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Geographical and Social Inequalities in Health and Health Behaviour in the Nord-Trøndelag Health Study (HUNT)

Thesis for the degree of Philosophiae Doctor

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Preface

This dissertation comprises most of the results from my work at the Department of Geography at the Norwegian University of Science and technology from 2005 to 2009. The Faculty of Social Sciences and Technology Management funded the work.

There are numerous people I wish to thank. First and foremost my supervisor Stig H. Jørgensen who was my mentor through my master thesis and now through the course of this doctoral degree project. He has been instrumental in developing my thinking through numerous discussions and I am deeply indebted for his supervision, support and collaboration. I have really enjoyed the companionship through many journeys to conferences and I sincerely hope we can continue to do some form of collaboration also in the future.

My co-supervisor has been Steinar Krokstad at the HUNT research centre and both he and Steinar Westin at the Department of Public Health and General Practice have been very supportive and helpful through several years. I have really appreciated their company through many meetings and journeys in this period. I am also grateful to them for introducing me to so many nice people who share my 'view of the world'. In the same vein I would like to thank Roar Johnsen and Johan Håkon Bjørngaard at the Department of Public Health and General Practice for their help and support.

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My co-author on two of the papers, Andy Jones from the University of East Anglia, has thought me a lot through these years and a sincere thank you goes to him as well. Kristen Ringdal at the Department of Sociology and Political Science have been a very helpful source to consult on statistics and modelling issues and also provided insightful and constructive comments on my final seminar before submitting my thesis. I would also like to say thank you to the sociologists Vera Skalická and Terje Andreas Eikemo. The data recoding wizard Kyrre Svarva at the faculty's data services has been, as always, very helpful and a warm thanks goes to him as well.

There are also many people that I have met in various networks during these years that should be mentioned, but I will thank them on a later occasion and just refer to the various networks.

The ‘social epidemiology network’ at the HUNT research centre, the ‘Norwegian-Danish network on social insurance and social medicine research’ and the annual meetings in ‘social medicine thinking’ have all been very rewarding and stimulating arenas to participate in. Similarly, the participation in the research group in ‘urban-, rural and regional studies’ at the Department of Geography have given me valuable feedback and also kept me updated on other people’s research.

I have been employed in Helse Nord since august 2009, and I am deeply indebted to my current boss Finn Henry Hansen for his patience and understanding during the finishing phase of my doctoral project.

Finally, I will also like to thank family and friends. My cousin Geir have always been supportive and helpful. My younger brother who has been on the same doctoral degree journey has provided psychosocial support (and hopefully received some in return) during the ups and downs. I guess we have both learned that a PhD-project is not always a picnic, but in the end, the occasional rewards grossly outweigh the setbacks.

Trondheim, May 2010

Erik R. Sund

Written in the spirit and memory of my dear mommy

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Summary of dissertation

Health and health behaviour varies both socially and geographically and individuals may experience different degrees of health according to their socioeconomic position and where they live. The fact that health varies geographically is usually given two interpretations. It may arise as a consequence of the composition of individuals according to sociodemographic markers. Alternatively, there may be features associated with the context in which they live that explains geographical health variation. Consequently, individuals' health may be influenced by both individual factors and contextual factors. The overall aim of this thesis is to analyse whether geographical health variation is due to composition or features associated with context.

Data from the Nord-Trøndelag Health Study (HUNT) in the county of Nord-Trøndelag, Norway, and the statistical technique of multilevel modelling were utilised to analyse these relationships at multiple geographical scales and also across non-geographical contexts.

The overall finding is that geographical health variation in Nord-Trøndelag is rather small and that place makes little difference to the health of individuals. This applies both to the level of municipalities and wards/neighbourhoods. The importance of the family context was also explored, and it was found that health and health behaviour within families seemed to cluster. There was strong behavioural conformity in terms of smoking habits whereas body mass index was weakly to moderately dependent on the family context.

The findings have some clear implications in terms of future disease prevention and health policy. First, targeted area based initiatives towards particular municipalities, or wards, is not warranted in this particular county. It is however difficult to generalise this particular finding across cultures and towards more urban areas, there may be societies where such initiatives may be of importance. Second, there are some clear indications that the family context is important for the health of individuals and this finding should be acknowledged in future research as well as in disease prevention and health policy.

1. Introduction

Globally, life chances differ greatly depending on where people are born and raised (Curtis, 2004; Marmot, Friel, Bell, Houweling, & Taylor, 2008). Grim poverty, poor living conditions, malnutrition and infectious diseases probably accounts for a substantial part of these differences. Also within countries, the differences are dramatic and they are seen worldwide (Boyle, Curtis, Graham, & Moore, 2004; CSDH, 2008). What is more surprising is that even in affluent societies with a so-called egalitarian ethos, like Norway and Sweden, there are stark differences in health and longevity (Mackenbach, 2005). These differences apply both to socioeconomic position and place of residence. This thesis concerns both these types of social inequality; socioeconomic and geographical, but is primarily about the latter.

Given that the average health of places varies, does this mean that places matter for health? This question has received systematic attention both within modern epidemiology, public health, health sociology and health geography and perspectives from all these disciplines appear in the magpie discipline of social epidemiology. If place truly makes a difference to health, we should expect health outcomes to depend not only on individual characteristics like age, gender, social class etc, but also on place, setting, "ecology", context or on the surrounding environment in which individuals live and work. This introduces the other crucial aspect in this thesis. Besides explaining geographical variation in health; are there *contextual* determinants affecting the health of individuals' "over and beyond" individual level factors?

Geographical health variation and contextual influences are closely related and for conceptual purposes it is useful to make a (preliminary) distinction between *compositional* and *contextual* effects. The first refers to the idea that geographical health variation is an artefact of differential socioeconomic composition of individuals, whilst the second implies that there are independent place characteristics (context) inflicting individual health directly. In other words, there may be causes to disease, or ill health, at the level of society that is not reducible to the individual.

