Social determinants and Cardiovascular Disease: **Analyses from the English Longitudinal Study of Ageing**

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Abstract

The study of the social determinants of health is a relatively new area of scientific enquiry. A plethora of different conceptualisations and measurements of the key social characteristics of people's lives have been developed and deployed in surveys. Both cross-sectional and, more interestingly, longitudinal studies, have yielded evidence of a link between various aspects of 'social engagement' (an umbrella term used here to denote one or more aspects of the social existence of study participants) and mortality/morbidity.

Earlier studies relied in most cases on self-reported health (and some still do); gradually, more objective measurements of health were included in prospective studies; and more recently a range of biomarkers have been incorporated into many longitudinal population studies. However, despite the plentiful evidence linking social engagement with health, the exact mechanisms or pathways that link social engagement to health are poorly understood. Using data from the English Longitudinal Study on Ageing, this paper has the aims to examine cardiovascular biomarkers that are associated with objective and subjective measurements of social engagement. This paper will contribute to the understanding of the mechanisms or pathways that associate social engagement to morbidity and mortality. Addressing this issue, it is hoped, will be helpful to scholars working at the intersection of the social and biomedical sciences and seeking to understand the complex connections between social relationships, human biology and health.

I. Introduction

The protective influence of social relationships on health is widely recognized in gerontological and social epidemiology research (Berkman and Kawachi 2000). Over the last two decades, findings from both clinical/experimental studies and population studies (e.g. Alameda County, Whitehall, Eastern Finland, Pittsburgh Common Cold) have been remarkably consistent in demonstrating a beneficial effect of social relations on various health outcomes, including overall mortality (Berkman and Syme, 1979; Berkman et al., 1992; Blazer, 1982; Brummett et al., 2001; House et al., 1982; Kaplan et al., 1988; Orth-Gomer and Johnson, 1987; Schoenback et al., 1986; Seeman et al., 1993; Welin et al., 1985), physical and psychiatric morbidity (Berkman et al., 2000; Cohen, 1988; House et al., 1988; Seeman 1996; Uchino, 2004), activities of daily living (Green et al, 2008; Park and Lee, 2007; Kondo et al. 2007) and cognitive decline (Bennett et al., 2006).

Socially integrated persons have been shown to be less likely to have heart attacks (Kaplan et al. 1988), less likely to develop upper respiratory illness when experimentally exposed to a common cold virus (Cohen et al. 1997), and more likely to survive breast cancer (Funch and Marshall 1983). They are also less likely to suffer from cognitive decline, even in the presence of Alzheimer's disease pathology (as seen in post-mortem brain biopsies) (Bennett et al. 2006). Conversely, the health risks associated with low levels of social integration have been argued to be comparable to those linked to smoking, high blood pressure and drinking (House et al. 1988; Cohen et al. 2000).

Despite the plentiful evidence linking social engagement with health, the exact mechanisms or pathways that link social engagement to health are poorly understood. In 1979, Berkman and Syme stated that "adequate tests of the hypothesis that social circumstances alter general susceptibility of disease in humans will not be possible… until data are available on physiologic mechanisms capable of mediating the relationship between social events and disease outcomes" (Berkman and Syme, 1979). Thirty years after Berkman

and Syme's seminal study little is known about mechanisms by which social engagement influences health outcomes.

Using the English Longitudinal Study on Ageing (ELSA), the aim of this paper is to examine cardiovascular biomarkers that are associated with objective and subjective measurements of social engagement. Addressing this issue, it is hoped, will be helpful to scholars working at the intersection of the social and biomedical sciences and seeking to understand the complex connections between social relationships, human biology and health.

II. Pathways Linking Social Engagement to Health Outcomes

Gerontological and social epidemiology research has identified three domains of social engagement that are associated with health outcomes, namely social networks, social support and social integration (Cohen 1988, 2004; House, 1988). These domains are not always correlated and each of them may influence health through different, but not necessarly independent, pathways (Cohen 2004; Uchino 2006). For the purpose of this paper, I use the term 'social engagement' as an umbrella term that can be used to refer to a combination of objective measurements of the salient aspects of people's 'social' existence, typically incorporating their social network (the 'structure' within which social interaction takes place), the (different types of) social support available to them (functional measure), and their level of social integration into the networks that are available to them (the 'content' of the structure). I will also use the term 'loneliness' as a subjective measurement of social isolation.

