)	988	21.6 (4.0)	1066	20.2 (5.1)	1037	19.0 (5.1)	742	17.9 (6.1)	474	17.4 (6.1)								

individual contribution. In the full model (Model 5), the intercept of low education still was significantly different from that of higher education.

Because the significant differences in slope between SES groups, that were found in the lowest age group, could not be explained by any of the variables that were added in model 2 to 4, we determined, in additional analyses, whether interactions between the explanatory factors and longitudinal time would reduce the slope differences. However, the coefficients were difficult to interpret which may be due to the complexity and potential over-fitting of the models and, therefore, did not add to the explanation of the slope differences.

DISCUSSION

In an older population, low SES was associated with poor physical function during nine years of follow-up. In contrast to people 70 years and older, where SES differentials in physical function did not further increase over time, SES differentials in physical function significantly increased over nine years in people who were younger than 70 years. Behavioral factors, mainly BMI and physical activity, explained a large part of the SES differentials in baseline physical function in the youngest age group. Behavioral factors could, however, not explain the SES-differential decline in physical function. In people 70 years and older, psychosocial factors reduced the SES differences most.

A recent study in women showed that SES inequalities in physical health may attenuate by the age of 70 (30). This latter study also found a strong cross-sectional association between SES and physical function, but no association with a decline in physical function (30). The present study shows that even in old age, SES differences in physical function were still present. In people of 70 years and older, SES differences in physical function did not increase over time, but still existed after nine years of follow-up. The lowest SES group probably consists of "healthy survivors" because of selection prior to baseline and attrition due to mortality during the follow-up. It may be due to this healthy survivor effect that SES differentials in physical function did not widen over time in this age group. This may also explain the weak association between the prevalence of diseases and SES that was found. Consequently, diseases explained only very little of the SES differences in physical function in the oldest age group.

In the youngest age group, a high percentage had no physical function problems at baseline (not tabulated). The decline in physical function that was found in this age group was mainly due to the onset of physical function problems rather

Figure 1. Longitudinal course of physical function * according to socioeconomic status in two age groups (<70 years old and ≥70 years old)
* Adjusted for age, sex, and interactions between age and longitudinal time, and sex and longitudinal time

Table 3. Intercept and slope of socioeconomic status on physical function in group <70 years old

	Model 1 *			Model 2 †			Model 3 ‡			Model 4 §			Model 5 ¶		
	Intercept	p	Slope	Intercept	p	Slope	Intercept	p	Slope	Intercept	p	Slope	Intercept	p	Slope
Education															
High	Ref.														
Medium	-0.16	0.45	-0.04	0.59	-0.08	-0.04	0.62	-0.07	0.73	-0.04	0.59	-0.14	0.49	-0.04	0.59
Low	-0.43	0.05	-0.24	<0.01	-0.24	0.26	<0.01	-0.07	0.74	-0.24	<0.01	-0.35	0.11	-0.24	<0.01
Income															
High	Ref.														
Medium	-0.22	0.26	-0.17	0.02	-0.05	0.79	-0.17	0.03	0.87	-0.17	0.02	-0.15	0.44	-0.17	0.02
Low	-0.83	<0.01	-0.36	<0.01	-0.62	0.01	-0.36	<0.01	-0.46	0.06	-0.36	<0.01	-0.56	0.02	-0.36

* Model 1: adjusted for age, sex, and interactions between age and longitudinal time, and sex and longitudinal time

† Model 2: adjusted for age, sex, interactions between age and longitudinal time and sex and longitudinal time, cardiac disease, peripheral arterial disease, stroke, diabetes mellitus, lung disease, cancer, arthritis

‡ Model 3: adjusted for age, sex, interactions between age and longitudinal time and sex and longitudinal time, smoking, drinking, BMI, and physical activity

§ Model 4: adjusted for age, sex, interactions between age and longitudinal time and sex and longitudinal time, partner status, network size, instrumental social support, emotional social support, mastery, and self-efficacy

¶ Model 5: all variables of previous models

Table 4. Intercept and slope of socioeconomic status on physical function in group ≥70 years old

	Model 1 *			Model 2 †			Model 3 ‡			Model 4 §			Model 5 ¶		
	Intercept	p	Slope	Intercept	p	Slope	Intercept	p	Slope	Intercept	p	Slope	Intercept	p	Slope
Education															
High	Ref.														
Medium	-0.74	0.12	0.16	0.39	-0.66	0.15	0.15	0.41	-0.76	0.10	0.16	0.39	-0.68	0.12	0.15
Low	-1.36	<0.01	0.01	0.94	-1.17	<0.01	-0.00	0.99	-1.16	<0.01	0.01	0.96	-1.04	0.02	0.01
Income															
High	Ref.														
Medium	0.30	0.52	-0.26	0.16	0.29	0.52	-0.25	0.17	0.18	0.68	-0.26	0.16	0.41	0.38	-0.26
Low	-0.86	0.06	-0.10	0.57	-0.67	0.12	-0.13	0.47	-0.80	0.05	-0.10	0.58	-0.59	0.19	-0.10

* Model 1: adjusted for age, sex, and interactions between age and longitudinal time, and sex and longitudinal time

† Model 2: adjusted for age, sex, interactions between age and longitudinal time and sex and longitudinal time, cardiac disease, peripheral arterial disease, stroke, diabetes mellitus, lung disease, cancer, arthritis

‡ Model 3: adjusted for age, sex, interactions between age and longitudinal time and sex and longitudinal time, smoking, drinking, BMI, and physical activity

§ Model 4: adjusted for age, sex, interactions between age and longitudinal time and sex and longitudinal time, partner status, network size, instrumental social support, emotional social support, mastery, and self-efficacy

¶ Model 5: all variables of previous models

than to a further functional decline related to an already existing disability. This may underlie the age-specificity that was found in both the pattern and the explanation of SES differences in physical function. Behavioral factors, in this study particularly high BMI and low physical activity, may be important in predicting the onset of physical function problems in the youngest age group. Adverse psychosocial factors, such as low social support and low self-efficacy, may cause a further decline in physical function in the oldest age group. Existing physical function problems may negatively influence someone's psychosocial profile, such as lower self-efficacy, which may further accelerate the decline in physical function as in a vicious circle (31). Such pathways and reciprocal associations need further detailed examination.

In this study, a wide range of different explanatory factors was considered in order to get more insight into the explanation of SES inequalities in physical function. A few drawbacks, however, have to be considered. The SES differential decline that was found in the youngest age group could not be explained. This suggests that other factors that were not measured in this study may have caused the widening over time in this age group. Furthermore, the contribution of the explanatory factors in reducing the SES differences and in particular the SES-differential decline in physical function could have been larger if these factors had also been considered longitudinally. However, there is evidence that behavioral factors, mainly smoking and alcohol consumption remain rather stable over time; physical activity showed greater variability over time. These changes in health behavior were, however, not related to SES (32). Psychosocial factors, such as network size and social support, probably also remained rather stable during the follow-up (26, 33). The prevalence of diseases, however, was more likely to have changed during the nine years of follow-up which may have led to an underestimation of the contribution of these factors. Finally, we must acknowledge that, had more objective information on diseases been available, the reduction in the strength of the SES effect might have been larger.

Loss to follow-up was a limitation of this study. Attrition was mainly due to mortality (74% of the total loss to follow-up). People who were lost to follow-up for other reasons had worse physical function scores at baseline, compared to those who remained in the study (data not shown). In addition, those who were lost to follow-up also had a significantly lower SES compared to our study population. The association between SES and physical function may therefore have been underestimated. Whether the relative contribution of explanatory variables was equally underestimated remains unknown.

This study contributes to the understanding of the mechanisms underlying the association between SES and physical function in older adults. In order to reduce physical disabilities in older adults and in particular the socioeconomic differences

therein, it may be helpful to improve health-related behaviors in younger-old adults and to intervene upon psychosocial characteristics in older-old adults. Further study is needed to confirm the age-specificity that was found in both the pattern and the explanation of socioeconomic differences in function in older adults.

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5

Association of inflammatory markers with socioeconomic status

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ABSTRACT

Objective. This study examines the association between socioeconomic status (SES) and inflammatory markers in well-functioning older adults and seeks to determine whether any association remains after adjusting for biomedical and behavioral factors typically related to elevated serum levels of inflammatory markers.

Methods. Data were from 3,044 men and women, aged 70-79 from Pittsburgh, PA and Memphis, TN participating in the Health, Aging and Body Composition study (Health ABC). Three indicators of SES were used: education, income, and ownership of financial assets. Serum levels of interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor- α (TNF- α) were measured.

Results. Low SES was associated with significantly elevated levels of IL-6, CRP, and TNF- α compared to high SES. Behavioral factors (including smoking, drinking, obesity) explained a substantial part of the inverse association between SES and inflammatory markers. Adjustment for prevalent diseases (including heart diseases, lung disease, and diabetes) associated with inflammation explained for less of the association.

Conclusions. This study suggest that even in old age, interventions to improve health behaviors, especially in low SES groups, may be useful in reducing risks associated with inflammation.

INTRODUCTION

Low socioeconomic status (SES) has recently been related to higher levels of inflammatory markers (1-4). This is important, as inflammation – being a biological response of the immune system – has been associated with increased morbidity and mortality across the age span, including in old age (5-7). Two recent studies show that persons with low SES have higher serum levels of C-reactive protein (CRP) than persons with high SES (1, 2). Another study found lower levels of interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α) in high SES groups, but the effect was non-linear and found in women only (3). Evidence relating inflammatory markers to SES is, however, limited at present especially regarding to what extent this association might be mediated by other health and behavioral factors commonly associated with low SES.

Increased levels of inflammatory markers, such as IL-6 and CRP, are observed in the presence of acute and chronic conditions including atherosclerosis (8), cerebrovascular disease (9, 10), coronary heart disease (11-14), congestive heart failure (15), and arthritis (16). Recent studies also show that depression and cognitive impairment are associated with elevated levels of inflammatory markers (17, 18). These health conditions are also more common in low SES groups and may thus contribute to the association between low SES and higher levels of inflammatory markers. Similarly, behavioral factors may also explain an association between low SES and increased inflammatory marker levels. Low SES is related to many adverse behavioral factors, such as smoking, excessive alcohol use, obesity, and physical inactivity (19). These factors are also related to higher levels of inflammatory markers (20-22).

Using data from a large sample of community-dwelling older adults this study examined the association between SES and several markers of inflammation (CRP, IL-6, and TNF- α). In addition, the present study sought to determine whether any association remained after adjusting for biomedical and behavioral factors typically related to elevated serum levels of inflammatory markers.

METHODS

Study population

The Health, Aging and Body Composition (Health ABC) study is a longitudinal cohort study consisting of 3,075 well-functioning, 70- to 79-year old, black and white men and women. Participants were identified from a random sample of White

Medicare beneficiaries and all age-eligible community-dwelling black residents in designated zip code areas surrounding Memphis, Tennessee, and Pittsburgh, Pennsylvania. Participants were eligible if they reported no difficulty in either walking one quarter of a mile, going up 10 steps without resting or performing any basic activity of daily living. Participants were excluded if they reported a history of active treatment for cancer in the prior three years, planned to move out of the study area in the next three years, or were currently participating in a randomized trial of a lifestyle intervention. Data, collected between April 1997 and June 1998, included an in-person interview and a clinic-based examination, with evaluation of body composition, clinical and sub-clinical diseases, and physical functioning.

Data on inflammatory markers were missing for 30 participants and SES data were missing for one participant, leaving 3,044 participants for the present analyses. All participants signed informed written consent forms approved by the institutional review boards of the clinical sites.

Measures

Socioeconomic status. Three indicators of SES were used: education, family income, and ownership of financial assets (23). Different SES indicators were used because these variables represent different dimensions of SES (24). Education is the socio-cultural dimension of SES and represents social class in an early stage of the life course compared to income representing a later life stage. Ownership of financial assets, as a proxy for wealth, describes availability of material resources accumulated over the life course. Categories for education were: less than 12 years, 12 years, and more than 12 years. Family income was defined as: wages, salaries, social security or retirement benefits, help from relatives, and rent from property. Five categories of family income from the year prior to interview were distinguished: less than \$10,000, between \$10,000 and less than \$25,000, between \$25,000 and less than \$50,000, greater than or equal to \$50,000, and missing ($n=371$, 12.2%). The number of assets a person reported was used as a third SES measure. Assets included: money market account, savings bonds or treasury bills, home ownership or investment property or housing, a business or farm, stock or stock mutual funds, and IRA or KEOGH account, and other investments. Three categories were created: none, one or two, and three to seven (23).

Inflammatory markers. Measures for the cytokines IL-6 and TNF- α and for CRP were obtained from frozen stored plasma or serum. Fasting blood samples were obtained in the morning, and after processing, the specimens were aliquoted into cryovials, frozen at -70°C , and shipped to the Health ABC Core Laboratory at the University of Vermont. Cytokines were measured in duplicate by enzyme-linked

immunosorbent assay (ELISA) kits from R&D Systems (Minneapolis, MN). The detectable limit was 0.10 pg/mL for IL-6 (by HS600 Quantikine Kit) and 0.18 pg/mL for TNF- α (by HSTA50 kit). Serum levels of CRP were also measured in duplicate by ELISA based on purified protein and polyclonal anti-CRP antibodies (Calbiochem, San Diego, CA). The CRP assay was standardized according to the World Health Organization First International Reference Standard with a sensitivity of 0.08 μ g/mL. Assays of blind duplicates collected for 150 participants showed an average interassay coefficient of variation of 10.3% for IL-6, 8.0% for CRP, and 15.8% for TNF- α .

Covariates. Sociodemographics included age, sex, race (black or white), study site (Memphis or Pittsburgh), and marital status (never married, previously married, married). For the analyses with family income we also controlled for whether the household included anyone in addition to the participant and his or her spouse. Behavioral factors included smoking (current, former, never smoker), cigarette use in pack-years, alcohol use during the past year (0, <1, 1-7, >7 drinks per week), physical activity (total kilocalories per kilogram per week), and body mass index (BMI, weight in kilograms divided by height in meters squared (kg/m^2)). Abdominal visceral fat (centimeters squared) was quantified by an analyses of CT scans by General Electric 9800 Advantage in Pittsburgh and a Siemens Somatron and Picker PQ2000S in Memphis. Presence of lung, heart, and cerebrovascular disease, diabetes mellitus, osteoarthritis, and cancer was determined using standardized algorithms considering self-report, use of specific medications, and clinical assessments. Depressed mood was assessed with the Center for Epidemiologic Studies Depression (CES-D) scale. A cutoff score of 16 was used as a criterion for major depressive symptoms (25). Cognitive impairment was defined as a Modified Mini-Mental State Examination (3MS) score less than 78 (26). Medications taken in the previous two weeks were recorded and coded according to the Iowa Drug Information System (27). By use of this system, the use of anti-inflammatory drugs and corticosteroids were assessed.

Statistical analyses

Analyses were performed using SPSS, version 12.0. Characteristics of the study population were stratified by race. Differences in main characteristics between black and white persons were determined using chi-square tests for categorical variables and t-test statistics or nonparametric Mann-Whitney statistics for continuous variables. A non-parametric test for differences in inflammatory marker levels was used because levels of inflammatory markers were not normally distributed. To compare serum levels of inflammatory markers across SES groups a Kruskal-Wallis

Table 1. Main characteristics of the study population

	Overall n=3,044	White n=1,780	Black n=1,264	p-value *
Age, mean (SD)	74.2 (2.8)	74.3 (2.9)	74.0 (2.9)	0.001
Women, %	51.4	47.5	57.0	<0.001
Memphis site, %	50.2	52.1	47.5	0.01
Married, %	56.5	67.5	42.6	<0.001
Education, %				
>12 years	42.0	53.5	26.0	<0.001
12 years	32.7	34.2	30.7	
< 12 years	25.1	12.3	43.4	
Income (\$), %				
≥50,000	14.1	21.0	4.4	<0.001
25,000-<50,000	27.9	35.1	17.7	
10,000-<25,000	34.2	27.8	43.3	
<10,000	11.6	3.7	22.8	
Missing	12.2	12.5	11.8	
Assets, %				
3-7	42.9	62.4	16.6	<0.001
1-2	39.7	28.8	55.8	
None	16.5	8.8	27.6	
Inflammatory markers				
IL-6, pg/mL, median (range)	1.83 (1.27-2.79)	1.74 (1.20-2.62)	1.99 (1.33-3.06)	<0.001
CRP, mg/L median (range)	1.59 (0.99-3.14)	1.48 (0.93-2.69)	2.08 (1.11-3.85)	<0.001
TNF-α, pg/mL, median (range)	3.16 (2.45-4.11)	3.29 (2.57-4.22)	2.99 (2.28-3.87)	<0.001
Smoking, %				
Never	43.7	43.0	44.8	<0.001
Current	10.3	6.2	16.1	
Former	45.9	50.7	39.1	
Smoking pack-years, mean (SD)	19.3 (27.9)	21.1 (30.2)	16.7 (24.1)	<0.001
Drinking, %				
None	50.1	41.5	62.7	<0.001
<1/wk	20.7	20.9	20.7	
1-7/wk	21.4	28.3	11.8	
>1/day	7.4	9.2	4.9	
BMI, kg/m ² , mean (SD)	27.4 (2.9)	26.5 (2.9)	28.6 (5.4)	<0.001
Abdomen visceral fat cm ² , mean (SD)	142.8 (67.6)	151.9 (69.8)	130.0 (62.2)	<0.001
Physical activity kcal/kg/week, mean (SD)	83.1 (69.4)	84.9 (64.4)	80.6 (75.9)	0.10
Diseases, %				
Lung disease	18.6	19.8	17.0	0.05
Heart disease	17.3	17.7	16.9	0.55
Cerebrovascular disease	7.2	6.5	8.5	0.03
Diabetes mellitus	18.9	14.1	25.7	<0.001
Osteoporosis	14.2	17.2	10.3	<0.001
Cancer	18.9	24.5	11.2	<0.001
Depressed mood	4.8	4.9	4.7	0.75
Cognitive impaired	7.7	2.0	15.8	<0.001
Medication use, %				
Anti-inflammatory drugs	53.2	56.7	48.7	<0.001
Corticosteroids	2.3	2.8	1.5	0.02

* Based on chi-square test for categorical variables and t-test or nonparametric Mann-Whitney statistics for continuous variables

test was used; median values of inflammatory markers with interquartile ranges were reported.