This last important aspect will, however, remain undiscovered in traditional risk factor epidemiology seeking to identify risk in individuals. Consider the following illustrative example (Schwartz, Diez Roux, & Susser, 2006 p. 449): "Suppose two societies have disparate rates of alcoholism primarily because they have different laws regulating the sale of alcohol. Although this social fact causes the rates of alcoholism to differ between the societies, it will not explain any of the interindividual differences within either society. Thus, the causes of between-group variation can be quite different from the causes of within-group variation". So while a traditional risk factor epidemiological study conducted *within* any of these societies could have identified numerous individual risk factors for alcoholism, they would indeed miss the truly important cause. They would merely provide "the right answer for the wrong question" (Schwartz & Carpenter, 1999).

The running theme throughout this thesis is, firstly, to investigate to what extent geographical variation in health is due to composition or context. Secondly, and partly contingent on the

first issue, examine to what extent causes located at higher levels of organisation, both in a relational as well as in geographical space, may independently influence the health of individuals. The keyword is context.

2. Structure of the thesis

The thesis consists of a superstructure and a collection of four articles. The superstructure is organised in the following manner. In the next section (section three), a brief historical overview of health inequality research is given along with the main tenets of some seminal writings from the UK and Norway up until the present time. This section also incorporates a short note on the concepts and principles of health equity. The theoretical framework is outlined in section four, starting with the influential concepts of 'population perspective' and 'social facts' from the epidemiologist Rose and the sociologist Durkheim. This is followed by 'structuration theory' which is linked to a general model of public health determinants known as the Dahlgren & Whitehead model. Finally, a description of various theories of neighbourhood effects and a short note on family effects is given.

Section five, which is the methods' section, starts with an initial discussion on various methodological limitations of previous research aiming at investigating individual as well as contextual influences on individual health. Thereafter, the statistical technique of multilevel modelling is introduced and outlined by both graphs and equations. This part also seeks to illustrate how the statistical procedures are integrated with the theoretical and conceptual frameworks. This is followed by a critical summary of pros and cons associated with this particular method. Section six takes the discussion of contextual influences on individual health to a more fundamental level and tries to highlight various challenges facing this line of inquiry. The study area and data material used in the thesis is described in section seven (and also in the attached appendix 2). Section eight summarises the four articles, of which the first one is more properly speaking an introduction to a theoretical book section (article 1) and not a summary. Finally in section nine the main findings from the thesis are discussed. The collection of four articles is reproduced after the list of references.

3. Background

Resurgent interest in health inequalities

Health inequalities are by no means a new phenomenon. In the 19th century, important figures in public health like Villermé in France, Chadwick in England and Virchow in Germany devoted a large part of their scientific work to this issue. Similarly, in Norway, the vicar and founding father of Norwegian sociology, Eilert Sundt, conducted research on the associations between mortality and living conditions (Sundt, 1855).

Restricting the time frame to the last four decades, some particular seminal writings have been published in the UK. In 1977 the Secretary of State for Health in the Labour government set up a working group on health inequalities led by Sir Douglas Black. Its threefold mandate was to review evidence about social class health inequalities, to consider possible causes, and point towards possible policy implications and further suggestions for research. The report documented wide-ranging inequalities in health. Four explanations were considered in the Black report (DHSS, 1980); artefact explanations (problems of measurement), theories of natural and social selection (sick people becomes poor), behavioural explanations (poor people have poor health habits) and material/structural factors (adverse life circumstances make people more vulnerable to disease). The commission landed firmly in favour of the material/structural explanation (Macintyre, 1997).

Some two decades later, again initiated by a labour government, Sir Donald Acheson chaired The Independent Inquiry into Inequalities in Health and his group basically took the material/structural view of the Black report (Acheson, 1998). Perhaps inspired by Virchow's famous statement that "medicine is a social science, and politics nothing but medicine at a larger scale" (Mackenbach, 2009), they claimed health inequalities to be rooted in society. Their policy advice reflect this, as only 3 of 39 recommendations were concerned with medical care (Marmot, 2004).

The Black report and the Acheson inquiry were instrumental in several respects; firstly, by highlighting health inequalities as a scientific and policy issue, secondly, by giving focus to the problem, and thirdly, in providing appropriate frameworks (Siegrist & Marmot, 2006). Crucially important is also the description of the *shape* of these health inequalities. Health inequalities between social groups in society are not confined to differences between rich and poor. Rather, health follows a social gradient; the higher the position in the social hierarchy, the lower the risk of ill health. Socioeconomic health inequalities are hence likely to be graded – shades of grey, rather than black and white.

More recently, a third labour government initiated review was published (SRHIE, 2010). This 'Marmot review', chaired by one of the Acheson members, Sir Michael Marmot, gave the following key messages; the reduction of health inequalities is a matter of fairness and social justice. The social gradient in health is the result of social inequalities, and reducing the steepness of the social gradient in health requires universal solutions. The scale and intensity of these solutions must be proportionate to the level of disadvantage; the authors called these solutions 'proportionate universalism'. Besides matters of fairness and justice, the reduction of health inequalities will have economic benefits for the society by reducing productivity losses, increasing revenues and by reducing welfare payments and treatment costs. After reviewing the evidence and suggesting policy measures, the authors state that *inaction* cannot continue because the human and economic costs are too high.

During the same period in Norway, research initiatives and descriptions of health inequalities were more scattered and uncoordinated, at least in the early part of the period. With some exceptions, public health monitoring was more concerned with describing the average health status of the population rather than the dispersion around these averages (Westin, 1994). A

report documenting large geographical disparities in mortality within the capital of Oslo did get some attention (Rognerud & Stensvold, 1998), but it was a cross-national comparative study published in The Lancet that really attracted attention (Mackenbach, Kunst, Cavelaars, Groenhof, & Geurts, 1997). The study, confined to European countries, documented that the largest relative inequalities in mortality were to be found in Norway and Sweden, thereby implicitly challenging the Scandinavian social welfare state model. The article was immediately criticised on methodological grounds because of their usage of relative measures of inequality (Vågerö & Erikson, 1997).