There are two primary mechanisms that link social engagement to health outcomes, namely *psychological* and *behavioural* processes which in turn affect physiological pathways (these pathways and mechanisms contained in each pathway are summarised in Figure 1). Of these proposed pathways, the best understood are those that operate via mechanisms that influence health behaviours, lifestyle and access to health and social care. According to the social control hypothesis, 'people obtain normative guidance by comparing

their attitudes with those of a reference group of similar others' (Marsden and Friedkin 1994, p.5). Normative pressure and example from friends, peers, and family influence the decisions to adopt or maintain (un)healthy behaviours (e.g. a spouse who insists on eating well, drinking less, ceasing smoking and so on or, conversely, peer pressure to start smoking among adolescents). Moreover, fewer social ties provide fewer sources of information and by extension more limited access to appropriate care and less scope to minimize stressful events. Christakis (2008), examining the structure of social networks and spread of health behaviours among Framingham heart study participants, showed that changes in norms (growing cultural opposition to cigarettes among study participants) caused changes in preferences and values and consequently in behaviour, resulting in network members ceasing to smoke.

The two main psychological processes through which social engagement influences health are the stress buffering effect and the promotion of positive psychological states. Social engagement can attenuate or eliminate stressful experiences by providing a solution to the problem, or by giving a new interpretation for adverse events (Cohen 2004), thereby buffering the harmful effects of stress. For example, social engagement can modulate cardiovascular reactivity (Cohen 2004) by reducing sympathetic nervous system activity and/or stress related hormonal activity (via the hypothalamic-pituitary-adrenal cortical axis). Social integration can also influence one's emotional state, giving a sense of purpose, meaning and belonging and reducing the intensity and duration of negative affective states (Thoits, 1982), thus resulting in suppressed neuroendocrine response (Cohen 1988, 2004).

III. Biomarkers: can they shed light on the link between social engagement and health?

Social engagement influences health by affecting behavioral responses (in relation to, for instance, smoking, drinking alcohol, drug use) and psychological processes, as outlined above. However, the precise physiological processes whereby social engagement, through these behavioral and psychological processes, is translated into good or poor health

(understood as morbidity and mortality) are not fully understood: 'The research task is to *give an account* of what links social structure to health outcomes – to ask, *what are the intermediary steps*?' (Marmot, 2001,p.35). The collection of physical measurements and biomarkers is intended to be of assistance in the attempt to unpick these 'intermediary steps'. The third column in Figure 1 lists some of the biomarkers that are believed to play a role in the impact of social engagement on health (Seeman & McEwen 1996; Uchino, Cacioppo and Kiecolt-Glaser, 1996; Uchino, 2006).

There are three physiological systems that link social engagement to health outcomes, namely *inflammatory*, *neuroendocrine* and *cardiovascular*. *neuroendocrine*. The inflammatory system is the body defense response to infections and other toxic stimuli. The neuroendocrine system is a network of nerve flares and hormones in the brain and the rest of the body. The cardiovascular system predominantly reflects the heart, blood vessels and blood pressure. In this paper, I will focus on the cardiovascular biomarkers to examine how social factors are associated with these markers.

Cardiovascular biomarkers

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality across the developed countries (Mathers & Loncar, 2006). The risk of vascular events increases with age and as a consequence cardiovascular disease is of particular importance in ageing research (Kannel, McGee and Gordon, 1976).

There is plentiful evidence that social factors influence the *cardiovascular* system and through that, the onset of, recovery from and survival after coronary heart disease (Orth-Gomer et al. 1993; Glass et al. 1993; Colantonio et al. 1993). These findings suggest that social engagement may be beneficial because it buffers the potentially harmful influences of stress-induced cardiovascular reactivity (usually measured by heart rate, and systolic and diastolic blood pressure) and consequently reduces the incidence of cardiovascular disease (Uchino 2006). However, the studies that have yielded evidence of the impact of social engagement, including functional and

structural support, are not based on community samples, but are typically clinical studies (see Uchino, Cacciopo and Kiecolt-Glaser 1996).

In terms of the risk of development of CHD, in one population study, National Heart, Lung, and Blood Institute (NHLBI) Family Heart Study, women with high risk for the development of coronary heart disease but high social support were found to develop less carotid artery atherosclerosis (Knox SS et al., 2000) Other population based study has also shown that people who are not socially isolated or/and have social support survive longer after occurrence of CHD; however, the biological mechanisms is still poorly understood. Does social component influence on development of risk factors, health behaviours, disease progression, or survival after an event? What exactly goes in the black box? What is about knowing people, talking to them, perceiving them to be there for you that have a positive impact on one's health (or the absence of these that has the negative impact).

Using the data from the English Longitudinal Study of Ageing, this paper will contribute to the understanding of the biological mechanisms or pathways that link social engagement to cardiovascular disease at population level.