Linear regression analysis was used to test the association between SES and serum levels of inflammatory markers. Log-transformed values of inflammatory markers were used as dependent variables in the regression models. Three models were fitted, the first model adjusted for sociodemographics. The second adjusted for sociodemographics and behavioral factors and the model 3 adjusted for sociodemographics and diseases, anti-inflammatory drugs and corticosteroids.

RESULTS

Characteristics of the study population are shown in table 1. The mean age of the study sample was 74.2, 51.4% were women, and 41.5% were black. Of the 3,044 participants, 25.1% had less than 12 years of education, 11.6% had an income less than \$10,000 and 16.5% had no assets. Because the Health ABC study includes one of the largest samples of older African-Americans, the main characteristics are also shown for blacks and whites separately. Black participants were less well educated, had lower income and had fewer assets compared with white participants. Levels of IL-6 and CRP were higher in blacks than in whites and TNF- α levels were higher in whites than in blacks. The association between SES and inflammatory markers, however, was not different between the two groups (data not shown). This was confirmed by the non-significant interactions between each indicator of SES and race; therefore, subsequent results are shown for both together. Interaction terms between SES and sex were also not statistically significant (all $p > 0.05$; data not shown). Table 2 presents median values of IL-6, CRP, and TNF- α in each SES group. People with low SES had significantly higher serum levels of IL-6 and CRP than people in high SES groups ($p < 0.001$). Median values of TNF- α were highest in the middle SES groups of education and income and results were not statistically significant for the number of assets.

Regression coefficients for the association of each indicator of SES with serum levels of IL-6, CRP, and TNF- α are shown in tables 3, 4, and 5 respectively. Table 3 shows that low SES was associated with significantly higher levels of IL-6 compared with high SES groups in the model that adjusted for age, sex, race, study site, and marital status (Model 1). When behavioral variables were added to the first model (Model 2), regression coefficients decreased from 42-66% (without taking into account the missing category for income) and most results did not remain statistically significant.

Table 2. Median (interquartile range) of IL-6, CRP, and TNF- α by education, income, and number of assets

	IL-6 (pg/mL)			CRP (mg/L)			TNF- α (pg/mL)		
	n=3,044 %	median (interquartile range)	p-value *	median (interquartile range)	p-value *	median (interquartile range)	p-value *	median (interquartile range)	p-value *
Overall		1.83 1.27 2.79		1.59 0.99 3.14		3.16 2.45 4.11			
Education									
>12 years	42.0	1.71 1.19 2.66	<0.001	1.49 0.95 2.70	<0.001	3.11 2.40 4.04	0.01		
12 years	32.7	1.90 1.27 2.83		1.68 1.02 3.25		3.28 2.55 4.23			
<12 years	25.1	2.01 1.34 3.04		1.85 1.07 3.68		3.11 2.37 4.07			
Income (\$)									
$\geq 50,000$	14.1	1.70 1.11 2.59	<0.001	1.47 0.91 2.70	<0.001	3.12 2.43 3.97	0.01		
25,000-<50,000	27.9	1.77 1.24 2.71		1.50 0.96 2.63		3.25 2.54 4.22			
10,000-<25,000	34.2	1.93 1.33 2.87		1.77 1.04 3.35		3.20 2.47 4.10			
<10,000	11.6	2.01 1.35 3.24		2.23 1.17 3.87		3.05 2.35 4.04			
Missing	12.2	1.76 1.19 2.80		1.69 0.99 3.07		2.97 2.26 3.92			
Assets									
3-7	42.9	1.72 1.18 2.59	<0.001	1.47 0.94 2.69	<0.001	3.20 2.53 4.09	0.12		
1-2	39.7	1.96 1.31 2.93		1.79 1.04 3.37		3.16 2.41 4.12			
None	16.5	1.97 1.31 2.99		1.95 1.10 3.69		3.06 2.33 4.05			

* Based on nonparametric Kruskal-Wallis test

Table 3. Regression coefficients (with SE) of interleukin-6 (log transformed)* by education, income, and number of assets

	Interleukin-6							
	Model 1 †		Model 2 ‡			Model 3 §		
	Regression coefficient (SE)	p-value	Regression coefficient (SE)	p-value	% red	Regression coefficient (SE)	p-value	% red
Education								
>12 years	Ref.							
12 years	0.065 (0.029)	0.02	0.028 (0.028)	0.32	57	0.053 (0.028)	0.06	18
<12 years	0.075 (0.034)	0.03	0.033 (0.033)	0.31	56	0.051 (0.034)	0.13	32
Income (\$)								
≥50,000	Ref.							
25,000-<50,000	0.078 (0.040)	0.05	0.034 (0.038)	0.38	56	0.053 (0.039)	0.17	32
10,000-<25,000	0.116 (0.041)	0.01	0.060 (0.040)	0.13	48	0.084 (0.040)	0.04	28
<10,000	0.177 (0.055)	0.001	0.103 (0.054)	0.06	42	0.136 (0.055)	0.01	23
Missing	0.060 (0.049)	0.22	0.017 (0.047)	0.72	72	0.044 (0.048)	0.36	27
Assets								
3-7	Ref.							
1-2	0.065 (0.030)	0.03	0.036 (0.029)	0.21	45	0.053 (0.029)	0.07	18
None	0.087 (0.040)	0.03	0.030 (0.038)	0.44	66	0.068 (0.039)	0.08	22

* Log transformed values of interleukin-6 ranged from -1.58 to 2.77

† Model 1: adjusted for age, sex, race, site, marital status, and other household members for income

‡ Model 2: adjusted for age, sex, race, site, marital status, other household members for income, smoking, drinking, BMI, abdominal visceral fat, and physical activity

§ Model 3: adjusted for age, sex, race, site, marital status, other household members for income, lung disease, heart disease, cerebrovascular disease, diabetes, osteoarthritis, cancer, depressed mood, cognitive impairment, anti-inflammatory drugs, and corticosteroids

|| Percentage reduction in regression coefficients from model 1 computed by $(\text{regression coefficient}_{\text{model 1}} - \text{regression coefficient}_{\text{model 2/3}}) / (\text{regression coefficient}_{\text{model 1}}) \times 100\%$

When prevalent diseases were added to the model with demographic variables only the regression coefficients decreased from 18-32%. There still was a statistically significant association between low income and elevated levels of IL-6 after adjustment. Low SES was also associated with significantly higher levels of CRP, but this was not statistically significant for assets (Table 4). The role of behavioral factors and diseases was similar to that seen for IL-6. Behavioral factors decreased the regression coefficients substantially (56% on average) while control for diseases decreased the regression coefficients much less (28% on average). There was a non-linear association of education and assets with TNF- α ; the highest levels of TNF- α were found in the middle SES groups compared with the highest SES groups (Table 5). The largest reduction in regression coefficients related to SES was also found in model 2 in which behavioral factors were added. In additional analyses it was determined which behavioral factors reduced the regression coefficient of SES the most (data not shown). BMI and visceral fat reduced the regression coefficients for all three inflammatory markers the most. Smoking and drinking were further able to reduce the regression coefficients; physical activity was least important.

Table 4. Regression coefficients (with SE) of C-reactive protein (log transformed)* by education, income, and number of assets

	C-reactive protein							
	Model 1 †		Model 2 ‡			Model 3 §		
	Regression coefficient (SE)	p-value	Regression coefficient (SE)	p-value	% red	Regression coefficient (SE)	p-value	% red
Education								
>12 years	Ref.							
12 years	0.055 (0.037)	0.13	0.019 (0.036)	0.59	65	0.048 (0.036)	0.19	13
<12 years	0.115 (0.043)	0.01	0.075 (0.042)	0.08	35	0.091 (0.044)	0.04	21
Income (\$)								
≥50,000	Ref.							
25,000-<50,000	0.043 (0.051)	0.40	0.014 (0.049)	0.78	67	0.016 (0.050)	0.76	63
10,000-<25,000	0.109 (0.053)	0.04	0.066 (0.052)	0.20	39	0.076 (0.052)	0.14	30
<10,000	0.144 (0.071)	0.04	0.078 (0.069)	0.26	46	0.104 (0.070)	0.14	28
Missing	0.020 (0.062)	0.74	-0.021 (0.061)	0.73	>100	0.005 (0.062)	0.94	75
Assets								
3-7	Ref.							
1-2	0.064 (0.038)	0.09	0.035 (0.037)	0.34	45	0.051 (0.037)	0.17	20
None	0.076 (0.051)	0.13	0.003 (0.049)	0.96	96	0.061 (0.050)	0.23	20

* Log transformed values of C-reactive protein ranged from -1.90 to 4.44

† Model 1: adjusted for age, sex, race, site, marital status, and other household members for income

‡ Model 2: adjusted for age, sex, race, site, marital status, other household members for income, smoking, drinking, BMI, abdominal visceral fat, and physical activity

§ Model 3: adjusted for age, sex, race, site, marital status, other household members for income, lung disease, heart disease, cerebrovascular disease, diabetes, osteoarthritis, cancer, depressed mood, cognitive impairment, anti-inflammatory drugs, and corticosteroids

|| Percentage reduction in regression coefficients from model 1 computed by $(\text{regression coefficient}_{\text{model 1}} - \text{regression coefficient}_{\text{model 2/3}}) / (\text{regression coefficient}_{\text{model 1}}) * 100\%$

We further excluded 1,437 persons with diseases that were strongly associated with elevated levels of inflammatory markers (heart disease, lung disease, cerebrovascular disease, and diabetes mellitus). The association between SES and all three inflammatory markers remained similar and significant (data not shown). In addition, interaction terms between SES and those diseases were not statistically significant, which confirmed that the association between SES and inflammatory markers was similar for people with and without prevalent disease.

Table 5. Regression coefficients (with SE) of tumor necrosis factor- α (log transformed)* by education, income, and number of assets

	Tumor necrosis factor- α							
	Model 1 †		Model 2 ‡			Model 3 §		
	Regression coefficient (SE)	p-value	Regression coefficient (SE)	p-value	% red	Regression coefficient (SE)	p-value	% red
Education								
>12 years	Ref.							
12 years	0.070 (0.019)	<0.001	0.052 (0.019)	0.01	26	0.064 (0.018)	0.001	9
<12 years	0.046 (0.022)	0.04	0.025 (0.022)	0.25	46	0.033 (0.022)	0.13	28
Income (\$)								
≥50,000	Ref.							
25,000-<50,000	0.057 (0.026)	0.03	0.039 (0.025)	0.12	32	0.052 (0.025)	0.04	9
10,000-<25,000	0.076 (0.027)	0.004	0.053 (0.027)	0.05	30	0.066 (0.026)	0.01	13
<10,000	0.093 (0.036)	0.01	0.068 (0.036)	0.06	27	0.073 (0.036)	0.04	22
Missing	0.001 (0.031)	0.99	-0.018 (0.031)	0.55	>100	0.004 (0.031)	0.90	>100
Assets								
3-7	Ref.							
1-2	0.051 (0.019)	0.01	0.040 (0.019)	0.04	22	0.046 (0.019)	0.02	10
None	0.034 (0.026)	0.19	0.013 (0.025)	0.62	62	0.016 (0.025)	0.53	53

* Log transformed values of tumor necrosis factor- α ranged from -0.57 to 3.39

† Model 1: adjusted for age, sex, race, site, marital status, and other household members for income

‡ Model 2: adjusted for age, sex, race, site, marital status, other household members for income, smoking, drinking, BMI, abdominal visceral fat, and physical activity

§ Model 3: adjusted for age, sex, race, site, marital status, other household members for income, lung disease, heart disease, cerebrovascular disease, diabetes, osteoarthritis, cancer, depressed mood, cognitive impairment, anti-inflammatory drugs, and corticosteroids

|| Percentage reduction in regression coefficients from model 1 computed by $(\text{regression coefficient}_{\text{model 1}} - \text{regression coefficient}_{\text{model 2/3}}) / (\text{regression coefficient}_{\text{model 1}}) \times 100\%$

DISCUSSION

In well-functioning older adults, low SES was associated with elevated levels of inflammatory markers, compared with high SES. The most consistent linear associations were found for IL-6 and CRP. Results were less clear and not always linear for TNF- α . A substantial portion of the inverse association between SES and inflammatory markers was explained by behavioral factors. Adjustment for prevalent diseases associated with inflammation explained less of the association. Both behavioral factors and diseases failed to explain the association between SES and inflammatory markers completely.

The prevalence of many diseases including heart disease, diabetes mellitus, cognitive impairment and depression is higher in low SES groups (28-32). However, the results suggest that the inverse association between SES and inflammatory markers is largely independent of prevalent disease despite these diseases being related to increased levels of inflammatory markers (6, 17, 18). On the other hand, behavioral factors explained a substantial part of the SES differences in inflammatory marker levels. An unhealthy lifestyle, including smoking, drinking and obesity, is more prevalent in low SES groups (19) and also related to higher levels of inflammatory

markers (20, 21). It is not surprising that BMI and abdominal visceral fat, both of which are inversely related to SES, are important in explaining SES differences in inflammatory marker levels because IL-6 and TNF- α are produced within adipose tissue (33). There is an indirect effect with CRP. Since IL-6 is a primary stimulus of CRP production in the liver, higher levels of IL-6 could result in higher levels of CRP (34).

This study has some limitations. First, this study was cross-sectional. Consequently it is impossible to draw conclusions regarding the causal pathways. It is, however, unlikely that high levels of inflammatory markers could have lowered SES, especially education level, as the latter indicator is less sensitive to change during adulthood. In this study similar results were found across all SES indicators, including education. Second, only limited information on severity of diseases was available. We cannot exclude the possibility that a better measurement of the disease burden would have resulted in a stronger contribution of diseases to the association between SES and inflammatory markers. In particular, sub-clinical cardiovascular disease is extremely common in older adults and is associated with high IL-6 levels (35). Finally, there may be other factors which could also be important in explaining SES differences in inflammatory marker levels. These factors may include psychological stress that are related to both low SES and increased levels of inflammatory markers (perhaps through adverse behavioral factors) (3, 36). This study also has several strengths. To our knowledge this is the first study that examined the association between SES and levels of inflammatory markers in a large sample of black and white older adults. Furthermore, a large number of potential confounders and mediators were considered in the association between SES and inflammatory markers.

In conclusion, low SES was associated with elevated levels of inflammatory markers. Rather than by a higher prevalence of diseases in low SES groups, the elevated levels of inflammatory markers in low SES groups were to a larger extent based upon a higher prevalence of unhealthy behaviors (especially obesity, smoking, and drinking) in these groups; visceral fat is, however, less amenable to modification. This suggests that even in old age, interventions to improve health behaviors, especially in low SES groups, may be useful in reducing risks associated with inflammation.

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6

Is there a biomedical explanation for socioeconomic differences in incident mobility limitation?

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ABSTRACT

Objective. The association between low socioeconomic status and poor physical functioning has been well described; biomedical factors may play an important role in explaining these differences. This study examines the association between socioeconomic status and incident mobility limitation in well-functioning older adults, and seeks to determine whether this link could be explained by biomedical factors.

Methods. Data were obtained from 3,066 men and women, aged 70-79 from Pittsburgh, Pennsylvania and Memphis, Tennessee participating in the Health, Aging and Body Composition (Health ABC) study. Three indicators of socioeconomic status were used: education, income, and ownership of financial assets. Mobility limitation was defined as reporting difficulty walking 1/4 mile or climbing 10 steps during two consecutive semiannual assessments over 4.5 years. Biomedical factors included a wide range of diseases (e.g., heart and cerebrovascular disease) and biological risk factors (e.g. hypertension, poor pulmonary function, and high serum levels of inflammatory markers).

Results. Adjusted hazard ratios of incident mobility limitation were significantly higher in those persons with low education, low income, and few assets. Hazard ratios ranged from 1.66 to 2.80 in the lowest socioeconomic groups. Additional adjustment for biomedical factors reduced the hazard ratios by an average of 41% for education, 17% for income, and 29% for assets.

Conclusions. Biomedical factors can account for some of the association between socioeconomic status and incident mobility limitation. However, to reduce physical disabilities and, in particular, the socioeconomic differences therein, it may not be sufficient to solely intervene upon biological risk factors and risks of diseases.

INTRODUCTION

The association between low socioeconomic status (SES) and poor physical functioning has been well described (1-4), and several authors hypothesize that biomedical factors such as diseases and biological risk factors play an important role in explaining these differences (5-7). Low SES is a risk factor for cardiovascular disease and stroke, as well as a variety of other diseases (8-10). Obesity, high blood pressure, glucose intolerance, and reduced lung function are also more prevalent among low SES groups in comparison with high SES groups (11-14). Several of these biomedical factors have been directly related to adverse functional outcomes (4, 15, 16). As important as disease and established disease risk factors may be in predicting function, data are emerging to suggest that certain inflammatory markers also play an important role, independent of prevalent clinical disease (17-20). Two recent studies (21, 22) also show that persons with low SES have higher serum levels of inflammatory markers than persons do with high SES.

Despite the knowledge that low SES is related to both poorer function and increased prevalence of many diseases and their risk factors, the extent to which biomedical factors can explain the relationship between low SES and functional decline has not been studied extensively. Knowledge of possible biological pathways underlying the association between SES and poor functional outcomes may give insight into potential ways of reducing SES differentials in physical functioning.

In the present study, we examine the association between SES and incident persistent mobility limitation in a large community-based cohort of well-functioning older adults and seek to determine whether this link can be explained by biomedical factors.

METHODS

Study population

The Health, Aging and Body Composition (Health ABC) study is a longitudinal cohort study consisting of 3,075 well-functioning, 70- to 79-year-old, black and white men and women. Participants were identified from a random sample of white Medicare beneficiaries and all age-eligible community-dwelling black residents in designated ZIP code areas surrounding Memphis, Tennessee, and Pittsburgh, Pennsylvania. Participants were eligible if they reported themselves to be well-functioning and free of difficulty with any activity of daily living. 'Well-functioning' was defined as having no difficulty in either walking 1/4 mile or going up 10 steps without

resting. Participants were excluded if they reported a history of active treatment for cancer in the prior 3 years, planned to move out of the study area within the next 3 years, or were currently participating in a randomized trial of a lifestyle intervention. Baseline data, collected between April 1997 and June 1998, included an in-person interview and a clinic-based examination, with evaluation of body composition, clinical and subclinical diseases, and physical functioning. Four and a half years of follow-up were used in this study. For nine participants, outcome data or SES data were missing, leaving 3,066 participants for the present analyses. All participants signed informed written consent approved by the institutional review boards of the clinical sites.