The article by Mackenbach and colleagues nevertheless served the important purpose as an eye-opener and contributed to awareness-raising around social health inequalities. In the aftermath of this Lancet article there have been several notable policy developments which are comprehensively described elsewhere (Dahl & Lie, 2009). Put shortly, the policy efforts have been so welcomed, that Dahl & Lie (2009), with reference to Whitehead's so-called "action spectrum" (Whitehead, 1998), recently proclaimed Norway as a pioneer in their health policy efforts in this field. To put this characterization into context, this is done only seven years after Dahl was describing Norway as a laggard (Dahl, 2002), hence there have been quite rapid developments within a short period of time. Action is certainly warranted; a recent article in the British Medical Journal documented noticeable widening in educational inequalities in mortality across the decades 1960 to 2000 for middle aged individuals (Strand, Grøholt, Steingrimsdottir, Blakely, Graff-Iversen, & Næss, 2010).

The fairly recently issued Norwegian "National strategy to reduce health inequalities" (MHCS, 2007) is characterized by three distinct features; it clearly acknowledges the gradient, it emphasizes universal solutions and has a long-term and comprehensive commitment. In addition, while clearly emphasising that health is influenced by peoples' everyday lives, and thereby adapting a "whole system perspective" (Curtis, 2004), the strategy clearly recognises geographical perspectives as being of crucial importance.

Concepts and principles of equity in health

Language is important, particularly so in disciplines communicating to policy makers on core human values. This section is an attempt to briefly outline some important concepts which inheres in this thesis. While such a topic may seem of primarily academic interest, it should become evident that language is more than mere words. Consider the term already used; health inequality. Health inequality could easily be termed as either "health variation" or "health inequity". The former is neutral and could in principle encompass all of epidemiology; the science of the distribution of diseases and risk factors across populations. The latter on the other hand, quite clearly draws our attention to unjust or unfair distributed health determinants and is implicitly making ethical judgements on distributive justice and core human rights. By using the terms inequality or inequity in our description of health variation, we implicitly put forward causal assumptions (Starfield, 2007) – as well as point towards how they should be dealt with (Braveman & Gruskin, 2003). Importantly, but perhaps less evident, language also have crucial bearings on how we measure and monitor

health inequality (Harper, Lynch, Meersman, Breen, Davis, & Reichman, 2008; Messer, 2008). Hence, even our measurements of health inequalities contain implicit value judgements (Harper, King, Meersman, Reichman, Breen, & Lynch, 2010).

The most widely cited definition of health inequity is articulated by Margaret Whitehead: "...differences which are unnecessary and avoidable but, in addition, are also considered unfair and unjust" (Whitehead, 1990 p. 5). This definition (essentially put forward to raise awareness and stimulate debate in a wide general audience in Europe) does not include all differences in health, but highlights the subset of differences that are avoidable, unfair, and unjust. These core terms do however lend themselves to widely varying interpretations, and this ambiguity may be problematic.

A number of definitions have been rigorously reviewed elsewhere (Braveman, 2006), and a relevant definition for this thesis is given by the International Society for Equity in Health (ISEqH, 2005) who describes equity in health as:"*The absence of systematic and potentially remediable differences in one or more aspects of health across populations or population subgroups defined socially, economically, demographically or geographically*". Given this definition, the mandate for research on health equity would then be to (ISEqH, 2005): "elucidate the genesis and characteristics of inequity in health for the purpose of identifying factors amenable to policy decisions and programmatic actions to reduce or eliminate inequities".

It is important to note, that not all inequalities in health are unjust or inequitable. If good health is unattainable, then this would be unfortunate, not unjust. If, on the other hand, inequalities in health are avoidable, yet are not avoided, they are inequitable (Marmot, 2007).
A vivid description well worth quoting, although stated in another context, was given by Sir Geoffrey Rose (Rose, 1992 p. 4): "It is better to be healthy than ill or dead". This was his humanitarian argument for disease prevention which became manifest in the so-called "population perspective" in preventive medicine described in the next section.

4. Theoretical framework

Population perspective and "social facts"

Consider the following rhetorical chain of thoughts: Why do individuals get lung cancer? The most likely answer is that the individual smoked for the last three or four decades. Next consider the highly unlikely situation where the aetiology behind lung cancer was unknown and the entire society smoked. Some individuals would eventually decease whilst others would not. Would you in this last scenario find the "true" cause for lung cancer?

This introductory illustration, although superficial and simplistic, is an attempt to stimulate thinking about causes, as well as the relationship between 'wholes and parts', and it touches upon classical philosophical questions in human sciences in general. In the social sciences, and to a less extent in epidemiology, there is a tension between those who believe that we

should study individuals to understand society (methodological individualists), and those who believe that we should study whole societies (methodological collectivists).

In the social sciences and in epidemiology, two proponents for a collectivist approach are Durkheim and Geoffrey Rose respectively. Their core assertion is that the lives of individuals are affected not only by their individual characteristics, but also by features of the social groups to which they belong (Durkheim, 1964; Rose, 1985). The underlying idea is that group properties, or social context, may also be viewed as distinct from those of individual members. These contexts may affect outcomes independent of individual characteristics or they may modify how individual characteristics are related to outcomes. Consequently, in order to understand human behaviour and outcomes, both levels of organisation; individual *and* society, should be analysed simultaneously.

Durkheim's investigation of the extremely individual act of suicide is illustrative (Durkheim, 1951). He began with the observation that suicide rates *within* European societies were reasonable stable over time whereas there was a fourfold difference in suicide rates *between* these societies. Although acknowledging that the causes of suicide was to be found in the specific life histories in individuals, there was a patterned regularity in suicide rates among populations over time – even as individuals moved in and out of these societies. His suggestion was that 'something' in the social environment promoted a distinct suicide rate for a particular society. This environmental influence, while unable to determine which individuals commit suicide, could hence explain societal differences in suicide rates over time.

Durkheim's study of suicide was a demonstration of a core concept in his intellectual reasoning: social facts. While starting with the proposition that "society is not a mere sum of individuals", (Durkheim, 1964 p. 103) Durkheim argued that society represents a system which has its own reality and its own characteristics. These characteristics subsequently shape the actions and outcomes of their constituent parts, the individuals. A social fact is hence, "every way of acting, fixed or not, capable of exercising on the individual an external constraint, or again, every way of acting which is general throughout a given society, while at the same time existing in its own right independent of its individual manifestation" (Durkheim, 1964 p. 13). So while 'social facts' are manifest in the behaviour of individuals (ways of acting), they are also distinct from those behaviours and can be usefully examined and manipulated at the societal level – i.e. outside of the level of the individual (Schwartz & Diez-Roux, 2001).