III. Data:

The English Longitudinal Survey of Aging (ELSA) is a national panel study and contains approximately 10,000 respondents providing a representative sample of the English population aged 50 years and older. The study contains detailed information on health, socioeconomic factors, pension arrangements and housing wealth. Overall, 10,770 participants interviewed at wave 1 (2002). Of these 8,688 (80.7%) participated in wave 2 (2004-5) (excluded proxy interview). The second wave introduced a nurse visit as well as an interview. The nurse visit was carried out in the respondent's home and involved a series of measurements such as blood pressure, grip strength, blood sample, standing height, sitting height, weight, waist and hip measurement, lung function, balance, leg raise and chair rises. For the purpose of this paper, I will use the second wave of the English Longitudinal

Study of Ageing (ELSA). The mean age of participants was 66.8 years and 55.1% were women.

Table 1: Biomarker Ascertained in ELSA

Sample	Start Year	Biomarkers Ascertained
Nationally representative survey of 12,000 people aged 50 and over	2002	**Fasting Blood Sample**:total cholesterol, HDL cholesterol, fibrinogen, C-reactive protein, ferritin, glycated haemoglobin and haemoglobin. Saliva sample: 24 hrs salivary cortisol and DNA for a genetic repository **Physical Examination**: 3 readings sitting down blood pressure, waist/hip ratio, weight and height and peak flow rate.

IV. Methods:

Statistical analysis included descriptive, bivariate, and multivariate analysis.

Descriptive Statistics

Table 2: Characteristics of participants for wave 2 of ELSA (excluding proxies). Values are numbers (percentage) unless stated otherwise

	Men	Women
Gender	46.21	53.79
Age group		
50-54	10.5	8.9
55-59	23.0	20.3
60-64	16.7	15.7
65-69	16.0	14.4
70-74	12.9	12.6
75-79	10.5	11.8
80+	10.3	16.3
Marital status		
single (never married/ divorced/separated/widowed)	25.4	42.6
married	74.6	57.4
Years of full-time education		
0-9		
10-13		
14+		
missing		
Social Integration		
Yes	84.89	83.77
No	15.11	16.23
Close relationship		
Yes	88.05	88.05
No	11.95	10.83
Loneliness		

Yes	11.51	12.16
No	88.49	87.84
Depression (CES-D)		
no depression	76.8	67.02
depression	23.2	32.98
Cigarrete Smoking		
Non-smokers		
Ex-smokers		
Current -Smokers		
Diagnosed disease		
CVD related disease*	33.2	28
Other chronic disease**	44.9	53.2
BMI		
<18.5	0.6	1.2
18.5 to 24.9	23.7	29.6
25 to 29.9	48.8	38.3
>30	27.2	31.0
Waist and hip ratio		
mean	0.96	0.85
raised WHR (%)	53.4	45.7
Systolic and diastolic blood pressure		
Mean systolic BP (mmHg)	135.9	135.1
Mean diastolic BP (mmHg)	75.8	74.1
Hypertension (%)	56.0	54.6
Dyslipidemia		
Cholesterol		
Mean total cholesterol	5.6	6.1
Percentage ≥ 5.0 mmol/l chol	69.6	84.0
HDL		
Mean total HDL cholesterol	1.4	1.6
Percentage <1.0 mmol/ l HDL chol	7.3	1.5
LDL		
Mean total LDL cholesterol	3.5	3.8
Percentage ≥3.0 mmol/ l LDL chol	72	80.6
Triglycerides		
Mean triglycerides	1.6	1.5
Percentage ≥1.6 mmol/ l LDL chol	51.3	42.8
HbA1c		
Mean HbA1c level (%)	1.7	2.1
Fibrinogen		
Mean Fibrinogen (g/l)	3.2	3.3

*angina, myocardial infection, stroke, heart failure, heart murmur, diabetes
**chronic lung diasese, arthritis, osteoporosis, cancer, parkinson's disease

Multivariate Analysis

Since it is unclear what precise biomarker levels (SBP, DBP, HDL, total cholesterol, HBA1c, and abdominal obesity (WHR)) correspond to varying levels of risk, I will define risk above or below distribution percentiles (10^{th}, 25^{th}, 75^{th}, 90^{th})

Using different cut-off points, linear and ordered logit regression analyses will be performed, to test whether social engagement is associated with these single biomarkers or combination of these biomarkers.