Measures

Socioeconomic status. Three indicators of SES were used: education, family income, and ownership of financial assets. Categories for education were: less than 12 years, 12 years, and more than 12 years. Family income was measured by adding all reported income components of all household members. Income was defined as any source of income including, wages, salaries, social security or retirement benefits, help from relatives, and rent from property. Four categories of family income were distinguished: less than \$10,000, between \$10,000 and less than \$25,000, between \$25,000 and less than \$50,000, and greater than or equal to \$50,000. Because of the large number of participants with missing income data a fifth category was created for this group. Financial assets included: money market account, saving bonds or treasury bills, home ownership or investment property or housing, a business or farm, stock or stock mutual funds, and individual retirement account (IRA) or KEOGH account, and other investments. Three categories were created: none, one or two, and three to seven (23). Rather than use a dichotomous variable of any versus no assets, categories were used because the literature has shown that various SES gradients have significant effect on health outcomes (24).

Incident/persistent mobility limitation. The occurrence of mobility limitation over four and a half years of follow-up was determined every 6 months, at study assessment visits (12, 24, 36, and 48 months after baseline), or during telephone follow-up assessments (6, 18, 30, 42, and 54 months after baseline). Incident/persistent mobility limitation was considered to be present when a person reported any difficulty walking one quarter mile or climbing 10 steps at two semiannual follow-up assessments. The requirement that mobility limitation needed to be present at two consecutive assessments selected more participants with chronic and severe functional limitation; therefore, this outcome was thought to be a more reliable indicator of a clinically relevant change in functional status. The incidence of mobility

limitation was adjudicated by a special Health ABC study committee that considered additionally available information such as reason for missing study contact (in nursing home, severe illness) and proxy information for those participants who died.

Biomedical factors. Possible biomedical explanatory variables cover a wide range of clinical and subclinical diseases. Baseline presence of diseases was determined using standardized algorithms considering self-report, use of specific medications, and some clinical assessments. Baseline prevalent cancer was based on self-reports of cancer or malignancy and/or use of anticancer medication. The presence of heart disease was defined as having coronary heart disease and/or congestive heart failure. Cerebrovascular disease was defined as a history of stroke or transient ischemic attack. Peripheral arterial disease was based on self-reports of intermittent claudication or a history of lower extremity bypass or angioplasty. Three categories, using American Diabetes Association criteria, were distinguished for glucose intolerance: not impaired (fasting glucose level <100 mg/dl), impaired fasting glucose (fasting glucose level between 100 mg/dl and 126 mg/dl), and diabetes mellitus (fasting glucose level >126 mg/dl). Hypercholesterolemic persons had high total cholesterol levels (≥ 240 mg/dl) and/or high low-density lipoprotein cholesterol levels (≥ 130 mg/dl) or were being treated with lipid-lowering drugs (25). Triglyceride levels were considered high if >150 mg/dl, and high-density lipoprotein cholesterol levels were considered low if <40 mg/dl in men and <50 mg/dl in women (26). For hypertension, four categories were created: normotensive (systolic pressure <140 mmHg and diastolic pressure <90 mmHg); normotensive and treated with blood pressure lowering drugs, hypertension stage 1 (systolic pressure ≥ 140 mmHg or diastolic pressure ≥ 90 mmHg), and hypertension stage 2 (systolic pressure ≥ 160 mmHg or diastolic pressure ≥ 100 mmHg) (27). Two variables for pulmonary function were used: a poor forced expiratory volume in 1 second/forced vital capacity ratio (FEV_1/FVC) of 70% or lower, and percentage predicted FEV_1 of 80% or lower (28, 29). Body mass index (BMI) was categorized as lower than 25 kg/m^2 , between 25 and 30 kg/m^2 , and higher than 30 kg/m^2 . Knee pain was considered if reported to be present at least 1 month of the past year. Ankle arm blood pressure index was considered indicative of peripheral arterial disease if lower than 0.9 (30). Finally, serum level of three inflammatory markers were assessed; interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor- α (TNF- α) (20). Each biomarker variable was dichotomized, where inflammation was defined as being in the highest tertile of IL-6 (≥ 2.40 pg/ml), CRP (≥ 2.55 mg/L), and TNF- α (≥ 3.72 pg/ml). In addition, an inflammation index was calculated as the number of inflammatory markers in the highest tertile, which has been shown to be an important predictor of incident mobility limitation (20).

Sociodemographics. Sociodemographics included age, sex, race (black, white), study site (Memphis, Pittsburgh), and marital status (never married, previously married, married). For the analyses with family income we also controlled for whether (yes/no) people had household members other than their spouse.

Statistical analyses

Analyses were performed using SPSS, version 11.5 (SPSS Inc., Chicago, IL). To determine if there were differences in the incidence of mobility limitation between SES groups, unadjusted Cox proportional hazard regression models were fitted. To determine if there were differences in prevalences of the biomedical variables between SES groups, the chi-square test was used. To evaluate the association of biomedical factors with time to incident persistent mobility limitation, Cox proportional hazard regression models were fitted, adjusting for sociodemographics. Cox proportional hazard regression models were also fitted to study the association of SES on time to incident mobility limitation. Persons surviving with no evidence of incident limitation were censored at the last study visit, those dying with no evidence of incident limitation were censored at time of death, and those lost to follow-up were censored at their last interview. Two models were fitted; the first model was adjusted for sociodemographics. The second model contained all variables of the first model as well as the biomedical variables. To determine the role of biomedical factors, a percentage reduction in hazard ratio (HR) from model 1 was computed: $(HR_{\text{model 1}} - HR_{\text{model 2}}) / (HR_{\text{model 1}} - 1) * 100\%$.

RESULTS

Figure 1 shows the incidence rate per 1,000 person years of incident mobility limitation according to SES. A p-value for trend was statistically significant ($p < 0.001$) for all three SES indicators. More people with low SES had incident mobility limitation than did those with high SES. Results are shown for black and white persons together because interaction terms between any of the SES indicators and race were not statistically significant (all $p > 0.10$).

In further analyses we used only those biomedical variables that were statistically significant predictors of incident persistent mobility limitation; because of their nonsignificance baseline presence of cancer and hypercholesterolemia were not further considered. All other biomedical factors were significant predictors of incident mobility limitation, adjusted for sociodemographics (not tabulated).

Incidence rate per 1,000 person years of mobility limitation
according to education

Incidence rate per 1,000 person years of mobility limitation
according to income

Incidence rate per 1,000 person years of mobility limitation
according to number of assets

Figure 1. Incidence rate per 1,000 person years of mobility limitation according to socioeconomic status

* p-value based on unadjusted survival analyses

Especially strong predictors were having three inflammatory markers in the highest tertile (HR: 3.21, 95% confidence interval [CI]: 2.65, 3.89), peripheral arterial disease (HR: 2.42, 95% CI: 1.97, 2.96), and knee pain (HR: 2.20, 95% CI: 1.93, 2.50).

Generally, the prevalence of disease and biological risk factors was higher in low SES groups except for high triglycerides levels and low high-density lipoprotein cholesterol levels for which the prevalence was higher in high SES groups. Glucose intolerance, hypertension, low predicted FEV₁, high BMI, low ankle arm blood pressure index, high serum levels of IL-6 and CRP, and having three inflammatory markers in the highest tertile were statistically significantly associated with low education, low income and few assets (Table 1).

Hazard ratios of incident mobility limitation, adjusted for sociodemographics, were significantly higher in low SES groups compared to the highest SES group (Table 2, Model 1). Low income showed a particularly increased risk: hazard ratios were 2.80 (95% CI: 2.06, 3.81) in the income group <\$10,000, and 1.82 (95% CI: 1.40, 2.36) in the income group \$10,000-\$25,000 compared to that of those participants with an income higher than \$50,000. Strong associations were found between SES and incident mobility limitation for all three SES indicators. When all SES indicators were included in one analytic model, all three indicators remained significant predictors of incident mobility limitation. Therefore, education, income, and number of assets all had an independent effect on incident mobility limitation.

Biomedical factors (Model 2) could explain a substantial part of the SES gradient in incident mobility limitation. The largest reduction in hazard ratios was found for education; 48% for the group with 12 years of education and 33% for the group with less than 12 years of education (41% on average). Biomedical factors decreased the hazard ratios by an average of 17% for income and 29% for the number of assets. Figure 2 shows the biomedical factors that reduced the hazard ratios for the lowest SES group versus the highest SES group by more than 5%, adjusted for sociodemographics. High serum levels of inflammatory markers and high BMI decreased the hazard ratios by more than 5% for all three SES indicators.

Table 1. Distribution (%) of biomedical factors by education, income and number of assets

Biomedical factors (%)	Education			Income			Number of assets						
	<12 years	12 years	>12 years	p-value	<\$10,000	\$10,000- <\$25,000	\$25,000- ≥\$50,000	p-value	None	1-2	3-7	p-value	
Heart disease	18.3	17.0	17.2	0.75	16.6	17.8	17.5	18.5	0.92	18.0	17.0	17.2	0.87
Cerebrovascular disease	7.7	6.7	7.6	0.63	8.7	8.7	6.6	5.6	0.11	9.0	7.4	6.7	0.24
Glucose intolerance													
Not impaired	59.9	65.0	67.4	<0.001	59.0	63.0	65.1	68.8	0.02	62.1	62.6	67.6	<0.001
Impaired fasting glucose	22.4	21.6	21.0		21.6	23.1	21.9	19.3		18.6	21.9	22.4	
Diabetic	17.8	13.4	11.5		19.4	13.9	13.0	11.9		19.4	15.6	10.0	
Triglycerides >150 mg/dl	23.9	34.3	31.6	<0.001	24.8	30.4	33.8	32.8	0.01	25.2	28.0	35.0	<0.001
HDL cholesterol <40mg/dl in men, <50 mg/dl in women	26.7	31.0	29.7	0.13	24.9	28.9	32.7	27.7	0.07	28.7	27.4	31.6	0.06
Hypertension													
Normotensive	28.1	28.0	34.9	0.001	24.2	26.4	34.4	39.0	<0.001	25.6	28.3	35.4	<0.001
Normotensive with medications	29.2	31.7	29.8		32.2	32.0	28.7	29.3		33.5	29.9	29.5	
Stage 1	27.9	26.6	24.5		26.4	27.8	24.7	23.1		26.0	28.5	23.8	
Stage 2	14.8	13.7	10.8		17.1	13.8	12.3	8.5		15.0	13.2	11.3	
FEV ₁ /FVC<70%	24.0	21.2	20.4	0.18	24.3	22.7	21.5	18.3	0.19	20.1	23.4	20.3	0.14
Predicted FEV1 <80%	28.7	22.3	22.7	0.004	28.6	24.5	24.6	19.5	0.04	23.8	26.8	21.6	0.01
BMI													
<25 kg/m ²	28.0	29.6	36.9	<0.001	29.8	30.1	34.5	35.8	<0.001	27.0	31.1	35.3	<0.001
25-30 kg/m ²	40.0	42.6	43.5		35.3	41.2	44.9	46.4		36.0	41.2	45.8	
>30 kg/m ²	32.0	27.8	19.6		35.0	28.7	20.6	17.8		37.0	27.7	19.0	
Peripheral arterial disease	6.4	4.7	5.1	0.30	6.1	5.4	5.2	4.0	0.60	6.6	5.5	4.7	0.25
Ankle arm index <0.9	19.4	16.4	10.9	<0.001	20.7	16.2	11.9	7.5	<0.001	20.6	17.5	9.0	<0.001
Knee pain	18.7	17.5	14.2	0.01	20.4	17.9	14.6	14.5	0.03	16.9	17.0	15.6	0.60
Highest tertile IL-6	37.5	34.5	29.5	<0.001	40.5	34.7	32.0	26.5	<0.001	38.7	36.4	27.9	<0.001
Highest tertile CRP	38.7	34.8	28.6	<0.001	43.8	36.6	27.1	28.2	<0.001	39.4	36.7	27.6	<0.001
Highest tertile TNF-α	33.5	35.6	31.4	0.12	31.8	35.3	34.2	31.3	0.42	30.8	34.5	33.2	0.35
Inflammation index *													
No high	36.6	34.9	43.9	<0.001	33.1	35.2	41.1	45.9	<0.001	36.1	34.7	44.3	<0.001
1 of 3 high	30.2	36.7	31.6		31.6	35.3	33.5	31.2		30.1	35.0	32.2	
2 of 3 high	24.1	20.3	18.7		26.3	21.2	19.4	17.2		26.3	21.8	17.2	
All 3 high	9.2	8.0	5.8		9.0	8.4	6.0	5.6		7.6	8.5	6.4	

* The number of inflammatory marker levels (IL-6, CRP, TNF-α) within the highest tertile

Table 2. Hazard ratios (HR) and 95% confidence interval (CI) of incident mobility limitation by education, income, and number of assets

Socioeconomic status	Model 1 *		Model 2 †		
	HR	95% CI	HR	95% CI	% red. ‡
Education					
>12 years	1.00		1.00		
12 years	1.33	1.13, 1.56	1.17	1.00, 1.38	48
<12 years	1.70	1.42, 2.04	1.47	1.23, 1.77	33
Income					
≥\$50,000	1.00		1.00		
\$25,000-<\$50,000	1.39	1.07, 1.80	1.33	1.02, 1.73	15
\$10,000-<\$25,000	1.82	1.40, 2.36	1.61	1.24, 2.09	26
<\$10,000	2.80	2.06, 3.81	2.64	1.93, 3.61	9
Missing	1.55	1.15, 2.09	1.45	1.07, 1.96	18
Number of assets					
3-7	1.00		1.00		
1-2	1.28	1.08, 1.51	1.20	1.02, 1.42	29
None	1.66	1.35, 2.04	1.47	1.19, 1.81	29

* Model 1: adjusted for age, sex, race, study site, other household members for income, and marital status

† Model 2: adjusted for age, sex, race, study site, other household members for income, marital status, and biomedical factors

‡ Percentage reduction in hazard ratio from model 1 computed by $(HR_{\text{model 1}} - HR_{\text{model 2}}) / (HR_{\text{model 1}} - 1) * 100\%$

DISCUSSION

In a well-functioning older population, low SES predicted an increased incidence of mobility limitation during four and a half years of follow-up. Biomedical factors explained a substantial part of the SES differentials in mobility limitation incidence. On average 41% of the education differences in mobility limitation incidence was explained by biomedical factors; this was 17% for income and 29% for the number of assets. However, even after taking into account a wide range of diseases and biological risk factors, persons in the lowest SES group still had a significant increased risk of mobility limitation incidence. Hazard ratios still ranged from 1.47 to 2.64 in the lowest SES groups compared to the highest SES groups. Hazard ratios were more modest in the middle SES groups compared to the highest SES groups. High serum levels of inflammatory markers, high BMI, and hypertension were found to be the most important biomedical variables in reducing the hazard ratios for SES. It is notable that these variables measured biological characteristics rather than diseases. These biological risk factors have in common that they are risk

Figure 2. Biomedical factors that reduced the hazard ratio for the lowest versus the highest socioeconomic group by more than 5%, adjusted for age, sex, race, study site, and marital status

factors for a wide variety of health outcomes and may represent indicators of disease severity.

Relatively few studies in older people have investigated the relationship between SES and functional outcomes (31-33). Studies have found that the relationship between SES and health outcomes were weaker in people aged 65 or older compared to younger people. In addition, it has been argued that disparities in income diminish after retirement. In our study, the effect of SES on poor functional outcomes in elderly persons was still quite strong.

Besides biomedical factors there may also be other factors that explain the association between SES and mobility limitation. These factors may be related to behavioral or psychosocial factors. Low SES is related to many adverse behavioral factors, such as smoking status and excessive alcohol consumption that in turn are related to poor health outcomes (3, 6, 34). In this study, the contribution of biomedical factors in explaining SES difference in incident mobility limitation is similar when we adjust for smoking status (current, former, never smoker), alcohol use during the past year (0, <1, 1-7, >7 drinks per week), and physical activity (total kilocalories per kilogram per week) in both models (data not shown). There is also evidence that psychosocial factors, such as control beliefs and stress, play an important role in functional outcomes (35), and also in the association between SES and poor functional outcomes (36).

This study has some limitations. First, information on mobility limitation was based on self-report. Second, only limited information was available on the severity of baseline diseases. It must be acknowledged that the approach followed is limited by both the comprehensiveness of the panel of biomedical factors considered and the reliability with which these factors have been assessed. We cannot exclude the possibility that, had the underlying biomedical factors been measured more exactly, the diminution in the strength of the SES effect may have been larger. Third, only baseline data on biomedical factors were analyzed, so the association between SES on the one hand and biomedical factors on the other hand might have resulted from reverse causation; therefore, biomedical factors could have partly confounded the link between SES and mobility limitation (37-39). If present, it is likely that this selection effect would have had a larger effect on income and assets than on educational level, as the latter indicator is less sensitive to change during adulthood. However, in this study, the same results were found across all SES indicators (including education) so reversed causation is not very likely.

Our study also has several strengths. First, the study consists of a large cohort of older black and white adults in which loss-to follow-up for our outcome was very limited (<4%). Second, because of the longitudinal character of our study we

establish that SES precedes the development of mobility limitation. Third, we had information on a wide variety of diseases and biological risk factors, that allowed for a more comprehensive assessment than has been possible previously. Fourth, all participants were well-functioning at baseline, so the study was designed to determine incident functional limitation. Mobility limitation is the most important outcome in the Health ABC study because of the need to identify risk factors at a stage of age-related decline that would be amenable to preventive interventions.

In summary, there is a strong association between low SES and incident mobility limitation in older persons. Part of this association could be explained by biomedical factors. This study suggests that biomedical factors are important in the mechanism underlying the association between SES and poor functional outcomes. However, to reduce physical disabilities and, in particular, the SES differences therein, it may not be sufficient to solely intervene on biological risk factors and risks of diseases.