Durkheim's intellectual legacy, with its main tenets briefly reviewed here, was later picked up by the epidemiologist Geoffrey Rose. For Rose, Durkheim's 'social facts' provided a framework for understanding one of his core contentions, namely that; the 'causes of cases of disease' (why a particular individual get a disease) and the 'causes of disease incidence' (why a population have a particular rate of disease whereas others do not) may be different. In order to understand these claims, two concepts are essential; 'cause' and 'wholes and parts'.

In what could loosely be described as mainstream individual level risk-factor epidemiology, the most important causes offered primacy, are those who define the pathophysiology of a

disease. These are the causes that come closest to Koch's postulate of being specific and universally present among the diseased individuals. They are prioritised due to both greater scientific certainty and also their universality with which causal attributions can be made.

Rose on the other hand, used other criteria to develop his hierarchy of causes. Rose favoured the distal causes which were defined at the population level rather than at the individual level. These causes, or exposures, affects the entire population and cannot be detected in traditional individual-level studies conducted within a population. According to Rose (1985 p. 34): "In those circumstances all that these traditional methods do is to find markers of individual susceptibility". To reiterate the introductory lung cancer example: in a population where everyone smoked, happily ignorant of the consequences, an individual-level study would merely provide markers of individual susceptibility i.e. a genetic explanation for lung cancer.

A different hierarchy of causes, does not however, fully explain Rose's important distinction between causes of cases and causes of incidence. It might still be argued that the causes of incidence simply reflect the distal causes of 'causes of cases'. And if so, the causes of incidence would simply be the summation of causes of cases, they would not be distinct. It is at this point Rose invokes a Durkheimian view and gets ontological guidance on the relationship between 'wholes and parts' (e.g. structure and agency). Rose contends, as did Durkheim, that a population has characteristics that are distinct from the simple summation of the characteristics of individuals. The characteristics of the population are shaped by the individuals and at the same time the characteristics and behaviours of the individuals are shaped by the characteristics by the population.

When examining individuals within a society or population one holds the 'social facts' or the characteristics of the population constant, and hence 'social facts' cannot be identified. But social facts, or population characteristics, can indeed interact with individual characteristics and create varied effects. One of the main controversies surrounding the 'income inequality hypothesis' is illustrative: Is it more health damaging to be poor in a highly unequal versus an egalitarian society? Social facts, or population level determinants, may hence have different influence across groups – varying steepness in socioeconomic health gradients across places (Shouls, Congdon, & Curtis, 1996) or countries (Siddiqi, Kawachi, Berkman, Subramanian, & Hertzman, 2007) are illustrative examples.

Notwithstanding that especially Durkheim's work have been challenged and may even be partly flawed (Selvin, 1958), both Durkheim's and Rose's core scientific contributions cannot be underestimated. Rose later published his seminal book 'strategy of preventive medicine' where these insights were developed further and were accompanied with suggestions on prevention. Crucially, Rose's reflections on prevention have tremendous importance for a number of reasons. Firstly, by suggesting that some of the main health determinants; the important causes, inhere in the structures of society. They are distal and are to be found 'upstream', and should correspondingly be prevented by population-level interventions. These were radical thoughts in a discipline dominated by individual-level explanations, but they are simultaneously a reorientation back to the 'old' school of public health. Secondly, and related, by redirecting attention away from individuals (and implicitly their self-chosen bad

behaviour) towards the structures of society, the often adverse effect of victim-blaming (Petersen & Lupton, 1996), as well as risk-imposing (Førde, 1998) is avoided to a larger extent.

Rose's key message is that the causes of within-population variability (causes of cases) can be very different from the causes of between-population variability (causes of incidence). Or stated differently; disease determinants may be found both in individuals as well as in the structures of society (or any other higher levels of organisation).

Structuration theory

'Wholes and parts' immediately draw our attention to the concepts 'structure and agency'. Similarly, the above statement that "the characteristics of the population are shaped by the individuals and at the same time the characteristics and behaviours of the individuals are shaped by the characteristics by the population" also point in a particular direction. In his book 'the constitution of society', the sociologist Anthony Giddens outlined his influential ontological framework for the social sciences, essentially incorporating some of the legacy from Durkheim and implicitly also Rose (Giddens, 1984).

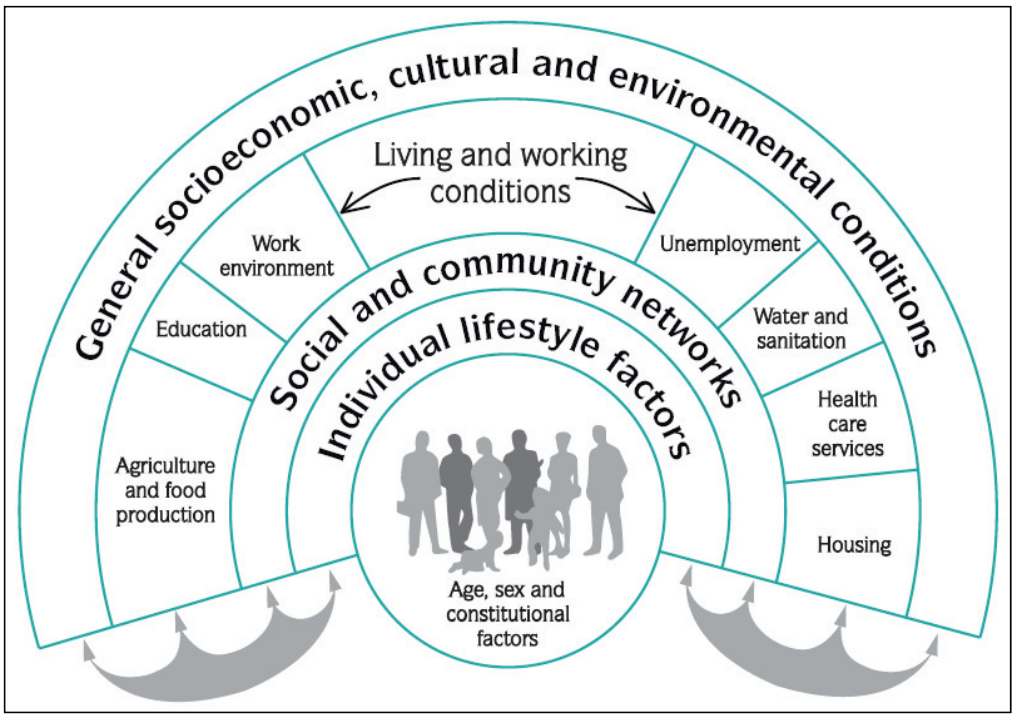
Giddens defines three major components in his ontology; structure, system and structuration. Structure is a set of rules and resources and does not include subjects, whereas social systems comprise the situated activities of the individuals. The structuration of social systems is essentially the modes in which these systems are both produced and reproduced by agents who are drawing upon various rules and resources. The structural characteristics of social systems are that they are both the medium as well as the outcome of social practices. Importantly, there is no one-way relationship between structure and agency, they are recursive and co-dependent, hence, structure is impossible without action because action reproduces structure. Similarly, action is not possible without structure because action starts within a given structure which is dependent of prior actions.