Cardiovascular variables

Control variables: information on history of cardiovascular disease and risk factors for CVD

- Hypertension
- Dyslipidemia
- Smoking status
- Diabetes
- Medication use (including medication for CVD)
- Physical activity

Cardiovascular biomarkers:

- BMI
- Waist/hip ratio (valid mean of two measures)
- Blood pressure
- HDL
- LDL
- HbA1c
- FIbrinogen
- Triglycerides

The Independent variables

Using data from English Longitudinal Study on Ageing (ELSA), I will create a **social engagement index** which consists of:

1) **social network**:
    - **Size:** The relationships that ELSA explores are relationships with (1) a husband, wife or partner the respondent lives with, (2)

children, (3) immediate family and (4) friends. All respondents are asked whether they have any of the above relationships.

- **Frequency of contact:** In order to look at the frequency of contact, respondents are further asked how often they meet up or speak on the phone with their children outside of the household, immediate family or friends. Frequency of contact with household members is not investigated.
- **Density**; In order to look at the density of a respondent's relationship, ELSA asks how close their relationship with their partner is and how many children, immediate family members and friends they have a close relationship with.
- **Quality of a relationships**

• How much do they really understand the way you feel about things?
• How much can you rely on them if you have a serious problem?
• How much can you open up to them if you need to talk about your worries?
• How much do they criticize you?
• How much do they let you down when you are counting on them?
• How much do they get on your nerves?

2) social participation:

Respondents were asked about their participation in a number of activities, including:

• going to the cinema
• visiting an art gallery or muse
• going to the theatre, concert or opera
• eating out of the house
• whether they had bee non a daytrip or holiday (in the UK or abroad) in the last year

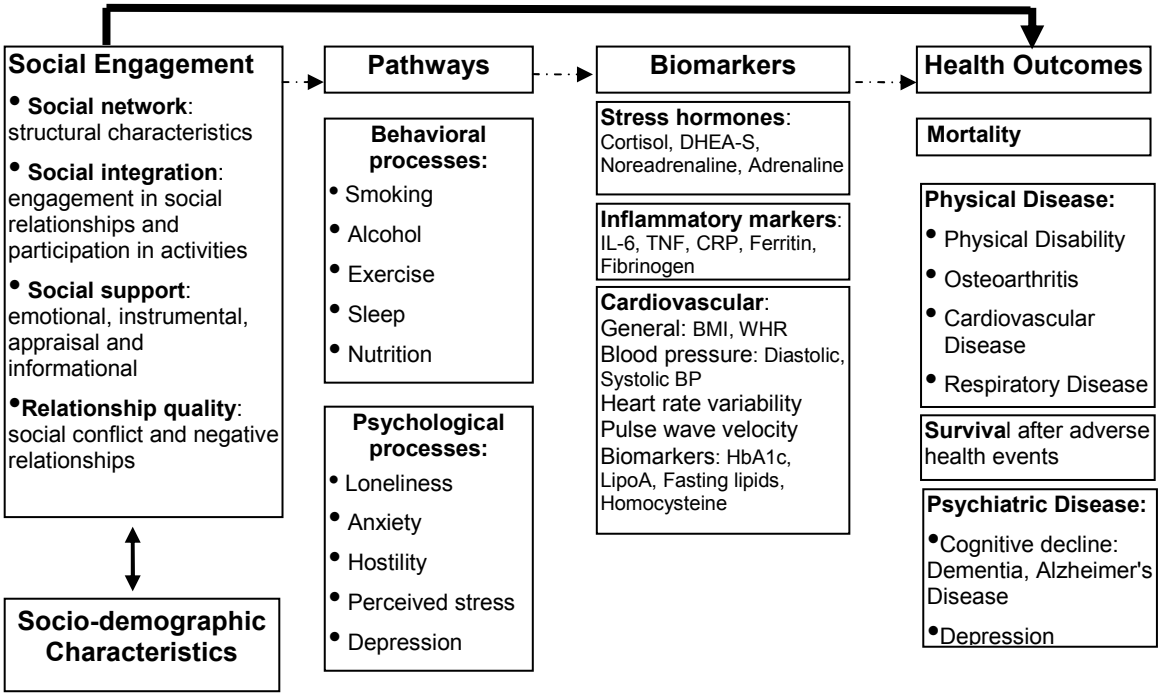
This limited list of activities is quite specific and might be associated with particular groups of society, such as white, middle class older people.

3) social integration:

Respondents were asked whether they were a member of political, trade union or environmental groups; tenants groups resident groups or neighbourhood watch; church or other religious organization; and charitable associations.

Other control variables: age, sex, marital status, race/ethnicity, depression (CES-D scale), income, and education

Figure 1: Understanding the pathways: Biomarkers, Physical Assessments



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