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Socioeconomic differences in cognitive decline and the role of biomedical factors

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ABSTRACT

Objective. This study examines the association between socioeconomic status and cognitive decline in a community-based cohort of well-functioning older adults and seeks to determine whether this link could be explained by biomedical factors.

Methods. Data are from 2,574 men and women, aged 70-79 from Pittsburgh, PA and Memphis, TN participating in the Health, Aging and Body Composition study (Health ABC). Three indicators of socioeconomic status were used: education, income, and ownership of financial assets. Cognitive decline over four years was defined as a decrease of five points or more in the Modified Mini-Mental State Examination (3MS) score. Biomedical factors measured at baseline, included heart disease, cerebrovascular disease, diabetes, hypertension, poor pulmonary function, and high serum levels of inflammatory markers.

Results. Adjusted odds ratios were significantly higher in those with low education, low income, and few assets. Odds ratios ranged from 1.51 to 2.16 in the lowest socioeconomic groups. Additional adjustment for biomedical factors reduced the odds ratios of cognitive decline by an average of 2% for education, 5% for income, and 8% for the number of assets.

Conclusions. Low socioeconomic status predicts a decline in cognitive function in older adults and this relationship is not mediated by biomedical factors.

INTRODUCTION

Accumulating evidence shows that low socioeconomic status (SES) is related to poor mental and physical functioning (1-5). However, longitudinal research on its association with decline in cognitive functioning remains scarce. Several studies have examined the association between educational level and cognitive change (6-8). Only few researchers have examined the association of other SES indicators, like occupational level and income, with cognitive function (9-12).

Biomedical factors, including common diseases and their biological risk factors, are likely to be an important mechanism that may help to explain a potential association between low SES and cognitive decline. Low SES is a risk factor for cardiovascular disease and stroke, as well as a variety of other diseases (13-15). Obesity, high blood pressure, glucose intolerance, and reduced lung function are also more prevalent among low SES groups in comparison to high SES groups (16-19). Several of these biomedical factors have also been associated with poor cognitive function (20-23). Two recent studies show that persons with low SES have higher serum levels of inflammatory markers than persons with high SES (24, 25). It is plausible to assume that SES differentials in inflammatory markers may also be important, as there is accumulating evidence that these markers are related to cognitive decline (26, 27).

To our knowledge the role of biomedical factors in the relation between SES and cognitive decline has not been studied extensively before. Insight into possible biological pathways underlying the association between SES and poor cognitive function may provide a better understanding of potential ways of reducing SES differentials in cognitive decline. This may be especially important in an aging population, given that maintaining good cognitive function is an essential part of successful aging (28).

The present study examines the association between low SES and cognitive decline and potential mediation effects of biomedical factors in a large community-based cohort study of older adults.

METHODS

Study population

The Health, Aging and Body Composition (Health ABC) study is a longitudinal cohort study consisting of 3,075 well-functioning, 70 to 79-year old, black and white men and women. Participants were identified from a random sample of White

Medicare beneficiaries and all age-eligible community-dwelling black residents in designated zip code areas surrounding Memphis, Tennessee, and Pittsburgh, Pennsylvania. Participants were eligible if they reported themselves to be well-functioning and free of difficulty with any activity of daily living. 'Well-functioning' was defined as having no difficulty in either walking one quarter of a mile or going up 10 steps without resting. Participants were excluded if they reported a history of active treatment for cancer in the prior three years, planned to move out of the study area in the next three years, or were currently participating in a randomized trial of a lifestyle intervention. Baseline data included an in-person interview and a clinic-based examination, with evaluation of body composition, clinical and sub-clinical diseases, and physical functioning. These data were collected between April 1997 and June 1998. The present study utilized four years of follow-up. For our analyses, individuals with missing data for SES or cognition at baseline or follow-up and those who died during the follow-up were excluded. Data on all SES measures were missing for one participant, 14 had missing baseline cognitive data, 204 people had missing follow-up cognitive data, and 282 people died during the follow-up, leaving 2,574 participants for the current analyses. All participants signed informed written consent forms approved by the institutional review boards of the clinical sites.

Measures

Socioeconomic status. Three indicators of SES, measured at baseline, were used: education, family income, and ownership of financial assets (29). Categories for education were: less than 12 years, 12 years, and more than 12 years. Family income was defined as: wages, salaries, social security or retirement benefits, help from relatives, and rent from property. Five categories were distinguished: less than \$10,000, between \$10,000 and less than \$25,000, between \$25,000 and less than \$50,000, greater than or equal to \$50,000, and missing ($n=316$, 12.3%). The number of financial assets a person reported was used as a third SES measure. Assets included: money market account, savings bonds or treasury bills, home ownership or investment property or housing, a business or farm, stock or stock mutual funds, and individual retirement account (IRA) or KEOGH account, and other investments. Three categories were created: none, one or two, and three to seven (29). Rather than use a dichotomous variable of any versus no assets, categories were used because the literature has shown that various SES gradients have significant effect on health outcomes (30).

Cognitive function. The Modified Mini-Mental State Examination (3MS) was used as an indicator of general cognitive status. This test includes orientation to time and place, recall, simple language, and construction (31). The 3MS has a minimum

score of zero and maximum score of 100 (best) and may be more sensitive for mild cognitive change than the traditional Mini-Mental State Examination (31). A decline in cognitive function between baseline and the fourth year of follow up was defined as a decrease of five points or more in 3MS score, as previously used and recommended in other studies (26, 32, 33). For 292 (11.3%) participants, 3MS data in the fourth year of follow-up were based on a shortened 3MS used in a telephone interview. A decline in cognitive function was also considered to be present if a participant was using specific medications for Alzheimer's disease, was hospitalized because of dementia as either the primary or concurrent diagnosis on a discharge summary during the four year follow-up ($n=44$, 1.71%) (26).

Biomedical factors. Possible biomedical explanatory variables covered a wide range of clinical and sub-clinical diseases. Baseline presence of diseases was adjudicated using standardized algorithms considering self-report, use of specific medications, and clinical assessments. The presence of heart disease was defined as having coronary heart disease and/or congestive heart failure. Cerebrovascular disease was defined as a history of stroke or transient ischemic attack. Peripheral arterial disease was based on self-reports of intermittent claudication or a history of lower extremity bypass or angioplasty. Three categories, using American Diabetes Association criteria, were distinguished for glucose intolerance: not impaired (fasting glucose level <100 mg/dl), impaired fasting glucose (fasting glucose level between 100 mg/dl and 126 mg/dl), and diabetes mellitus (fasting glucose level >126 mg/dl). Hypercholesterolemic persons had high total cholesterol levels (≥ 240 mg/dl) and/or high LDL cholesterol levels (≥ 130 mg/dl) or were being treated with lipid-lowering drugs (34). Dyslipidemic persons had high triglycerides (>150 mg/dl) and/or low HDL cholesterol levels (<40 mg/dl in men and <50 mg/dl in women) (35). For hypertension, four categories were created: normotensive (systolic pressure <140 mmHg and diastolic pressure <90 mmHg), normotensive and treated with blood pressure lowering drugs, hypertension stage 1 (systolic pressure ≥ 140 mmHg or diastolic pressure ≥ 90 mmHg), and hypertension stage 2 (systolic pressure ≥ 160 mmHg or diastolic pressure ≥ 100 mmHg) (36). Two indicators of poor pulmonary function were used. A poor forced expiratory volume in one second/forced vital capacity ratio (FEV_1/FVC) of 70% or lower, and percentage predicted FEV_1 of 80% or lower based on predicted values derived by Hankinson et al. (37, 38). Body mass index (BMI) was categorized as lower than 25 kg/m^2 , between 25 and 30 kg/m^2 , and higher than 30 kg/m^2 . Ankle arm blood pressure index was considered indicative of peripheral arterial disease if lower than 0.9 (39). Finally, serum level of three inflammatory markers were assessed; interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor- α (TNF- α) (40). Each biomarker variable was

dichotomized, and inflammation was defined as being in the top tertile level of IL-6 (≥ 2.40 pg/ml), CRP (≥ 2.55 mg/l), and TNF- α (≥ 3.72 pg/ml). In addition, a summary inflammation index was calculated as the number of inflammatory markers in the highest tertile (41).

Sociodemographics. Sociodemographics included age, sex, race (black, white), study site (Memphis, Pittsburgh), and marital status (never married, previously married, married). For the analyses with family income we also controlled for whether the household included anyone in addition to the participant and his or her spouse.

Statistical analyses

Analyses were performed using SPSS, version 11.5. Mean 3MS scores at baseline were compared across SES groups using analyses of variance. A chi-square test was conducted to assess the distribution of cognitive decline by SES. To determine if there were differences in the prevalence of biomedical variables between SES groups and to evaluate the association between biomedical factors and cognitive decline, chi-square tests were used. Logistic regression models were fitted to study the association between SES and cognitive decline. Two models were fitted, the first model adjusted for sociodemographics, other household members for income, and baseline 3MS score. The second model contained all variables of the first model as well as biomedical variables. To determine the role of biomedical factors a percentage reduction in odds ratio (OR) from model 1 was computed: $(OR_{\text{model 1}} - OR_{\text{model 2}}) / (OR_{\text{model 1}} - 1) * 100\%$.

RESULTS

Table 1 shows baseline 3MS scores and the distribution of cognitive decline by SES. People with low education, low income or few assets had statistically significantly lower baseline 3MS scores than people in the high SES groups ($p < 0.001$). The percentage of people with a cognitive decline over four years was also significantly higher in low SES groups in comparison to higher SES groups ($p = 0.01$ to $p < 0.001$).

Generally, the prevalence of disease and biological risk factors were higher in low SES groups, except for dyslipidemia for which the prevalence was significantly higher in high SES groups (Table 2). Glucose intolerance, hypercholesterolemia, hypertension, high BMI, low ankle arm index, high serum levels of IL-6 and CRP, and having three inflammatory markers in the highest tertile were significantly associated with low education, low income, and few assets. The prevalence of heart disease,

cerebrovascular disease, low FEV₁/FVC, peripheral arterial disease, and high TNF- α levels were not significantly associated with any of the SES indicators.

The prevalence of cerebrovascular disease, glucose intolerance, low predicted FEV₁, low ankle arm index, high serum levels of IL-6, and having one or more inflammatory markers in the highest tertile were significantly associated with a four year decline in cognitive function (all $p < 0.03$) (Table 3).

Table 1. Baseline 3MS score (mean and standard deviation (SD)) and percentage of four year cognitive decline according to socioeconomic status

Socioeconomic status	n=2,574	3MS score at baseline		4 year decline	
	%	Mean (SD)	p-value	%	p-trend
Education					
>12 years	43.8	93.8 (5.1)	<0.001	16.7	<0.001
12 years	32.8	90.9 (6.3)		23.4	
<12 years	23.2	84.1 (10.0)		37.0	
Income					
≥\$50,000	15.0	94.2 (5.0)	<0.001	14.2	0.01
\$25,000-<\$50,000	28.7	92.7 (5.8)		21.0	
\$10,000-<\$25,000	33.4	89.1 (8.2)		25.1	
<\$10,000	10.6	85.4 (10.3)		35.9	
Missing	12.3	90.1 (8.3)		27.2	
Assets					
3-7	45.5	93.2 (5.7)	<0.001	17.1	<0.001
1-2	38.5	88.7 (8.4)		28.3	
None	15.1	87.6 (9.6)		31.4	

Further analyses were done using only those biomedical variables that were significantly associated with both a four year cognitive decline and SES. Glucose intolerance, low predicted FEV₁, low ankle arm index, high serum levels of IL-6, and having one or more inflammatory markers in the highest tertile were significantly associated with low SES and a decline in cognitive function. Odds ratios for cognitive decline in four years, adjusted for sociodemographics, other household members for income, and baseline 3MS score were significantly higher in low SES groups compared with high SES groups (Table 4, Model 1). Low education showed a particularly increased risk: odds ratios were 2.16 (95% confidence interval (CI): 1.59-2.94) for less than 12 years of education, and 1.42 (95% CI: 1.10-1.83) for 12 years of education compared with those with more than 12 years of education. Statistically significant associations between low SES and cognitive decline were found for all three SES indicators. Additional adjustment for biomedical factors (Model 2) decreased the odds ratios by an average of 2% for education, 5% for income, and 8% for the number of assets.

Table 2. Distribution of biomedical factors by education, income, and number of assets

Biomedical factors	Education				Income				Number of assets					
	<12 years	12 years	>12 years	p-value	<\$10,000	\$10,000-<\$25,000	\$25,000-<\$50,000	≥\$50,000	Missing	p-value	None	1-2	3-7	p-value
Heart disease	17.4	15.6	15.2	0.47	15.0	16.1	16.1	16.6	14.6	0.96	17.8	15.1	15.6	0.47
Cerebrovascular disease	7.3	6.8	6.9	0.94	8.6	8.2	6.1	5.2	6.4	0.13	9.2	7.0	6.3	0.16
Glucose intolerance														
Not impaired	56.5	64.4	66.4	<0.001	57.0	61.5	63.8	68.8	66.7	0.01	59.7	60.9	66.7	<0.001
Impaired	18.4	19.1	18.5		17.0	19.4	19.6	17.4	17.5		16.1	18.3	19.9	
Diabetic	25.1	16.4	15.2		25.9	19.1	16.6	13.8	15.8		24.2	20.8	13.5	
Hypercholesterolemia	38.1	43.2	37.5	0.03	42.9	40.9	39.0	33.2	41.5	0.04	45.6	39.8	36.9	0.01
Dyslipidemia	40.5	45.2	45.9	0.09	37.2	43.6	49.3	44.2	42.0	0.01	39.8	42.0	48.1	0.003
Hypertension														
Normotensive	28.1	28.8	36.3	<0.001	23.4	27.4	35.6	40.7	32.3	<0.001	23.2	30.2	36.3	<0.001
Normotensive with medications	30.0	31.8	29.1		34.4	32.3	28.6	27.5	27.8		33.8	30.5	28.9	
Stage 1	28.0	27.0	23.9		27.5	27.7	23.8	22.8	28.2		28.4	27.9	23.4	
Stage 2	13.9	12.4	10.6		14.7	12.6	12.0	9.1	11.7		14.7	11.3	11.4	
FEV ₁ /FVC<70%	22.7	20.2	19.0	0.34	22.6	20.4	21.0	17.1	18.9	0.35	16.9	21.7	19.8	0.15
Predicted FEV ₁ <80%	26.1	20.8	21.5	0.05	26.6	21.9	23.6	18.8	21.3	0.12	21.4	24.5	20.8	0.12
BMI														
<25 kg/m ²	27.0	29.6	36.1	<0.001	28.2	29.9	33.8	35.0	31.3	<0.001	25.8	30.5	34.7	<0.001
25-30 kg/m ²	40.7	42.4	44.0		34.1	41.4	45.5	46.6	42.4		36.6	40.7	46.4	
>30 kg/m ²	32.3	28.0	19.9		37.7	28.7	20.7	18.4	26.3		37.6	28.7	18.9	
Peripheral arterial disease	5.8	4.7	4.0	0.27	5.8	4.8	4.4	3.2	5.8	0.44	6.5	4.7	4.0	0.14
Ankle-arm index <0.9	16.6	13.6	9.8	<0.001	18.1	13.9	10.9	7.0	15.4	<0.001	19.0	15.8	7.8	<0.001
Highest tertile IL-6	36.4	31.0	26.8	<0.001	38.6	32.1	29.3	24.4	28.8	0.001	36.5	33.4	25.8	<0.001
Highest tertile CRP	38.4	33.5	27.8	<0.001	44.0	35.4	26.4	27.4	32.8	<0.001	39.1	35.5	27.1	<0.001
Highest tertile TNF-α	32.5	33.8	29.9	0.19	28.9	33.8	32.7	30.4	28.1	0.42	29.7	32.6	31.8	0.62
Inflammation index *														
No high	38.1	37.0	45.5	<0.001	34.7	37.0	42.9	47.0	45.5	<0.001	37.3	37.1	45.5	<0.001
1 of 3 high	29.0	36.8	32.0		32.1	35.1	33.1	31.6	28.3		30.6	34.5	32.3	
2 of 3 high	24.2	20.0	17.9		25.4	21.1	19.2	16.7	19.1		25.6	21.5	17.1	
all 3 high	8.6	6.2	4.6		7.8	6.8	4.8	4.7	7.0		6.5	6.9	5.2	

* The number of inflammatory marker levels (IL-6, CRP, TNF-α) within the highest tertile

Table 3. Percentage of four year cognitive decline according to the presence of diseases and biological risk factors

Biomedical factors	%	p-value
Heart disease		
No	23.8	0.94
Yes	23.6	
Cerebrovascular disease		
No	23.1	0.03
Yes	30.3	
Glucose intolerance		
Not impaired	22.7	0.02
Impaired fasting glucose	23.0	
Diabetic	29.8	
Hypercholesterolemia		
No	24.2	0.47
Yes	23.0	
Dyslipidemia		
No	23.8	0.96
Yes	23.7	
Hypertension		
Normotensive	22.1	0.56
Normotensive with medications	25.0	
Stage 1	23.6	
Stage 2	24.9	
FEV1/FVC<70%		
No	23.0	0.42
Yes	24.7	
Predicted FEV1 <80%		
No	22.3	0.03
Yes	27.0	
BMI		
<25 kg/m ²	23.8	0.97
25-30 kg/m ²	23.5	
>30 kg/m ²	23.9	
Peripheral arterial disease		
No	23.5	0.11
Yes	29.9	
Ankle-arm index <0.9		
No	21.8	<0.001
Yes	30.6	
IL-6		
Not in highest tertile	21.9	<0.001
In highest tertile	27.6	
CRP		
Not in highest tertile	22.8	0.15
In highest tertile	25.4	
TNF- α		
Not in highest tertile	22.6	0.14
In highest tertile	25.4	
Inflammation index *		
No high	21.6	0.02
1 of 3 high	23.4	
2 of 3 high	25.7	
All 3 high	31.8	

* The number of inflammatory marker levels (IL-6, CRP, TNF- α) within the highest tertile

Results are shown for black and white, men and women, and all ages together because interaction terms between SES and race, SES and sex, and SES and age were not statistically significant (all $p>0.05$). Interactions between SES and biomedical factors were also not statistically significant. In additional analyses we also determined the association between SES and cognitive change as a continuous variable. Results were very similar and the role of biomedical factors also was the same.