An important element of Giddens' ontology is his emphasis given to practical consciousness. This concerns the individuals understanding of "what is going on" in social life. Structure does not exist apart from the knowledge agents have regarding their daily activities. This is embodied in Giddens' construct of routinisation; the everyday activities that are constantly produced and reproduced. Routinised activities do not "just happen", but arise as the result of "reflexive monitoring of action which individuals sustain in circumstances and co-presence" (Giddens 1984 p. 64). Agents are hence considered to be conscious individuals. Action are the result of purposive, reasoning behaviour of agents within constraining and enabling features of the social and material context of that behaviour. For the individual, routinisation gives ontological security whereas at the collective level routinisation is important for the workings of institutions that exist because of the continued reproduction of routines.

The suggested framework acknowledges that structures shape social practices and actions, and in turn, the structures are continuously recreated by these practices and actions (Gatrell, 2002). Importantly, this interaction occurs within a socio-geographical context (Curtis, 2004).

The framework is consequently suited for studies investigating contextual, or area effects on health, and in particular health related behaviour (Duncan, Jones, & Moon, 1996). While also Bourdieu and his structure-agency-mediating concept of 'habitus' (Bourdieu, 1977) may offer a convenient framework, and arguably a more suitable one with respect to class-health-and-lifestyles (Williams, 1995), the clearer recognition to the concept of *place* in Giddens theory makes it more useful for the present analysis. While the framework is inherently tautological and not suitable for empirical testing, the structuration theory may nevertheless serve as a template. Furthermore, it may also be depicted in the well-known figure over the main determinants of health (fig 1).

Figure 1 The main determinants of health (Dahlgren & Whitehead, 1993)



While the Dahlgren/Whitehead model is very broad and general, it is nevertheless convenient. Importantly, the layers in the model have thin membranes, they are porous, and interact in complex ways (Popay, Williams, Thomas, & Gatrell, 1998). The porosity between the various hierarchical layers and the individuals illustrates the continuous interaction in a variety of *settings and locales*; from the microenvironment up to the macrolevel. As Curtis & Jones have noted;" locales' structure people's life-paths in class specific ways and are the context for daily routines, socialisation, interaction, exclusion and conflict" (Curtis & Jones, 1998 p. 651). The everyday social practices of actors interact with these social structures and they maintain and shape these social structures. At the same time the social structures also frame the actions of the individuals. There is hence a complex interaction between individual thought and action and the broader social context in which individuals live which is strongly influenced by the geographical context for social interaction (Curtis, 2004). "The relationship

between knowledge, risk, and behaviour can therefore be thought of as a complex interaction between structure, agency, beliefs, accounts and action" (Curtis & Jones, 1998 p. 651).

At a more general level the Dahlgren/Whitehead model attempts to capture the relationship between different modes of explanation in the public health field. At the heart of the model are the largely fixed biological givens in terms of age, sex and hereditary factors where the latter also includes *in utero* programming (Barker, 1991) of disease risk (e.g. poor nutrition in foetal life). Surrounding these biological givens is a set of factors labelled 'lifestyle' which includes smoking, alcohol consumption, dietary patterns and physical activity. Combined with the biological determinants these lifestyle factors (understood as individual behaviour) comprise the body of work widely referred to as 'risk factor epidemiology'.

The set of factors in the second ring represents individuals' interaction with peers and the immediate community, whereas the third layer highlights living and working conditions, food supply and access to general goods and services. At the outermost layer we find macro-level features, socioeconomic, cultural and environmental influences on population health which is usually associated with countries.

Consequently, the model clearly acknowledges that health is not just a matter of rational individuals making informed choices; it is heavily dependent on *contextual* features in which individuals are nested. Furthermore, as the authors of the model underlines;"This model for describing health determinants emphasizes *interaction*: individual lifestyles are embedded in social norms and networks, and in living and working conditions, which in turn are related to the wider socioeconomic and cultural environment" (Dahlgren & Whitehead, 2006 p. 20 *my emphasis*). The rationale for describing the model here is that it is illustrative for a range of contextual influences on individuals' health and that disease determinants may not always be adequately understood or prevented at the level of individuals.

Neighbourhood contextual effects

While the previous sections dealt with more fundamental ontological theories, this section aims to give a condensed overview of theories and perspectives inhering in this thesis which are concerned with the question of how "context" influence individual health. There have been major advancements in statistical modelling the last two or three decades, but lack of theory articulating the mechanisms concerned with how context affects health is a recurrent issue (Osypuk & Galea, 2007). This is especially true within epidemiology and public health where "contextual influences tend to be seen as a residual category, a black box of contentless miasma of unspecified influences on health" (Macintyre & Ellaway, 2003 p. 26). As lamented by Kaplan, there is considerable speculation but little understanding on exactly how context influences individuals' health (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996) and without some well founded theory we are left with a plethora of studies without any reasoned etiologic considerations (Kaplan, 2004).

Health geography is centrally concerned with the question of health variation in populations in different parts of the world and how we may ultimately understand these (Curtis, 2004).