Table 4. Odds ratios (OR) and 95% confidence interval (CI) for decline in cognitive function over four years by socioeconomic status.

Socioeconomic status	Model 1 *		Model 2 †		% red. ‡
	OR	95% CI	OR	95% CI	
Education					
>12 years	1.00		1.00		
12 years	1.42	1.10-1.83	1.41	1.09-1.83	2
<12 years	2.16	1.59-2.94	2.14	1.57-2.91	2
Income					
≥\$50,000	1.00		1.00		
\$25,000-<\$50,000	1.72	1.17-2.53	1.70	1.15-2.50	3
\$10,000-<\$25,000	1.65	1.11-2.47	1.64	1.10-2.45	2
<\$10,000	1.67	1.00-2.77	1.61	0.97-2.68	9
Missing	2.05	1.31-3.21	1.99	1.27-3.13	6
Assets					
3-7	1.00		1.00		
1-2	1.27	0.98-1.64	1.25	0.97-1.62	7
None	1.51	1.07-2.12	1.47	1.04-2.07	8

* Model 1: adjusted for age, sex, race, study site, marital status, other household members for income, baseline cognitive function

† Model 2: adjusted for age, sex, race, study site, marital status, other household members for income, baseline cognitive function, and biomedical factors (glucose intolerance, predicted FEV1, ankle arm index, IL-6, inflammation index)

‡ Percentage reduction in odds ratio from model 1 computed by $(OR_{model\ 1} - OR_{model\ 2}) / (OR_{model\ 1} - 1) * 100\%$

DISCUSSION

In an older population, people with low SES had an increased risk of a decline in cognitive function in comparison to people with high SES. Baseline presence of glucose intolerance, low predicted FEV1, low ankle arm index, high serum levels of IL-6, and having one or more inflammatory markers in the highest tertile were significantly associated with low SES and a decline in cognitive function. In spite of these associations, biomedical variables explained only very little of the SES differentials in cognitive decline.

The association between low SES and cognitive decline was found across all SES indicators. Strongest associations were found with education. Besides a SES indicator, education may also be a proxy for general intellectual abilities. Higher

levels of education may also improve an individual's ability to perform on tests of cognitive function (test-taking skills) or can even mask mild cognitive deficits. Our study has a longitudinal design and therefore the potential for this effect is minimized because this differential ability in performing tests would be present at baseline as well as follow-up and we adjusted for baseline cognitive score (4).

Statistically significant associations with poor cognitive function were also found for low income and few assets; this shows the importance of SES as a predictor of cognitive decline. This corroborates a recent study by Mehta et al. (12). In this study, literacy was used as a proxy for SES; this measure was also associated with cognitive test performance. Because of the longitudinal character of this study and because we controlled for cognitive score at baseline, it is clear that SES precedes the decline in cognitive function. To our knowledge the association between income, as SES indicator, and cognitive function has not been studied extensively before. A recent study by Lee et al. (9) showed no statistically significant associations between income and cognitive decline after adjusting for age, in 70-79 year old community-dwelling women. Another study by Evens et al. (6) showed weak associations with income and a decline in cognitive function in a community population of persons over the age of 65 years. Turrell et al. found a significant association between SES, including income, and levels of cognitive function in men aged between 58 and 64 years; this was, however, a cross-sectional study (42).

Biomedical factors explained only 5% of the association between SES and cognitive decline in this study, which does not support the hypothesis that high SES may simply represent better health that effects cognitive function (4). It is plausible that high SES is associated with greater exposure to more cognitively stimulating environments resulting in more extensive brain development or activation that has a positive effect on cognitive function (4, 43). The 'use it or lose it' theory suggests that neurological activation, such as participation in cognitively challenging activities, keeps persons mentally fit until old age (44). Childhood SES, early life conditions, and whether childhood disadvantages persist, are also important in cognitive development and abilities in adulthood (42, 45). A recent study, however, indicates that a higher SES during childhood or more exposure to a cognitively stimulating environment are not associated with a less rapid decline in cognitive function in old age (46). Genetic factors and environmental differences may also be important in cognitive development. A study by Hendrie et al. found lower incidence rates of dementia and Alzheimer disease in Yoruba residents of Nigeria in comparison with African-American residents (all aged 65 years or older), despite the higher SES in the African-American population. It was hypothesized that this was partly explained by different genetic or environmental factors (47). Future research should also take into

account behavioral factors, such as physical activity, smoking, alcohol use, and diet (48-51).

In this study, we had information on a wide variety of diseases and biological risk factors, allowing for a more comprehensive assessment than has been possible previously. Biomedical factors were measured at baseline and we did not have information on childhood health that could also have affected cognitive function. It must also be acknowledged that the approach followed is limited both by the comprehensiveness of the panel of biomedical factors considered, and the reliability with which these factors have been assessed. We cannot exclude the possibility that had the biomedical factors been measured more exactly, the diminution in the strength of the SES effect may have been larger.

Loss to follow-up for our outcome was a limitation in this study. Four hundred eighty-six people, of whom 282 people died, had no cognitive data in the fourth year of follow-up. Those who did not take the 3MS at follow-up had less education, lower income, and fewer assets compared with the study population ($p < 0.001$). Furthermore, they had significantly worse 3MS scores at baseline compared with those who remained in the study ($p < 0.001$). In a decedent proxy interview it was determined whether a person had memory problems prior to death. Of the 282 people who died during the follow-up, 60 persons had memory problems. The association between SES and cognitive decline may have therefore been underestimated. Furthermore, income data were missing for 316 participants, these persons had the highest risk of a cognitive decline and had more often a lower educational level compared with persons with full data. This may have led to an underestimation of the effect of income on a decline in cognitive function. Whether the relative contribution of the biomedical factors is equally underestimated remains unknown.

In summary, low SES predicts a decline in cognitive function in older adults. Besides the evidence that higher prevalence of diseases and their risk factors are related to both low SES and poor cognitive function, in this study, biomedical factors could explain only 5% of the SES differentials in cognitive decline. We therefore could not support the hypotheses that biomedical factors are important in the mechanism underlying the association between SES and cognitive function.

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8

Explaining socioeconomic differences in incident depression in older adults

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ABSTRACT

Objective. The objective of this study was to examine the association between socioeconomic status and the onset of depression in older adults and to determine the relative contribution of psychosocial factors, physical health status, and behavioral factors in explaining this link.

Methods. Data were from 2,593 men and women, aged 55-85 years, participating in the Longitudinal Aging Study Amsterdam (LASA). Two indicators of socioeconomic status were used: education and income. The onset of depression was measured over nine years of follow-up with the Center for Epidemiologic Studies on Depression (CES-D) scale. Cox proportional hazard regression models were fitted to study the association of SES on time to onset of depression.

Results. Adjusted hazard ratios of incident depression were significantly higher in those with low education (hazard ratio (HR):1.52, 95% confidence interval (CI): 1.17-1.99) and low income (HR:1.48, 95% CI: 1.13-1.94). Psychosocial factors, explained on average 16% socioeconomic differences in incident depression. Physical health status reduced the effect of socioeconomic status on depression incidence with on average 7%, and behavioral factors with less than 5%. A small network size, low self-efficacy, and poor physical function had the largest individual contribution in reducing the effect of socioeconomic status on incident depression in this study.

Conclusions. In older adults, low socioeconomic status predicted the incidence of depression. Part of this association was explained by psychosocial factors and physical health status. The SES differences in incident depression remain, however, to a large extent unexplained.

INTRODUCTION

Generally, low socioeconomic status (SES) is related to higher rates of morbidity and mortality and has been shown to be an important predictor of poor functioning (1, 2). SES differences have also been found in mental disorders, including depression (2-5). Low SES is related to a higher prevalence and incidence of depression (3-5). Depression is a common disorder in late life with negative consequences for well-being and daily functioning (6-8) and is also associated with higher mortality rates (9). In order to reduce depression in older persons and the SES differences therein, it is important to understand the underlying mechanisms that may mediate the association between SES and depression. This may help to provide tools for prevention and intervention strategies. Psychosocial factors as well as physical health status and behavioral factors may be important in explaining SES inequalities in depressive symptoms (4, 10, 11).

People with low SES generally have fewer psychosocial resources than people with high SES. Low SES is related to low control beliefs, negative coping styles, poor social support, and more stressful life events (12-14). The association between psychosocial factors and depression has been studied extensively. Several studies have found a favorable effect of social support on mental health (15-17). Stressful life events have a negative impact on depressive symptoms (10, 18).

Physical health status may also be important in the explanation of SES differences in depressive symptoms. Low SES is a risk factor for many diseases, including cardiovascular disease, stroke, and diabetes (5, 19-22). The prevalence of chronic diseases is associated with increased depressive symptoms (23-25). Physical limitations should also be taken into account in the explanation of SES differences in depressive symptoms. Low SES is a strong predictor of poor physical function in older adults (26, 27), which is in turn a predictor of depression (16, 28).

Finally, behavioral factors may play a role in the explanation of SES inequalities in depressive symptoms. Low SES is related to many adverse behavioral factors, such as smoking, excessive alcohol consumption and decreasing physical activity (29, 30). There is also some evidence that adverse behavioral factors are associated with depressive symptoms (11, 31, 32).

The aim of the present study is to examine whether low SES predicts the onset of depression over nine years of follow-up in a representative sample of Dutch older adults, who participated in the Longitudinal Aging Study Amsterdam. The contribution of a number of potentially explanatory factors to explain SES differences in depression has been studied before. In this study, however, several explanatory factors including psychosocial factors, physical health status, and behavioral factors,

will be simultaneously examined in the explanation of SES inequalities in depression incidence.

METHODS

Study population

Data for this study were collected in the Longitudinal Aging Study Amsterdam (LASA), an ongoing cohort study on predictors and consequences of changes in well-being and autonomy in older persons in the Netherlands (33). A random sample of persons, 55-85 years of age, was drawn from the population registers of 11 municipalities. This sample was originally recruited for the NESTOR study on Living Arrangements and Social Networks of older adults (LSN) in 1992 (34). The response rate was 62.3% ($n=3,805$). About eleven months after the LSN interview, 3,107 (81.7%) subjects were enrolled in the main baseline interview of LASA conducted between September 1992 and September 1993. Since then three follow-up measurements took place, the first in 1995/1996, the second in 1998/1999, and the third in 2001/2002. At the first follow-up in 1995/1996, 2,545 participants took part in the study; 416 (13.4%) had died and 146 (4.7%) were lost to follow-up for other reasons (refusal, too frail, could not be contacted). In 1998/1999, at the second follow-up measurement, 2,076 persons participated in the study; 343 (13.5%) had died and 126 (5.0%) were lost to follow-up for other reasons. At the third follow-up measurement, 1,691 persons took part in the study; 289 (13.9%) had died and 96 (4.6%) were lost to follow-up for other reasons. In order to study the onset of depression, respondents with depression at baseline ($n=448$) were excluded (CES-D score ≥ 16). Furthermore, 66 persons were excluded because of incomplete data, resulting in 2,593 participants. For 80 participants, income data were missing resulting in 2,513 participants for the analyses with income.

Measures

Socioeconomic status. Two indicators of SES, measured at baseline, were used: level of education and household income. Categories for level of education were: high (university, college, higher vocational, general secondary, and intermediate vocational education); medium (general intermediate, and lower vocational education); and low (elementary education or less). For monthly net household income three categories (tertiles), in euros, were distinguished: high (greater or equal to 1,035); medium (between 625 and less than 1,035); low (less

than 625) (35). For participants with a partner living in the household, household income was multiplied by 0.7 to make them comparable to the incomes in a one-person household. Income data at baseline were missing for 370 respondents. For 290 respondents, imputations were made using income data collected at the LSN measurement in 1992 or follow-up measurements (35). For 80 persons income data at all measurements were missing. They were excluded from the analyses with income.

Depression. Depressive symptoms were assessed at each measurement cycle with the Center for Epidemiologic Studies on Depression (CES-D) scale, a 20-item self-report scale designed to measure depressive symptoms during the previous week (36). Scores ranged from 0 to 60, with higher scores indicating more depressive symptoms. A cut-off score of 16 was used as a criterion for clinically significant depressive symptoms, here called depression (37). In addition to this cut-off score, subjects were classified with incident depression when they also had a relevant increase in depressive symptoms which was determined as an increase of $0.5 \times \text{standard deviation}$ ($=4$ points) in CES-D score between baseline and follow-up measurement (38, 39). First incident depression was defined as an increase of 4 points or more in CES-D score and thereby crossing the cut-off score of 16.

Mediators. Psychosocial factors, measured at baseline, included partner status (presence of a partner, yes or no), network size, instrumental and emotional social support, mastery, and self-efficacy. The size of the personal network was determined by asking the respondent to identify the persons, other than their partner, whom they had frequent contact with and who were important to them (range is 0-75) (40). For a maximum of nine network members with whom contact was most frequent, information was collected regarding the intensity of received instrumental and emotional support (range is 0 (no social support)-36). Mastery was measured by an abbreviated version (5-item) of the Pearlin Mastery Scale (range is 5 (lowest)-25) (41). Self-efficacy was measured with a 12-item version of the General Self-efficacy Scale of Sherer et al. (range is 12 (lowest)-60) (42, 43). Missing values for network size ($n=126$), instrumental support ($n=133$), emotional support ($n=135$), mastery ($n=74$), and self-efficacy ($n=115$) were replaced by group means in order to maintain an optimal sample size for the analyses.

Physical health status covered diseases and physical functioning. Baseline presence of diseases was determined by asking the participants whether they had any of the following chronic diseases: cardiac disease, peripheral arterial disease, stroke, diabetes mellitus, lung disease, cancer, and arthritis (44). Physical functioning was measured by the degree to which the respondent had difficulty performing six daily activities: walking up and down a 15-step staircase without resting, getting

(un)dressed, getting up from and sitting down in a chair, cutting own toenails, walking 5 minutes outdoors without resting, and using own or public transport (45). The sum score of the six items ranged from 0 to 24, with lower scores indicating more limitation in physical function.

Behavioral factors included smoking, alcohol use, physical activity, and body mass index (BMI). Four categories of smoking were created: current, former, never, and missing. Categories of alcohol use were: not drinking, moderate drinking, excessive drinking (at least three glasses daily), and missing. Physical activity was defined as the total number of physical activities performed in the past two weeks including walking, cycling, gardening, performing light and heavy household tasks, and two sports activities. Four categories were created: 1-3, 4-5, 6-7, and missing. BMI (weight in kilograms divided by height in meters squared) was categorized as lower than 25 kg/m², between 25 and 30 kg/m², greater of equal to 30 kg/m², and missing. Data on behavioral factors were missing because these data were collected in a medical interview in which 86% of the study sample took part.

Sociodemographics that were included as covariates were age and sex.

Statistical analyses

Differences in baseline characteristics between SES groups were determined using chi-square tests for categorical variables and analyses of variance for continuous variables. To determine if there were differences in time to incidence of depression between SES groups, unadjusted Cox proportional hazard regression models were fitted. If a person developed depression, person time was set at the midpoint between the previous interview and the interview during which the person reported a depression. Persons surviving with no evidence of incident depression were censored at the last follow-up measurement. Those who died with no evidence of incident depression were censored at time of death, and those lost to follow-up were censored at their last follow-up measurement.

To evaluate the association of psychosocial factors, physical health status, and behavioral factors with time to incident depression, Cox proportional hazard regression models were fitted, adjusting for sociodemographics. To study the association of SES with time to incident depression and the role of the mediators therein, five models were fitted. The first model adjusted for age and sex. In model 2, 3, and 4, psychosocial factors, physical health status, and behavioral factors, were separately introduced into the first model. The fifth model included all variables of the previous models. A percentage reduction in hazard ratio from model 1 was computed: $(HR_{\text{model 1}} - HR_{\text{model x}}) / (HR_{\text{model 1}} - 1) * 100\%$. The proportional hazards assumption was investigated by testing the constancy of the log hazard ratio over

time by means of log-minus-log survival plots and interactions with time (log transformed). According to the tests, the proportional hazard assumption was not violated. All analyses were performed using SPSS version 12.0.

RESULTS

Table 1 shows baseline characteristics of the study population by education and income. There were significantly more women in low SES groups and respondents were older in low SES groups. People with low education or income had significantly higher baseline CES-D scores, indicating more depressive symptoms, compared with low SES groups ($p < 0.01$). People with a low education or income level less often had a partner ($p < 0.01$). Low SES was also associated with a smaller network, less emotional support, lower feelings of mastery, and lower self-efficacy compared with high SES people ($p < 0.01$). Generally, the prevalence of diseases was higher in low SES groups. Only the prevalence of diabetes mellitus and arthritis was significantly higher in both the lowest education and income group ($p < 0.01$). People with low education or income had significantly lower baseline physical function scores, indicating worse function, compared with high SES groups ($p < 0.01$). In low SES groups, there were more non-smokers and less people drank alcohol ($p < 0.01$). Furthermore, people in low SES groups were less physically active and had a higher BMI than those in high SES groups.

Results are shown for men and women, and all ages together because interaction terms between SES and sex, and SES and age were not statistically significant. The incident rate per 1,000 person years of depression according to SES is shown in figure 1. People with low SES had a two times higher incidence rate of depression than those with high SES ($p < 0.01$).

Table 2 shows the association between psychosocial factors, physical health status, behavioral factors and incident depression. Of the psychosocial factors, a small network (HR: 1.51, 95% CI: 1.19-1.92), low mastery (HR: 2.01, 95% CI: 1.59-2.53) and low self-efficacy (HR: 1.64, 95% CI: 1.30-2.07) were significant predictors of incident depression. The prevalence of peripheral arteriosclerosis (hazard ratio (HR): 1.50, 95% confidence interval (CI): 1.09-2.05), lung disease (HR: 1.44, 95% CI: 1.06-1.94), cancer (HR: 1.72, 95% CI: 1.28-2.31), and arthritis (HR: 1.48, 95% CI: 1.21-1.81) were also significant predictors of incident depression. Furthermore, poor physical function was a strong predictor of incident depression with a hazard ratio of 1.84 (95% CI: 1.39-2.43) in the group with the lowest physical function scores. Of the behavioral factors, only low physical activity was significantly associated with the

Table 1. Baseline characteristics of the study population by SES

Baseline characteristics	Education n=2,593				Income n=2,513			
	High n=706	Medium n=803	Low n=1084	p	High n=845	Medium n=850	Low n=818	p
Women, %	33.7	46.8	61.8	<0.01	38.9	44.8	64.2	<0.01
Age, mean (SD)	68.8 (8.7)	69.1 (8.5)	72.2 (8.6)	<0.01	66.8 (8.6)	69.6 (8.3)	74.1 (7.5)	<0.01
Depressive symptoms CES-D (0-60), mean (SD)	4.8 (4.0)	5.2 (4.1)	5.8 (4.2)	<0.01	4.9 (4.1)	5.4 (4.2)	5.6 (4.2)	<0.01
Psychosocial factors								
With partner, %	76.3	77.8	61.4	<0.01	79.2	79.8	54.3	<0.01
Network size (0-75), mean (SD)	15.6 (9.0)	14.4 (8.1)	13.2 (7.5)	<0.01	16.0 (9.0)	14.2 (7.8)	12.6 (7.6)	<0.01
Instrumental support (0-36), mean (SD)	14.1 (6.5)	13.9 (6.4)	14.3 (6.7)	0.43	14.1 (6.2)	14.1 (6.5)	14.3 (7.1)	0.83
Emotional support (0-36), mean (SD)	22.9 (7.7)	21.9 (7.7)	20.7 (8.0)	<0.01	22.8 (7.5)	21.9 (7.9)	20.6 (8.3)	<0.01
Mastery (5-25), mean (SD)	17.9 (3.0)	17.8 (2.9)	17.4 (3.1)	<0.01	18.0 (3.0)	17.8 (3.0)	17.4 (2.9)	<0.01
Self-efficacy (12-60), mean (SD)	43.8 (4.9)	42.5 (4.7)	41.2 (5.0)	<0.01	43.4 (4.9)	42.4 (5.0)	41.2 (5.0)	<0.01
Physical Health status								
Cardiac disease, %	18.0	18.3	19.3	0.75	16.8	18.2	21.8	0.03
Peripheral atherosclerosis, %	7.4	7.9	10.2	0.07	5.3	10.0	11.1	<0.01
Stroke, %	3.3	5.1	5.5	0.08	2.5	3.9	7.7	<0.01
Diabetes mellitus, %	5.0	5.7	10.0	<0.01	4.4	7.3	9.7	<0.01
Lung disease, %	9.5	10.2	10.8	0.66	8.6	11.0	11.3	0.15
Cancer, %	6.7	8.4	9.8	0.07	7.0	8.5	10.6	0.03
Arthritis, %	27.6	28.9	34.4	<0.01	26.7	30.5	36.4	<0.01
Physical function (0-24), mean (SD)	22.5 (3.4)	22.1 (4.0)	20.7 (5.0)	<0.01	22.7 (3.1)	22.2 (3.7)	20.1 (5.4)	<0.01
Behavioral factors								
Smoking, %								
Non-smoker	18.4	26.2	32.3	<0.01	19.4	23.4	37.3	<0.01
Former	46.9	43.3	31.9		46.9	42.4	30.7	
Smoker	24.4	20.2	19.9		24.3	22.5	17.1	
Missing	10.3	10.3	15.9		9.5	11.8	14.9	
Drinking, %								
Non-drinker	11.2	17.3	23.2	<0.01	11.4	17.1	25.4	<0.01
Moderate	72.4	67.2	58.4		72.3	66.4	58.2	
Excessive	5.8	4.6	2.1		6.3	4.5	1.1	
Missing	10.6	10.8	16.3		10.1	12.1	15.3	
Physical activity, %								
6-7	21.5	19.7	14.2	<0.01	23.0	17.4	14.3	<0.01
4-5	42.8	44.8	41.1		45.1	45.6	39.6	
0-3	32.4	28.9	36.2		28.9	31.4	39.7	
Missing	3.3	6.6	8.6		3.1	5.5	6.4	
BMI, %								
<25	38.2	30.6	22.5	<0.01	35.9	28.9	23.5	<0.01
25-<30	39.1	43.5	38.7		41.8	41.5	39.1	
≥30	9.5	14.4	19.8		10.8	15.3	20.0	
Missing	13.2	11.5	19.0		11.6	14.2	17.4	

Table 2. Hazard ratios (HR) and 95% confidence interval (CI) for incident depression by physical health status, behavioral factors, and psychosocial factors *

	Incident depression	
	HR	95% CI
Psychosocial Factors		
Without partner	1.23	0.97-1.54
Network size		
Highest tertile	1.00	
Middle tertile	1.09	0.85-1.40
Lowest tertile	1.51	1.19-1.92
Instrumental support		
Highest tertile	1.00	
Middle tertile	0.98	0.77-1.25
Lowest tertile	0.96	0.76-1.20
Emotional support		
Highest tertile	1.00	
Middle tertile	1.03	0.81-1.30
Lowest tertile	1.11	0.87-1.41
Mastery		
Highest tertile	1.00	
Middle tertile	1.22	0.94-1.59
Lowest tertile	2.01	1.59-2.53
Self-efficacy		
Highest tertile	1.00	
Middle tertile	1.24	0.95-1.61
Lowest tertile	1.64	1.30-2.07
Physical Health Status		
Cardiac disease	1.22	0.95-1.56
Peripheral atherosclerosis	1.50	1.09-2.05
Stroke	1.22	0.75-1.99
Diabetes mellitus	0.89	0.57-1.38
Lung disease	1.44	1.06-1.94
Cancer	1.72	1.28-2.31
Arthritis	1.48	1.21-1.81
Physical function		
Highest tertile	1.00	
Middle tertile	1.80	1.40-2.32
Lowest tertile	1.84	1.39-2.43
Behavioral Factors		
Smoking		
Non-smoker	1.00	
Former	0.93	0.72-1.20
Smoker	1.27	0.95-1.69
Missing	0.64	0.43-0.95
Drinking		
Non-drinker	1.00	
Moderate	0.95	0.74-1.21
Excessive	1.15	0.65-2.02
Missing	0.62	0.41-0.93
Physical activity		
6-7	1.00	
4-5	1.16	0.87-1.55
0-3	1.37	1.01-1.86
Missing	1.20	0.73-1.95
BMI		
<25	1.00	
25-<30	1.13	0.89-1.44
≥30	1.16	0.87-1.56
Missing	0.77	0.53-1.11

* Adjusted for age and sex

incidence of depression. Subjects with missing data on smoking and drinking, had a significantly lower risk for incident depression compared with non-smokers and non-drinkers.

Hazard ratios of incident depression, adjusted for age and sex, were significantly higher in low SES groups compared with the highest SES group (Table 3: Model 1). Hazard ratios were 1.52 (95% confidence interval (CI): 1.17-1.99) in the lowest education group and 1.48 (95% CI: 1.13-1.94) in the lowest income group. Additional adjustment for psychosocial factors in model 2 reduced the hazard ratios by 7% in the medium education group and 19% in the lowest education group (13% on average) and by 14% in the medium income group and 25% in the lowest income group (20% on average). Network size and self-efficacy had the largest individual contribution in reducing the hazard ratios for SES (not tabulated). Physical health status, mainly due to physical function, decreased the hazard ratios by an average of 6% for education and 8% for income. The reduction in hazard ratios due to behavioral factors was on average less than 5% (Model 4). In the full model (Model 5) that included all variables of the previous models, low education and low income still significantly predicted the incidence of depression.

Figure 1. Incidence rate per 1,000 person years of depression according to socioeconomic status

* p-value based on unadjusted survival analyses

Table 3. Hazard ratios (HR) and 95% confidence interval (CI) of incident depression by education and income

	Model 1 *			Model 2 †			Model 3 ‡			Model 4 §			Model 5		
	HR	95% CI		HR	95% CI	% red ¶	HR	95% CI	% red ¶	HR	95% CI	% red ¶	HR	95% CI	% red ¶
Education															
High	1.00			1.00			1.00			1.00			1.00		
Medium	1.30	0.98-1.72		1.28	0.97-1.70	7	1.29	0.98-1.71	3	1.30	0.98-1.73	0	1.27	0.96-1.70	10
Low	1.52	1.17-1.99		1.42	1.08-1.86	19	1.48	1.13-1.93	8	1.51	1.15-1.98	2	1.39	1.05-1.83	25
Income															
High	1.00			1.00			1.00			1.00			1.00		
Medium	1.44	1.11-1.85		1.38	1.07-1.78	14	1.42	1.10-1.83	5	1.42	1.10-1.84	5	1.37	1.06-1.77	15
Low	1.48	1.13-1.94		1.36	1.04-1.78	25	1.43	1.09-1.88	10	1.47	1.12-1.93	2	1.33	1.01-1.75	31

* Model 1: adjusted for age and sex

† Model 2: adjusted for age, sex, partner status, network size, instrumental social support, emotional social support, mastery, and self-efficacy

‡ Model 3: adjusted for age, sex, cardiac disease, peripheral arterial disease, stroke, diabetes mellitus, lung disease, cancer, arthritis, physical function

§ Model 4: adjusted for age, sex, smoking, drinking, BMI, and physical activity

|| Model 5: all variables of previous models

¶ Percentage reduction in hazard ratios from model 1 computed by $(HR_{\text{model 1}} - HR_{\text{model x}}) / (HR_{\text{model 1}} - 1) \times 100\%$

DISCUSSION

In our study among older adults, low SES predicted an increased incidence of depression during nine years of follow-up. Those with the lowest education or income level had a 50% higher risk of incident depression compared to those in the highest SES groups. The largest reduction in SES differences in incident depression was found after adjustment for psychosocial factors. Psychosocial factors, mainly network size and self-efficacy, explained on average 13% of the educational differences in incident depression and 20% of the income differences in incident depression. Physical health status had less impact in reducing the effect of SES on depression incidence. Finally, behavioral factors explained less than 5% of the association between SES and incident depression. Even after taking into account psychosocial factors, physical health status, and behavioral factors, low education and low income remained significant predictors of depression incidence.

A small network size, low self-efficacy, and poor physical function had the largest individual contribution in reducing the effect of SES on incident depression in this study. People with a low SES had a smaller network of persons who were important to them, which in turn was also associated with a higher incidence of depression. It is unclear why both emotional and instrumental support – important predictors of depression in other studies (15, 17) – were not related to incident depression in our study. Self-efficacy, the extent to which people believe that they can perform a certain behavior, was significantly lower in low SES groups and can be seen as a personal resource to buffer the impact of stress on depression (46). Poorer physical function also contributed to the explanation of SES differences in depression incidence, while diseases were less important. Behavioral factors were only weakly associated with the incidence of depression. This explains the minor role of these factors in reducing the SES gradients in incident depression, which was also found in other studies (4, 47).

Together, psychosocial factors, physical health status, and behavioral factors explained on average less than a quarter of the SES differences in incident depression. Low education and low income remained significant predictors of depression incidence. Only baseline data on explanatory factors were analyzed. The contribution of the explanatory factors in reducing the SES differences in depression incidence may have been larger if these factors had also been considered longitudinally. However, there is evidence that behavioral factors, mainly smoking and alcohol consumption remain rather stable over time; physical activity showed greater variability over time. These changes in health behavior were, however, not related to SES (48). Psychosocial factors, such as network size and social support,

probably also remained rather stable during the follow-up (49, 50). The prevalence of physical diseases, however, is more likely to have changed during the nine years of follow-up which may have led to an underestimation of the contribution of these factors.

Although access to a large representative sample of elderly which could be followed up for a relatively long period, using identical methods and including a wide range of potential explanatory variables are methodological assets of the studies, some limitations must be considered. First, information on physical health status was based on self-report, which may have led to an under- or overestimation of these factors in the explanation of SES differences in depression incidence. Second, missing values for psychosocial factors were replaced by group means, which could have led to an attenuation of the effect of these factors in explaining the SES differentials. In additional analyses (data not shown), however, in which subjects with missing values on psychosocial factors were excluded, the contribution of these factors in the explanation of SES differences in incident depression was very similar. Third, because of a large number of missing data on behavioral factors, a missing category was created and included in the main analyses. Persons with missing behavioral data had significantly lower education and lower income (data not shown). Subjects with missing data on smoking and drinking, however, had a significantly lower risk for depression compared with non-smokers and non-drinkers. It remains unclear how this may have affected our findings. Finally, other factors that were not taken into account in this study could also have been important. In the Whitehall II Study, work characteristics, low job control in particular, explained much of the social inequalities in depressive symptoms (4, 10). That study consists of white-collar middle-aged men and women, while our study consists of people of 55 years and older of whom the largest part (83%) had no paid job anymore and work characteristics in the current or previous job were not measured. Furthermore, material factors, e.g. deprivation, as well as life events have been shown important in the explanation of SES differences in depression (10).

To our knowledge, this is one of the few longitudinal studies specifically studying the association between SES and the incidence of depression in older adults as well as taking into account a broad range of possible explanatory factors. Subjects with depression at baseline were excluded from this study in order to determine the association of SES on time to incident depression during the follow-up. Persons with depression at baseline had a significantly lower educational level and income compared with non-depressed persons (data not shown). To the extent that the baseline association is based on causation rather than selection (depression influences SES), we may have underestimated the association between SES and

depression incidence. Furthermore, people who were lost to follow-up had a significantly lower SES compared to our study population. This may also have led to an underestimation of the association between SES and incident depression.

This study shows that older people with a low level of education or low income are especially at risk of developing depression. In primary care services, special attention is needed for persons with low SES, not only for early recognition but also for care and treatment. Furthermore, a recent study shows that people with depression with low education less often receive specialized mental health services (51).

In summary, low SES predicted the incidence of depression in older adults. Part of this association was explained by psychosocial factors and physical health status. Intervention upon psychosocial characteristics and improving physical function may lower the SES differences and reduce the risk for depression and its consequences for well-being. The SES differences in incident depression remain, however, to a large extent unexplained. Thus, further study is needed to unravel in even more detail the pathways through which low SES leads to depression in older adults.

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General discussion

This thesis described the associations between socioeconomic status (SES) and longitudinal changes in physical, cognitive, and mental function, particularly in late middle-aged and older adults. Additionally, the role of biomedical, behavioral, and psychosocial factors in explaining these differences was explored. This chapter summarizes and discusses the main findings and some conceptual and methodological considerations are made. Furthermore, implications for public health and recommendations for future research are given.

MAIN FINDINGS

Describing socioeconomic differences in health

The first objective of this thesis was to describe the associations between SES and physical, cognitive, and mental function. Strong and consistent associations between SES indicators and functional outcomes were found. Table 1 shows an overview of the associations that were found in the different chapters of this thesis.

Table 1. Overview of the associations between SES and changes in physical, cognitive, and mental function

	Chapter	Education	Income	Occupation	Assets *
Physical function					
Mobility decline (GLOBE)	2 & 3	NS	+	+	
Physical function decline (LASA)	4	+	+		
Incident mobility limitation (Health ABC)	6	+	+		+
Cognitive function					
Cognitive decline (Health ABC)	7	+	+		+
Mental function (depression)					
Incident depression (LASA)	8	+	+		

+ = significant association; NS = not significant association; blank = not investigated

* Assets included money market accounts, saving bonds or treasury bills, home ownership or investment property or housing, a business or farm, stock or stock mutual funds, and IRA or KEOGH account, and other investments.

Low SES predicted a decline in physical function as well as the onset of physical limitations. SES differences in physical function were found in both younger and older adults of the GLOBE study. In people who were between 55 and 85 years old (LASA), low SES was associated with poor physical function during nine years of follow-up. SES differences in physical function even significantly increased over nine years of follow-up in people who were younger than 70 years. SES differences in physical function did not further widen in people of 70 years and older. This may be due to a healthy survivor effect. In the oldest old, the lowest SES group probably

consists of “healthy survivors”, because of selection prior to baseline and attrition due to mortality during the follow-up. For the Health ABC study, a fairly healthy group of older persons, aged between 70 and 79 years old, was selected. All participants were well-functioning at baseline. In this cohort, a strong association between SES and the onset of mobility limitation during four and a half years of follow-up was found. Altogether, our studies show that SES differences in physical function still exist in old age. Low SES is an important predictor of poor physical function.

In an older population (Health ABC), low SES also predicted a decline in cognitive function. The association between low SES and cognitive decline was found across different SES indicators. Many previous studies investigated the association between educational level and cognitive function in which education is treated as a proxy for intellectual abilities rather than as an indicator for SES (1-3). In our study, significant associations with poor cognitive function were also found for low income and few assets. This shows the importance of SES as a predictor of cognitive decline. This study is an important contribution to the existing literature as the association between SES and cognitive function had not been studied extensively before (especially the number of longitudinal studies is limited).

Among older adults of LASA, low SES predicted the onset of depression during nine years of follow-up. This is one of the few longitudinal studies that specifically studied the association between SES and the incidence of depression in older adults.

In sum, low SES was strongly associated with poor physical, cognitive, and mental function over time. This was found across different indicators of SES and these health inequalities persist in old age. An important strength of our studies is that we studied the associations between SES and function longitudinally. Therefore we may conclude that there probably is a causal relationship between SES and changes in function.

Are socioeconomic differences in health still important in old age?

SES differences in physical function were found in both younger and older adults. In the GLOBE study, which consisted of both younger and older adults (aged 15 to 74 years), the association between SES and mobility decline was similar in a group of 55 years or older ($n=736$) compared to those younger than 55 years old ($n=647$). Additionally, interaction terms between any of the SES indicators and age were not statistically significant which confirmed that the association between SES and mobility decline was similar for all ages. In LASA, SES differences in physical function were found in both younger-old (55 to 70 years) and older-old (70 to 85 years) adults. SES differences in physical function even increased over time in the

youngest-old. Low SES also predicted a decline in cognitive function and the onset of depression in older adults. Overall, our studies indicate that SES differences are still important in old age.