This concern applies to macrolevel variations as well as to small scale variations. While this is a longstanding issue, a resurgence of interest came about as a consequence of work outside of geography by the publication of "Unhealthy Societies" in the mid 1990's (Wilkinson, 1996). The discussions following this book literally spurred an avalanche of studies and commentaries in various journals. Crudely summarised, Wilkinson claimed, based on cross-national ecological associations, that in modern societies it is the egalitarian societies that have the best health records. This is partly because these societies experience smaller income inequalities and are more 'socially cohesive'. Despite the premise of an unequal distribution of income, the postulated mechanisms were psychosocial, not material, and this quickly became a hotly debated issue (Lynch, Smith, Kaplan, & House, 2000; Lynch, Smith, Hillemeier, Shaw, Raghunathan, & Kaplan, 2001). Notwithstanding the limitations surrounding these findings, the work was instrumental in the sense that it clearly acknowledged the 'context' as an important explanatory factor for the health of the population as well as that of individuals.

Confining the discussion to sub-national contexts, it is quite possible that there are systems of organisation at multiple, yet lower levels, that may be of importance for the health of individuals. There is an extensive body of research that supports an argument that contextual effects, associated with place and space, may contribute independently or interactively to health variations (Curtis & Jones, 1998). However, a clear understanding of the ways that place may interact with health still remains 'under theorised'.

Curtis has put forward the "whole systems" perspective as a organising framework (Curtis, 2004). The framework uses biological rather than mechanical metaphors to understand reality, and emphasises the need to view factors influencing population health as complex systems. This focuses attention on the connections between the different parts of the system as well as on the individual parts. While it is very complex and extensive and virtually impossible to 'model' it is useful because (1) it examines the relationships which connects a variety of factors relevant to health, (2) it focuses attention on processes that are dynamic, and (3) accepts that these processes are sensitive to context (Curtis, 2004). The 'whole system' perspective also share some affinities with another framework using biological metaphors, namely that of *ecosocial* theory of disease distribution. Put crudely, the ecosocial (multilevel) framework seeks to integrate social and biological reasoning and a dynamic, historical, and ecological perspective to develop new insights into determinants of population distributions of disease (Krieger, 2001b, a; Krieger & Davey Smith, 2004; Krieger, 2005; McLaren & Hawe, 2005).

As Curtis (2004) notes, geography is a discipline well suited to the 'whole system' way of thinking, as geographers are concerned to examine the interaction between population and environment, and how this varies across space and in different types of places. Furthermore, the geography of health is focused on the ways that the health of populations is differentiated between places and the range of factors that explain these differences. Curtis & Jones (1998) argue that a perspective on the importance of place in health geography is informed by elements from a number of theoretical frameworks. They further suggest that these questions can be expressed in terms of conceptual landscapes.

The ‘landscape’ term is put forward and used metaphorically to convey the idea of a system of factors and processes that interact in particular settings to produce geographical variations. The included elements in a conceptual landscape depend on knowledge of the key relationships and causal pathways producing geographical variation. These different landscape terms (table 1) are associated with different theoretical perspectives and the key is to imagine these conceptual landscapes overlying each other in the same place (Curtis, 2004).

Table 1 Theoretical frameworks (source: Curtis, 2004)

Theoretical framework	*Landscape focus*
theories of sense of place/identity	therapeutic landscapes
theories of social and political control	landscapes of power and resistance
theories of production/structuration	landscapes of poverty and wealth
theories of consumption and lifestyle	landscapes of consumption
theories of ecological/epidemiological processes	ecological landscapes

Importantly, these theoretical frameworks envisaged in table 1 also incorporate ideas of space and place into the discussion of geographical health variation. Many studies refer to where people live is space, omitting any consideration of place and often end up with a ‘black box in which unidentified, ‘non-individual’ processes takes place’. To make a very simple yet useful distinction; space describes *where* a location is, whereas place describes *what* a location is (Tunstall, Shaw, & Dorling, 2004). As noted by health geographers quite a while ago; ”Seldom, however, does location itself play a real part in the analysis; it is the canvas on which events happen but the nature of the locality and its role in structuring health status and health related behaviour is neglected” (Jones & Moon, 1993 p.515).

The present thesis is predominantly drawing on the production/structuration framework, but there are also connotations to some of the others referred to below. Crudely summarising an entire book devoted to these frameworks (Curtis, 2004) the middle three in table 1 may be described in the following manner. Landscapes of power consider the significance of geographies of power for health variations. Here, the thought, choice, and agency of individuals is seen to interact with power structures in society producing health differences between more or less powerful groups in the space-time context of their everyday lives. The processes involve control over resources, territoriality and surveillance all of which contribute to landscapes of power.

Landscapes of poverty and wealth devote attention to the importance of inequality in wealth. The links between poverty and health disadvantage is important, and a particular focus is on the importance of material poverty in *places*. An important distinction between *people poverty* and *place poverty* is highlighted (Smith, 1977). In the former, people are deprived by virtue of their position in the socioeconomic system and geographical variations in poverty and health emerges as a result of different compositions of deprived individuals, or stated differently; it is the spatial manifestation of the well-known socioeconomic health gradient. In the latter it

refers to poor access to locationally specific goods and services (Moon, Subramanian, Jones, Duncan, & Twigg, 2005). Importantly, individual poverty and place poverty may interact. This underlines the role of places and the interaction between people and places ultimately being detrimental to their health. Crucially, the ultimate consideration is to regard 'place effects' in terms of processes which are geographically differentiated in space and time (Curtis, 2004). The core proposition is that wealth distribution is important to health.

A landscape of consumption draws attention to varying distribution and consumption of goods and services, including health services, to individuals and places. While individual differences in wealth may explain differences in private consumption, there are also other 'structural' factors of relevance. These structural factors may be of three types; administrative and political structures, social structures operating in different social milieux, and also spatial organisation of infrastructure for delivery of goods and services (Curtis, 2004). Importantly, these structures are variable in different geographical settings and are also dynamic, i.e. they change through time. This change may be due to; health system reforms, commodification of health and health care, and also technological developments. The core issue, according to this perspective, is essentially that conditions in the political economy influence variability due to some key processes. These processes include; social reproduction, public choice, bureaucratic organization, uneven market development, technological advances, commodification and harmonisation of consumption practices within societies or places (Curtis, 2004).