Explaining socioeconomic differences in health

The second objective of this thesis was to gain more insight into the mechanisms underlying the associations between SES and the three functional outcomes, specifically the role of biomedical, behavioral, and psychosocial factors. Table 2 shows an overview of the contributions of biomedical, behavioral, and psychosocial factors in explaining SES differences function.

Table 2. Overview of the contributions of biomedical, behavioral, and psychosocial factors in explaining SES differences in physical, cognitive, and mental function

	Biomedical factors	Behavioral factors	Psychosocial factors
Physical function			
Mobility decline (GLOBE)	0		0
Physical function decline (LASA)			
<70 years old	+	+	+
≥70 years old	+	0	+
Incident mobility limitation (Health ABC)	+		
Cognitive function			
Cognitive decline (Health ABC)	0		
Mental function (depression)			
Incident depression (LASA)	0	0	+

+ = significant contribution (explained ≥10%); 0 = minor contribution (explained <10%); blank = not investigated

SES differences in mobility decline could hardly be explained by biomedical factors (disease severity and comorbidity) in the GLOBE study even though low SES was associated with a higher prevalence of severe disease and comorbidities. This shows the cumulative adversities of people with a low SES. People with a low SES had increased risk of mobility decline, which was independent of their more severe disease and the more comorbid diseases they had, compared to people with a higher SES. Psychosocial factors did not further explain the SES differences in mobility decline. Especially low income remained a strong predictor of mobility decline in this chronically ill population. In LASA, biomedical, behavioral, and psychosocial factors were simultaneously examined in the explanation of SES differences in changes in physical function. Behavioral factors largely explained the SES differences in baseline physical function in the 55 to 70 year old age group; psychosocial factors contributed most strongly in the group of 70 years and older. The significant SES

differential decline in physical function that was found in the youngest age group could not be explained by any of the explanatory factors (not tabulated).

In an attempt to explore a possible biomedical mechanism in more detail, the association between SES and serum levels of inflammatory markers, one of the biomedical factors, was studied among 70 to 79 year old well-functioning adults (Health ABC) (not tabulated). Low SES was associated with elevated serum levels of interleukin-6 (IL6), C-reactive protein (CRP), and tumor necrosis factor- α (TNF- α) compared to high SES. This is an important finding, because in the literature high serum levels of inflammatory markers have been prospectively associated with poor functional outcomes and increased mortality (4-7). Given that serum levels of inflammatory markers and other biomedical factors were strongly related to incident mobility limitation, these factors explained a substantial part of the SES differences in incident mobility limitation in the same study population (Health ABC). However, even after taking into account a wide range of diseases and biological risk factors, persons in the lowest SES group still had a significant increased risk of mobility limitation incidence. High serum levels of inflammatory markers, high BMI, and hypertension had the largest contribution in reducing the SES differences in mobility limitation. It is notable that these variables measured biological characteristics rather than diseases. In GLOBE and LASA, biomedical factors included a range of (chronic) diseases rather than biological risk factors.

The biomedical factors that explained a substantial part of the SES differences in incident mobility limitation, explained only very little of the SES differentials in cognitive decline. The hypotheses that biomedical factors are important in the mechanism underlying the association between SES and cognitive function could not be supported.

Part of the SES differences in depression incidence was explained by psychosocial factors. Biomedical factors and mainly behavioral factors were less important. A small network size, low self-efficacy, and poor physical function had the largest individual contribution in reducing the effect of SES on incident depression. SES differences in incident depression remained, however, to a large extent unexplained.

In sum, biomedical, behavioral, and psychosocial factors explained only part of SES differences in health and could not fully explain SES differences in physical, cognitive, and mental function. Hence, SES differences in function to some extent remained unexplained.

CONCEPTUAL AND METHODOLOGICAL CONSIDERATIONS

There are several conceptual and methodological issues that should be considered when interpreting the results of our studies. These issues concern the use of self-reported data, the selection of explanatory factors and outcomes, selection bias, and generalizability of our findings (8).

Neglected interrelations of explanatory factors

Our research model as presented in the first chapter is rather simplified. First, there will be other explanatory factors that were not included in any of our studies; these will be discussed in the section on 'selection of explanatory factors and outcomes'. Second, we assumed that the three groups of explanatory variables have an independent contribution to the explanation of SES differences in function. The mechanisms probably are interrelated, indicating that some mechanisms work through others rather than work independently from each other. For example, psychosocial factors and biomedical factors are causally interrelated, as well as psychosocial factors and behavioral factors.

Generally, lower SES persons live and work in more stressful environments (9-11). Stress may derive from daily problems, life events, job demands or financial problems. Additionally, people with a low SES are less participating in social networks and receive less social support which has negative consequences for physical and mental health (10, 12, 13). Lower SES groups have less buffering resources available to cope with stressful situations. Low SES is, for example, related to lower self-efficacy; this refers to the extent to which people believe that they can perform a certain behavior and can be seen as personal resource to buffer the impact of stress. Chronic stress elevates activities of different physiological systems, such as the immune system, which may lead to diseases (10, 14, 15). The concept of "allostatic load" has been proposed as the wear and tear in the body and brain resulting from chronic overactivity or inactivity of physiological systems (16). Chronic stress and low control may eventually result in vital exhaustion (10). A higher vulnerability to stress may also lead to unhealthy behavior such as smoking or excessive alcohol consumption (10). The contribution of behavioral and psychosocial factors in explaining SES differences in function will partly be through biomedical factors. Adverse behavioral factors, such as smoking or excessive alcohol consumption in the past, may have resulted in diseases in later life. In chapter 5, we studied the association between SES and inflammatory markers, a biomedical factor, in more detail. Low SES was associated with significantly elevated serum levels of inflammatory markers. A substantial part of the inverse association between SES and

serum levels of inflammatory markers was explained by behavioral factors (including smoking, drinking, and obesity). This study shows that adverse behavioral factors may result in higher serum levels of inflammatory markers.

The above-mentioned considerations have consequences for the estimation of the exact (direct and indirect) contribution of each group of explanatory factors in the explanation of SES differences in function. However, the contribution of biomedical, behavioral, and psychosocial factors together is not affected by whether these factors are interrelated or not.

Self-reported data

The use of self-reported data may have introduced a potential source of bias. Our outcomes, physical function, cognitive function, and depression, were entirely based on self-reports. This could have led to an under- or overestimation of function (17, 18). However, the potential for an under- or overestimation of changes in function is minimized when this is due to a stable characteristic of a person, such as negative affectivity (complaining attitude towards health), because this would be present at baseline as well as at follow-up.

Information about biomedical, behavioral, and psychosocial factors was largely based on self-reported data as well. Bias due to social desirability and not knowing or remembering, possibly leads to underestimated contributions of these factors in the explanation of SES differences in function. Bias due to negative affectivity may, however, lead to an overestimation of, for example, the prevalence of diseases (19). Regarding diseases, misreporting differs between conditions (20-22). Moreover, this type of bias may also be related to SES which may lead to differential misclassification (20). More objective information about biomedical factors was available in the Health ABC study. Information about a wide variety of diseases and biological risk factors was partly based on use of specific medications, some clinical assessments, and self-report.

Imprecision of the measurements is a possible bias that may have led to an underestimation of the contribution of the explanatory factors. For example, in LASA and Health ABC only limited information was available about the severity of diseases. Furthermore, the measurements are not a perfect marker for life-time exposure to risk factors (23, 24).

Selection of explanatory factors and outcomes

Three important groups of explanatory variables were included in our studies. Although we selected a comprehensive selection of variables as possible in each study, this set of variables was not exactly the same in GLOBE, LASA, and Health

ABC. Furthermore, we must acknowledge that there were factors that were not measured or that we did not include. First biomedical factors. In the Health ABC study, a wide variety of diseases and biological risk factors were included in the study, allowing for a more comprehensive assessment than has been previously possible. In GLOBE and LASA, a range of (chronic) diseases was included in the study. Less information, however, was available about biological risk factors. This may explain the small contribution of the biomedical factors in explaining SES differences in physical function in GLOBE and LASA. In Health ABC, we show that particularly the biological risk factors, such as high serum levels of inflammatory markers and hypertension, had the largest contribution in reducing the SES differences in mobility limitation. In LASA and GLOBE, we therefore may have underestimated the role of biomedical factors. Second, a potentially important behavioral factor that was not included in our studies is diet. Generally, low SES is related to unhealthier diets (25, 26). Since an unhealthier diet is also associated with poor function (27, 28), the estimation of the role of behavioral factors might have been underestimated. Third, important psychosocial factors that were not included in our studies are job related stressors, for example job control. These factors are known to be related to low SES and poor health outcomes, such as physical function and depression (10, 29-31). In older populations, these factors are probably less important as a large part has no paid job anymore. Finally, we did not examine the role of material factors in the explanation of SES differences in function. Income, being a strong predictor of function in our studies, suggests that the direct availability of financial resources might be important (32). Low SES is related to adverse material factors, such as financial problems (lack of money), housing problems, and adverse physical working conditions (31, 32). Previous studies showed that material factors explained part of SES health inequalities (33-37). Part of the contribution of material factors was, however, through behavioral and psychosocial factors (33, 34, 36). In future research, the factors that were mentioned above should be included to obtain a more detailed picture of the background of SES differences in function.

Besides adult socioeconomic circumstances, childhood socioeconomic circumstances also play a part in the explanation of SES differences in health. From a life-course perspective, the onset of SES health inequalities is a continual process. Studies have shown that childhood SES and early life factors have an independent effect on adult health (38-42). Although we used different indicators of SES that represent different dimensions of SES in different life stages, life course indicators were not incorporated in our studies.

In all our studies, we determined whether SES differences in changes in function could be explained by explanatory variables measured at baseline. The

contribution of explanatory factors in reducing the SES differences in function may have been larger if these factors had also been considered longitudinally. The severity of a disease and comorbidity, for example, is likely to have changed during the follow-up which may have led to an underestimation of the contribution of these factors. Additionally, information about life time exposure to risk factors was not available which may also have led to an underestimation of these factors (43).

Because only baseline data on explanatory factors were analyzed, the association between SES on the one hand and explanatory factors on the other hand might have resulted from reverse causation (explanatory factors influence SES) (44-46). It is likely that this selection effect has a larger effect on income than on educational level, as the latter indicator is less sensitive to change during adulthood. In our studies, similar results were found across all SES indicators, including education.

Our model consisted of three types of function that covered health status; physical, cognitive, and mental function. Social function, another dimension of health and well-being was, however, not included. Social health is perceived as essential and an important condition for well-being and successful aging (47, 48). Studies have shown that low SES is associated with poor social function (49, 50). It has not been studied extensively how SES differences in social function can be explained. Some studies treat social function as a separate subscale of function while other studies incorporate social function in a summary measure for mental function (35, 51). Depression was used as an indicator for mental function. Anxiety, as another common mental disorder, was however not included. Anxiety disorders are also more common among low SES groups (52, 53).

Attrition

Data from three longitudinal cohort studies were used. Especially for the studies that involve older persons, attrition due to morbidity and mortality is a disadvantage. Generally, attrition was higher among low SES groups. Furthermore, people who were lost to follow-up often had poorer function compared to those who remained in the study. Consequently, the remaining study sample is no longer representative of the original study population. This selection may have led to an underestimation of the association between SES and function. However, attrition may not always be a serious problem when associations between variables are the focus of study (54, 55). It remains unknown whether the relative contribution of explanatory variables in the explanation of SES differences in function was equally underestimated.

Generalizability

The sample of the GLOBE study consisted of 15-74 year old men and women with a chronic disease (asthma/COPD, heart disease, diabetes mellitus, and low back pain). Because all participants had a chronic disease we cannot generalize our findings to a healthy population. It is likely that chronically ill persons experience more mobility problems because of their chronic disease than healthy people do. Whether SES differences and the contribution of explanatory variables are different between healthy and chronically ill persons remains unknown.

The LASA study was originally based on randomly selected population-based sample. Attrition, however, has resulted in a relatively healthy study sample; therefore, our result may not be generalizable to more frail older persons or institutionalized persons. The Health ABC study is based on an even healthier older population; all participants were well-functioning at baseline. The latter study is conducted in the United States and therefore may not be comparable to studies in the Netherlands. SES differences in health have found to be larger in the United States than in the Netherlands (56, 57). If we measure the gap between the 5% richest and the 5% poorest in the Health ABC study, the 5% richest had incomes about 10 times those of the poorest 5%. In GLOBE and LASA this ratio was about 5 (data not shown). This may explain the particularly large income differences in physical function in Health ABC compared to GLOBE and LASA. SES differences in function may be larger in the Health ABC study, whether the contribution of explanatory variables is different remains unclear.

IMPLICATIONS

The primary aim of the studies presented in this thesis was to provide a better understanding of the factors that contribute to SES health differences. However, this may also provide more guidance to policy recommendations to reduce these inequalities. Over the past 15 years, the Dutch ministry of health has followed a systematic research based approach to reduce SES differences in health (58, 59). The main focus of the program was on developing and evaluating interventions and policies. The strategy covered four intervention and policy measures: (1) reducing inequalities in socioeconomic goods; (2) diminishing the effect of health on SES; (3) changing the distribution of specific risk factors across SES groups; (4) offer extra health care for people in low SES groups.

Despite the efforts to reduce SES differences in health, our studies show that SES differences are still a major public health problem. SES differences in physical

function even significantly increased over time in younger-old adults (55 to 70 years). Our findings have implications for the third type of policy measures as mentioned above in order to reduce SES inequalities in health. We showed that a low SES was associated with a higher prevalence of diseases and their risk factors, adverse behavioral factors, and fewer psychosocial resources compared to high SES. The unequal distribution of these risk factors explained part of the SES differences in function. Policy measures that focus on a change in the distribution of risk factors might therefore help to reduce the SES inequalities. Programs to promote, for example, healthy behavior may be most beneficial in early age (58). However, also in an older population it may be useful to improve health behaviors, especially in low SES groups. In one of our studies we showed that elevated serum levels of inflammatory markers in low SES groups were to a large extent mediated by unhealthy behaviors (especially obesity, smoking and drinking) in these groups (Chapter 5). Improving health behaviors may reduce risks associated with inflammation and indirectly reduce SES differences in health. Unhealthy behavior is not simply a lifestyle choice, it is shaped and constrained by social and physical environments linked to SES (9). Interventions should therefore be directed to both the individual and the environment (such as improving housing and neighborhood conditions). Intervention upon psychosocial characteristics might also be possible to reduce SES differences in health. For example, improving control beliefs in low SES groups by self-management skills (60, 61). In general, interventions should be directed to specific target populations with a specific approach for each group. It might be useful to identify and focus on groups that are especially at risk to develop health problems (62). For example, people with a low SES, who also smoke and have low self-efficacy, might be at higher risk than other low SES people.

Several interventions have proven to be effective and a few have been implemented on a larger scale, such as programs to prevent school children from starting smoking (58). Whether it is possible to substantially reduce SES differences in health among older persons remains unknown. Policy developments on health inequalities in different European countries between 1990 and 2001 were analyzed (63). The UK, the Netherlands, and Sweden have proposed comprehensive approaches to reduce health inequalities. Strategies differed between countries, which suggest that policymaking in this area is still in a growth stage and more accurate evidence-based approaches are needed. International exchange of experiences with developing, implementing, and evaluating interventions and policies is desirable to increase the development in this field (63).

FUTURE RESEARCH

Our studies indicate that SES differences in health are an important issue in old age. SES differences existed in different dimensions of health in older persons. As there is particularly still limited research on SES differences in cognitive function, more longitudinal studies on this subject are required that particularly examine the underlying mechanism. We only studied the role of biomedical factors that could hardly explain the SES differences in cognitive decline; the role of behavioral factors and psychosocial factors is still unknown. This research may be especially important, because within an aging population and the increasing life expectancy, the number of people with cognitive impairment and dementia will substantially increase.

The Dutch government advisory committee that developed intervention and policy strategies to reduce SES differences in health recommended that continuing explanatory research is necessary because this may lead to new entry points for interventions (58, 59). The studies presented in this thesis contribute to this by unraveling the contribution of biomedical, behavioral, and psychosocial factors. None of these factors could, however, fully explain the SES differences in health. Studies in which health and exposure to risk factors are measured during the entire life-course are needed to study SES differences in health and the explanation in more detail. Further study is also needed to examine how these factors are interrelated in order to determine the exact (direct and indirect) contribution of these factors.

IN CONCLUSION

Low SES was strongly associated with poor physical, cognitive, and mental function over time and these health inequalities persist in old age. Biomedical, behavioral, and psychosocial factors explained part of the SES health differences, but could not fully explain SES differences in physical, cognitive, and mental function. Hence, SES differences in function to some extent remained unexplained.

Further study in unraveling the mechanisms underlying SES differences in health is needed, followed by interventions, in both younger and older persons, to decrease SES health differences. Older people with a low SES are especially at risk to develop problems regarding physical, cognitive, and mental function. In health care, special attention is needed for persons with low SES for early recognition of functional problems, care, and treatment. Especially low SES persons are worse off as they have a shorter life expectancy and also live a greater proportion of their lives in poor health. A decrease in SES differences in function will also decrease SES differences in (healthy) life expectancy.

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Summary

Chapter 1 outlines the background of the studies in this thesis and presents the objectives of this thesis. Low socioeconomic status (SES) has been shown to be an important predictor of different functional health outcomes. Particularly the association between low SES and poor physical function has been well described in the literature. There is also evidence that low SES is related to poor cognitive function, although longitudinal studies are limited. Furthermore, SES differences have been found in mental disorders, such as depression. In general, relatively few studies have investigated the association between SES and functional outcomes in older persons and longitudinal studies are limited. In order to prevent the negative consequences of poor function and the SES differences therein, understanding the pathways underlying these associations is important. Biomedical factors (such as diseases), behavioral factors (such as smoking and drinking), and psychosocial factors (such as long term difficulties and social support) possibly play an important role herein.

The first objective of this thesis is to describe the associations between SES and changes in physical, cognitive, and mental function. The second objective of this thesis is to explore the role of biomedical, behavioral, and psychosocial factors in the explanation of SES differences in the three functional outcomes. The studies that are presented in this thesis have important strengths. First, the associations between SES and three different types of function, that represent different aspects of a person's health status, are studied. Second, the role of three different groups of explanatory factors is studied. Third, the associations between SES and function are studied longitudinally. Fourth, it will be determined whether SES differences in function still exist in old age. Finally, different indicators of SES, including education and income, that represent different dimensions of SES, will be used. Including these strengths, the studies will contribute to a better understanding of the mechanism underlying SES health differences.

Data from three different large longitudinal cohort studies were used; the GLOBE (Dutch acronym for Health and Living conditions of the population of the city of Eindhoven and surroundings in the Netherlands) study, LASA (Longitudinal Aging Study Amsterdam), and the Health ABC (Health, Aging and Body Composition) study.

Chapter 2 describes the association between SES and mobility decline and whether this could be explained by disease severity and comorbidity in four different chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, and low back pain), using data from the GLOBE study. This study shows that in 15 to 74 year old persons, low occupational level and low income predicted an increased risk of

mobility decline during six years of follow-up. Only little of this association could be explained by the higher disease severity and comorbidity in these patients. Findings were similar for patients with asthma/COPD, heart disease, diabetes mellitus, and chronic low back pain.

Chapter 3 elaborates – using data from the GLOBE study – on the previous chapter by examining the role of psychosocial factors, independent of disease severity, comorbidity, and health-related behavior, in the further explanation of SES differences in mobility decline in chronically ill persons. Although low SES was associated with a more negative psychosocial profile, psychosocial factors could not explain the SES differences in mobility decline.

In *Chapter 4* the association between SES and changes in physical function is investigated in younger-old (55 to 70 years) and older-old (70 to 85 years) adults. Additionally, several explanatory factors, including diseases, behavioral, and psychosocial factors are simultaneously examined in the explanation of SES differences in physical function. Using data from LASA, we reported that in older adults, low SES was related to a poorer level of physical function during nine years of follow-up. In persons aged 55 to 70 years, the SES differentials in physical function became larger during nine years of follow-up, while SES differentials did not further widen over time in subjects 70 years and older. Behavioral factors largely explained the SES differences in physical function, whereas psychosocial factors reduced SES differences most in the oldest age group. These findings indicate age-specificity of both the pattern of SES differences in function and the mechanisms underlying these associations.

Chapter 5 reports on the association between SES and different markers of inflammation and whether any association remains significant after adjusting for diseases and behavioral factors typically related to elevated serum levels of inflammatory markers. In well-functioning older adults, participating in the Health ABC study, low SES was associated with elevated serum levels of interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor- α (TNF- α) compared to high SES. This is an important finding, as high serum levels of inflammatory markers have been associated with increased mortality. Elevated serum levels of inflammatory markers in low SES groups were to a large extent mediated by unhealthy behaviors (especially obesity, smoking, and drinking), rather than by a higher prevalence of diseases in low SES groups.

Chapter 6 describes the association between SES and incident mobility limitation in 70 to 79 year old persons who were well-functioning at baseline (Health ABC study). Furthermore, the role of biomedical factors in explaining these differences is examined. Low SES strongly predicted an increased incidence of mobility limitation during four and a half years of follow-up. Biomedical factors, including a wide range of diseases and biological risk factors, explained part of this association. On average 41% of the educational differences was explained by biomedical factors; this was 17% for income and 29% for the number of financial assets (such as saving bonds, home ownership, and investments) that a person owned. High serum levels of inflammatory markers, high body mass index, and hypertension were found most important in reducing the SES differences in mobility limitation incidence. This study suggests that biomedical factors are important in the mechanism underlying the association between SES and functional outcomes. However, in order to reduce physical disability and particularly the SES differences therein, it may not be sufficient to solely intervene upon biological risk factors and risks of diseases.

Chapter 7 shows that in a well-functioning older population (Health ABC study), people with low SES also had an increased risk of a decline in cognitive function in comparison to people with high SES. Although a higher prevalence of diseases and their risk factors were related to both low SES and poor cognitive function, in this study, biomedical factors could explain only 5% of the SES differences in cognitive decline. The hypothesis that biomedical factors are important in the mechanism underlying the association between SES and cognitive function could not be supported.

In *Chapter 8*, it is examined whether low SES predicts the onset of depression in older adults who participated in LASA. The contribution of several explanatory variables, including psychosocial factors, physical health status, and behavioral factors is simultaneously examined in the explanation of SES inequalities in depression incidence. Low SES predicted an increased incidence of depression during nine years of follow-up. The largest reduction in SES differences in incident depression was found after adjustment for psychosocial factors. A small network size, low self-efficacy, and poor physical function had the largest individual contribution in reducing the effect of SES on depression incidence in this study. The SES differences in incident depression remained, however, to a large extent unexplained.

Chapter 9 summarizes and discusses the main findings. Furthermore, conceptual and methodological issues are considered. Despite some limitations, we conclude that low SES was strongly associated with poor physical, cognitive, and mental function over time and that these health inequalities persist in old age. Biomedical, behavioral, and psychosocial factors could not fully explain SES differences in physical, cognitive, and mental function. Hence, SES differences in function to some extent remained unexplained. Further study in unraveling the mechanism underlying SES differences in health is needed, followed by interventions – also in old age – to decrease SES health differences.

Samenvatting

Hoofdstuk 1 schetst de achtergrond van de studies die in dit proefschrift worden beschreven en presenteert de doelstellingen van dit proefschrift. Een lage sociaal-economische status (SES), gemeten door bijvoorbeeld inkomen of opleiding, is een belangrijke voorspeller voor verschillende domeinen van functioneren. Vooral de relatie tussen een lage SES en slecht fysiek functioneren is goed beschreven in de literatuur. Er zijn ook aanwijzingen dat een lage SES gerelateerd is aan een lager niveau van cognitief functioneren, hoewel er nog niet veel longitudinale studies op dit terrein zijn uitgevoerd. Daarnaast zijn er ook SES verschillen gevonden in mentaal functioneren, waaronder depressie. Over het algemeen is er relatief weinig onderzoek gedaan naar de relatie tussen SES en functioneren bij oudere mensen en zijn er weinig longitudinale studies. Om de negatieve gevolgen van slecht functioneren te voorkomen en vooral de SES verschillen daarin, is het belangrijk om inzicht te krijgen in de onderliggende mechanismen. Biomedische factoren (zoals ziekten), gedragsfactoren (zoals roken en drinken) en psychosociale factoren (zoals langdurig belastende omstandigheden en sociale steun) spelen hierin mogelijk een belangrijke rol.

Het eerste doel van dit proefschrift is het beschrijven van de relaties tussen SES en veranderingen in fysiek, cognitief en mentaal functioneren. Het tweede doel van dit proefschrift is om te onderzoeken in hoeverre biomedische factoren, gedragsfactoren en psychosociale factoren de relatie tussen SES en verschillende domeinen van functioneren kunnen verklaren. De studies die beschreven worden in dit proefschrift hebben belangrijke sterke punten. Ten eerste, de relaties tussen SES en drie verschillende domeinen van functioneren die verschillende aspecten van iemands gezondheidsstatus weergeven, worden onderzocht. Ten tweede, de rol van drie verschillende groepen van verklarende factoren wordt onderzocht. Ten derde, de relaties tussen SES en functioneren worden longitudinaal bestudeerd. Ten vierde, er wordt onderzocht of deze SES verschillen in functioneren nog steeds aanwezig zijn op oudere leeftijd. Tot slot, verschillende indicatoren van SES, waaronder opleiding en inkomen, die verschillende dimensies van SES weergeven, worden gebruikt. Deze punten dragen bij aan het verkrijgen van een beter inzicht in de mechanismen die ten grondslag liggen aan SES verschillen in functioneren.

Er wordt gebruik gemaakt van gegevens van drie longitudinale cohortonderzoeken: de GLOBE (Gezondheid en LevensOmstandigheden Bevolking Eindhoven en omstreken) studie, LASA (Longitudinal Aging Study Amsterdam) en de Health ABC (Health, Aging and Body Composition) studie.

Hoofdstuk 2 beschrijft de relatie tussen SES en achteruitgang in mobiliteit in 4 groepen chronisch zieken (astma/COPD, hartziekte, diabetes mellitus en lage

rugpijn). Gegevens van de GLOBE studie werden gebruikt. Daarnaast wordt onderzocht in hoeverre de ernst van de chronische ziekte en co-morbiditeit (het hebben van andere ziekten) deze relatie kunnen verklaren. De resultaten laten zien dat bij mensen in de leeftijd van 15 tot 74 jaar een laag beroepsniveau en een laag inkomen een verhoogd risico vormen op een achteruitgang in mobiliteit gedurende een follow-up van 6 jaar. Slechts een klein deel van deze relatie kon verklaard worden door een ernstigere ziekte en co-morbiditeit in de lage SES groepen. De bevindingen waren vergelijkbaar voor mensen met astma/COPD, hartziekte, diabetes mellitus en lage rugpijn.

Hoofdstuk 3 bouwt – eveneens met gegevens uit de GLOBE studie – voort op het vorige hoofdstuk waarbij de rol van psychosociale factoren, onafhankelijk van ernst van de ziekte, co-morbiditeit en gedragsfactoren, wordt bepaald in de verdere verklaring van SES verschillen in fysiek functioneren bij chronisch zieken. Ondanks dat een lage SES geassocieerd was met meer negatieve psychosociale kenmerken konden deze factoren de SES verschillen in een achteruitgang in mobiliteit niet verder verklaren.

In *Hoofdstuk 4* wordt de relatie tussen SES en veranderingen in fysiek functioneren bij jongere ouderen (55 tot 70 jaar) en oudere ouderen (70 tot 85 jaar) onderzocht. Bovendien worden verschillende verklarende factoren waaronder ziekten, gedragsfactoren en psychosociale factoren, gelijktijdig onderzocht in het verklaren van de SES verschillen. Gebruikmakend van gegevens van LASA laten we zien dat bij oudere mensen een lage SES is gerelateerd aan slechter fysiek functioneren gedurende 9 jaar follow-up. In de groep van 55 tot 70 jaar werden de SES verschillen in fysiek functioneren zelfs groter gedurende de follow-up; dit was niet het geval in de groep van 70 jaar en ouder. Gedragsfactoren verklaarden het meest van de SES verschillen in de jongere groep, terwijl in de oudere groep psychosociale factoren de grootste bijdrage hadden in het verkleinen van de SES verschillen. Deze bevindingen wijzen erop dat zowel de beschrijving van de SES verschillen in fysiek functioneren als de verklaring hiervan leeftijdspecifiek is bij ouderen.

Hoofdstuk 5 beschrijft de relatie tussen SES en verschillende ontstekingsmarkers. Daarnaast bepalen we of de relatie blijft bestaan na correctie voor ziekten en gedragsfactoren die geassocieerd zijn met hoge bloedwaarden van ontstekingsmarkers. Bij 70 tot 79 jaar oude mensen uit de Health ABC studie was een lage SES geassocieerd met hoge bloedwaarden IL-6 (interleukine-6), CRP (C-

reactieve proteïne) en TNF- α (tumor necrosis factor- α) vergeleken met een hoge SES. Dit is een belangrijke bevinding omdat hoge waarden van ontstekingsmarkers samenhangen met een hogere kans op sterfte. Verhoogde waarden van ontstekingsmarkers in lage SES groepen werden vooral veroorzaakt door ongezonde gedragsfactoren (vooral overgewicht, roken en drinken), meer dan door het vaker voorkomen van ziekten in lage SES groepen.

Hoofdstuk 6 beschrijft de relatie tussen SES en het ontstaan van beperkingen in mobiliteit bij 70 tot 79 jaar oude mensen zonder beperkingen in mobiliteit bij aanvang van de studie (Health ABC studie). Daarnaast werd de rol van biomedische factoren in de verklaring van deze verschillen bepaald. Een lage SES was een sterke voorspeller voor het ontstaan van beperkingen in mobiliteit gedurende 4,5 jaar follow-up. Biomedische factoren, waaronder een breed scala aan ziekten en biologische risicofactoren, verklaarden een deel van dit verband. Gemiddeld verklaarden de biomedische factoren 41% van de opleidingsverschillen; dit was 17% voor inkomen en 29% voor het aantal financiële bezittingen (zoals spaarrekeningen, beleggingen en huisbezit) dat iemand had. Hoge waarden van ontstekingsmarkers, een hoge body mass index en hoge bloeddruk waren de belangrijkste factoren in het verklaren van de SES verschillen in het ontstaan van beperkingen in mobiliteit. Deze studie laat zien dat biomedische factoren belangrijk zijn in het mechanisme dat ten grondslag ligt aan de relatie tussen SES en fysiek functioneren. Echter in het terugdringen van lichamelijke beperkingen (en mogelijk invaliditeit) en vooral de SES verschillen daarin is het niet voldoende om te interveniëren op biologische risicofactoren en de kans op ziekten.

Hoofdstuk 7 laat zien dat in een gezonde oudere populatie (Health ABC studie), mensen met een lage SES ook een verhoogd risico hebben op een achteruitgang in cognitief functioneren in vergelijking met mensen met een hoge SES. Ondanks dat in deze studie een hogere prevalentie van ziekten en de risicofactoren geassocieerd is met zowel een lage SES als slecht cognitief functioneren, konden de biomedische factoren slechts 5% van de SES verschillen in een cognitieve achteruitgang verklaren. De hypothese dat biomedische factoren belangrijk zijn in het mechanisme dat ten grondslag ligt aan de relatie tussen SES en cognitief functioneren kon niet ondersteund worden.

In *Hoofdstuk 8* wordt onderzocht in hoeverre een lage SES het ontstaan van depressie bij oudere mensen voorspelt (LASA). De bijdrage van verschillende verklarende factoren, waaronder psychosociale factoren, ziekten en gedragsfactoren,

zal daarnaast gelijktijdig worden onderzocht in de verklaring van de SES verschillen. De resultaten laten zien dat een lage SES inderdaad het ontstaan van depressie gedurende 9 jaar follow-up voorspelt. Psychosociale factoren konden een deel van deze SES verschillen verklaren. Een klein sociaal netwerk, een lage eigen effectiviteit (geloof in eigen kunnen) en slecht fysiek functioneren hadden de grootste individuele bijdrage in het verkleinen van de SES verschillen in het ontstaan van depressie. SES verschillen in het ontstaan van depressie bij ouderen bleven echter voor een groot gedeelte onverklaard.

In *Hoofdstuk 9* worden de belangrijkste bevindingen samengevat. Verder worden er dit hoofdstuk conceptuele en methodologische beperkingen van de studies besproken. Ondanks een aantal beperkingen, kunnen we concluderen dat een lage SES sterk gerelateerd is aan slechter fysiek, cognitief en mentaal functioneren. Dit werd gevonden voor verschillende indicatoren van SES en deze gezondheidsverschillen blijven bestaan op oudere leeftijd. Biomedische factoren, gedragsfactoren en psychosociale factoren konden SES verschillen in functioneren slechts gedeeltelijk verklaren. Derhalve blijven SES verschillen in functioneren voor een deel onverklaard. Verder onderzoek is nodig om de onderliggende mechanismen verder te onderzoeken, gevolgd door interventies – ook op oudere leeftijd – om SES verschillen in gezondheid terug te dringen.

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Annemarie

Maastricht, oktober 2005

About the author

Annemarie Koster was born on April 5, 1978 in Helmond, the Netherlands. She spent her youth in Gemert, Zoetermeer, Aalst, and Doetinchem. In 1997 she finished her secondary education at the St. Ludgercollege in Doetinchem. That same year she started with the study Nutrition and Health at Wageningen University. She wrote her major master thesis in the field of Human Nutrition and an additional thesis in the field of Marketing and Marketing Research. After an internship at CSIRO (Commonwealth Scientific and Industrial Research Organisation) in Adelaide, Australia, she obtained her Master of Science degree in 2002. In 2003 she also obtained her Master of Science in Epidemiology degree and is registered with the Netherlands Epidemiological Society.

In December 2002 she started as a PhD student at Maastricht University, department of Health Care Studies, Medical Sociology section where she conducted the work described in this thesis. From July to December 2003 she conducted part of her research at the J. Paul Sticht Center on Aging, Wake Forest University in Winston Salem, North Carolina in the United States.

In the coming years Annemarie will remain affiliated with the University of Maastricht as a postdoc. Within this period, she will spend one year (2006) at the National Institute on Aging (NIA) in Bethesda, USA for which she will receive a fellowship from the American National Institutes of Health. During this fellowship, she will continue her aging research activities through collaborations with Dr. Tamara Harris (NIA) and Dr. Stephen Kritchevsky (Wake Forest University).

De auteur

Annemarie Koster werd op 5 april 1978 geboren in Helmond. Zij bracht haar jeugd door in Gemert, Zoetermeer, Aalst en Doetinchem. In 1997 behaalde zij haar VWO diploma aan het St. Ludgercollege in Doetinchem. In datzelfde jaar begon zij met haar studie Voeding en Gezondheid aan de Wageningen Universiteit. Doctoraalonderzoeken werden gedaan bij de vakgroep Humane Voeding en bij de vakgroep Marktkunde en Marktonderzoek Na een afsluitende stage bij CSIRO (Commonwealth Scientific and Industrial Research Organisation) in Adelaide, Australië studeerde zij in 2002 af. Sinds 2003 staat zij geregistreerd als Epidemioloog A bij de Nederlandse Vereniging voor Epidemiologie.

In december 2002 begon zij als assistent in opleiding bij de sectie Medische Sociologie, capaciteitsgroep Zorgwetenschappen van de Universiteit Maastricht waar zij het onderzoek deed zoals in dit proefschrift beschreven staat. Van juli tot december 2003 deed zij een deel van haar onderzoek in Amerika bij het J. Paul Sticht Center on Aging, Wake Forest University in Winston Salem, North Carolina in Amerika.

De komende jaren zal Annemarie verbonden blijven aan de Universiteit Maastricht als postdoc. In deze periode zal zij een jaar (2006) doorbrengen bij het National Institute on Aging (NIA) in Bethesda, Amerika waarvoor zij een beurs van het American National Institutes of Health zal ontvangen. Gedurende deze tijd zal zij haar werk op het gebied van verouderingsonderzoek voortzetten en daarbij samenwerken met Dr. Tamara Harris (NIA) en Dr. Stephen Kritchevsky (Wake Forest University).