These landscape focuses are associated with some broad theoretical *frameworks* and researchers may to varying degrees draw on one or several of them, including those not summarised here. There is however, still a need for a better understanding about the mechanisms by which area of residence *actually* influences health (Frohlich, Corin, & Potvin, 2001; Boyle & Duke-Williams, 2004) and this requires some further and careful considerations. Firstly, the question of scale, or level, at which contextual processes may operate. Secondly, and closely related, what types of contextual features one may be interested in. Thirdly, it requires some critical reflections as to how, directly or indirectly, context influences individual health. Finally, and importantly, all these issues should ideally be tailored for the particular outcome at hand. Correspondingly, it is important to have a differentiated picture on area effects. "Rather than being one single, universal "area effect" there appear to be some area effects on some health outcomes, in some population groups, and in some types of areas" (Macintyre, Ellaway, & Cummins, 2002 p. 128).

Following Macintyre and colleagues (Macintyre, MacIver, & Sooman, 1993) and Curtis & Jones (Curtis & Jones, 1998) the following broad categories of neighbourhood features may potentially influence individual health: (1) the physical environment; (2) the availability of healthy and unhealthy environments at home, work, or at play; (3) provision of private and public services; (4) socio-cultural features of the neighbourhood, and; (5) neighbourhood reputation. Consequently, 'context' includes attributes of both the physical and social environment that extend beyond individuals. Two more specific features, essentially associated with several of these broad categories are *deprivation* and *social capital*.

There is a 200 year tradition amongst British researcher to study the impact of poverty on health (Davey Smith, Dorling, & Shaw, 2001). As noted in the introductory section, there is a health gradient between individuals in term of their socioeconomic position. Similarly, we find a graded relationship between deprivation and health for geographical areas (Curtis, 2004), and this is occasionally interpreted as a mere "spatial manifestation of the social class gradient". Research from the UK does however give a more nuanced picture. Not only are individuals deprived, but deprived individuals are also likely to be living in the sorts of neighbourhoods that also lack the infrastructure to live a healthy life. This so-called "deprivation-amplification effect" applies across a whole range of potential environmental influences on health (Macintyre & Ellaway, 2003). This also has clear resemblance with "the inverse care law" whereby the provision of health services can be inversely associated with the need for them (Hart, 1971). The key message is that some individuals may experience a double jeopardy in terms of deprivation and both may be health detrimental. It should however be noted that there may be exceptions to this deprivation-amplification effect; it is not always the case that individuals in deprived places have poorer access to various kinds of health related infrastructure (Macintyre, 2007; Pearce, Witten, Hiscock, & Blakely, 2007, 2008).

According to some authors a distinction can be made between *material deprivation* and *social deprivation*. In the former, a neighbourhood may be lacking various services, amenities and resources as well as having a poor physical environment, whereas in the latter, lack of participation and poorly developed social relations are implicated in poor health (Gatrell & Elliott, 2009). It could be argued that it is not entirely correct to equate social deprivation with the concept of social capital. While the former has an inherent pathogenic flavour (a risk factor), the latter is usually regarded as a salutogenic concept (a health promotive factor) (Kawachi, Subramanian, & Kim, 2008). That said, social capital also has a clear similarity with the deprivation concept, because it may be conceived of as an individual attribute as well as a contextual feature.

The social capital concept is described in article 1 (Sund & Krokstad 2009) hence a brief summary is provided here. Social capital means different things to different theorists, but there is general agreement that social capital is related to social interaction, membership in social networks and the consequences of these (Mitchell, Bartley, & Shaw, 2005). Although the origins of the concept may be traced back to Durkheim and even Marx, it is the work by political scientist Robert Putnam (exposed within public health through Richard Wilkinson's concept of social cohesion) that has attracted most attention within the public health domain (Moore, Shiell, Hawe, & Haines, 2005). Its main tenets are that social capital is a feature of communities and that participation in civil society enhances trust, norms and networks, which in turn are conducive to the health of individuals as well as the health of societies. The social capital concept is disputed and the literature is more comprehensively discussed and also reviewed in article 1 in this thesis.

As a preliminary summary, there are two neighbourhood constructs that have received much attention in the public health area; place deprivation and social capital. Both of them may contribute in explaining geographical inequality in health as well as having an independent

effect on individual health. A number of reviews have been carried out (Macinko & Starfield, 2001; Pickett & Pearl, 2001; Diez Roux, 2003b; Almedom, 2005; Islam, Merlo, Kawachi, Lindstrom, & Gerdtham, 2006; Diez Roux, 2007b; Riva, Gauvin, & Barnett, 2007; Kim, Subramanian, & Kawachi, 2008; Lindström, 2008; Diez Roux & Mair, 2010), but as will become apparent later (in sections 5 and 6), they also highlight serious methodological limitations; hence, any firm conclusions cannot be drawn. It is also important to note that associations between both these constructs usually have been investigated in urban settings, i.e. across urban neighbourhoods and that less is known about contextual influences across rural settings (Riva, Curtis, Gauvin, & Fagg, 2009). A recent study on mental health suggests that contextual features may indeed operate differently in rural and urban settings (Peterson, Tsai, Petterson, & Litaker, 2009).

Family, or household, contextual effects

The neighbourhood, or area of residence, may constitute an important grouping of individuals and may inhibit features of importance for their health. There is also reason to believe that a more basic fundamental grouping; the family or household, may be of importance for the health of individuals. Although these are closely related lines of inquiry, they seem to be separate bodies of research. Family, or household effects, is not the major foci in this thesis, and came admittedly through the course of the project, hence a rather brief summary is provided in the following.

We find an extensive literature on health concordance between spouses, spanning disciplines such as sociology, psychology and epidemiology (Meyler, Stimpson, & Peek, 2007). Several theories have been proposed to explain health concordance. *Assortative mating* suggests that individuals are more likely to marry people who share similar sociodemographic characteristics as well as attitudes and behaviours, ultimately giving rise to health similarities, or health concordance. This would be the equivalent of selective migration where similar individuals are sorted into neighbourhoods on the basis of individual characteristics (this is later discussed in sections 5 & 6). A competing hypothesis contends that health similarities arise as a consequence of *shared resources*. When individuals marry they often share the same environment, financial resources and social networks, hence features of this shared environment can be both beneficial and health detrimental. An alternative theory highlights the role of *social control*, where one of the spouses (usually the wife) will attempt to control the behaviours of the other to keep the husband healthy (e.g. to quit smoking). These two latter theories share similarities with contextual neighbourhood features described previously; individuals are situated in the same environment and consequently exposed to many of the same features.

There are also more specific theories concerned with health behaviour and behavioural conformity for families and households. Although this is a diverse set of theories, they may be grouped under two broad headings; *the role of information* and *the nature of social interaction* (Rice, Carr-Hill, Dixon, & Sutton, 1998). The notion of bounded rationality which emphasises limited knowledge of available opportunities and their consequences, highlights

the role of information in determining behaviour. Theories of social interaction suggest that behavioural conformity arises as a consequence of how individuals relate to other family members. Individuals may be guided by a desire to conform with others giving rise to common behaviours, or there may be social norms or conventions underlying these group interactions. In contrast to these theories of transmission of information and social interaction, there may also be truly exogenous factors, or contextual family features over and above its constituent parts that create behavioural conformity. Finally, there may of course be processes of assortative mating giving rise to correlated behaviour within families and households.

In summary, there is much complementarity between how we conceive neighbourhood clustering and clustering at the family or household level. One difference however, is that families or households are more easily defined than the former. Additionally, this family level could also have been depicted as a separate layer in figure 1, residing between individual lifestyle factors and social and community networks. However, families and households have received scarce attention within social epidemiology so far (Merlo, 2010). The family level is incorporated in the analyses in article 3 and 4 in this thesis with BMI and smoking as the outcomes respectively.

5. Multilevel analysis

Preface – limitations of, and fallacies from, single-level analyses

What hopefully should be apparent given the previous discussions is that studies with individuals as the sole unit of analysis may inhibit various important limitations. While both public health and epidemiology in its origins was ecological, relating environmental and community characteristics to health and disease, the growing importance of chronic diseases led to a search for individual behavioural and biological causes for disease (Pearce, 1996; Susser & Susser, 1996a, b). This individualisation of risk, usually referred to as risk-factor epidemiology, has perpetuated the idea that risk is individually determined rather than socially determined. This has ultimately discouraged research into the effects of contextual or environmental variables on individual level outcomes, essentially de-contextualising disease. There are indeed critical voices towards such individualisation of risk within epidemiology (see e.g. Pearce, 1996; Susser & Susser, 1996a, b; Koopman & Lynch, 1999; Schwartz & Carpenter, 1999; Schwartz, Susser, & Susser, 1999; Diez Roux, 2000; Smith, 2001; Rockhill, 2005), however these authors constitute a very small minority.

Group-level, or ecological, analyses pays more attention to context (table 2). These are analyses based on data that are limited to characteristics of groups (usually aggregates) of individuals. Typically, these are aggregates of geographically defined populations such as wards, municipalities, counties and countries (such groupings may also be done according to social or demographic categories or time periods) (Curtis & Cummins, 2007). The literature is extensive, and in terms of their objectives they may be classified into two broad categories: inference on individual level and purely contextual inference (Greenland, 2002).

Table 2 Types of studies (source: Diez Roux, 1998)

Independent variable(s)	Dependent variable	Type of study
Group-level	Group-level	Ecological
Individual-level	Individual-level	Individual-level
Group- and individual-level	Individual-level	Multilevel or contextual

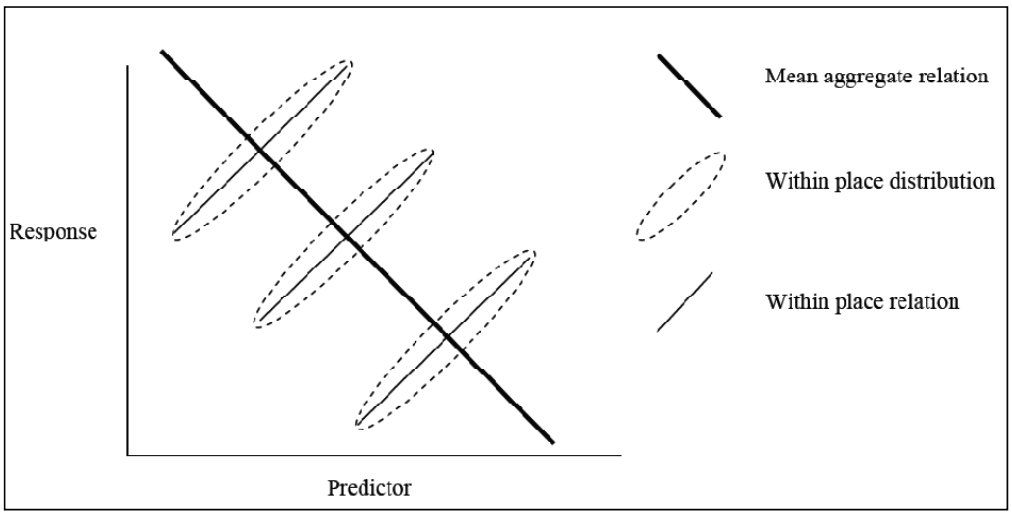
It is, however, clear that both individual-level and ecological analyses have serious limitations and may be subject to a number of fallacies (table 3). This is mainly because the methods fail to fit the conceptual model (Diez Roux, 1998). The ecological fallacy concerns the fallacy of drawing inference at the individual level based on group-level data (Robinson, 1950; Selvin, 1958; Susser & Susser, 1996a; Wakefield, 2008). As figure 2 shows, the aggregate relation may even be of opposite sign to the within-place, individual-level relation on which it is based.

Table 3 Types of fallacies (source: Diez Roux, 1998)

Unit of analysis		Level of inference	Type of fallacy
Group		Individual	Ecological
Individual		Group	Atomistic*
Individual	(relevant group-level variables excluded)	Individual	Psychologistic*
Group	(relevant individual-level variables excluded)	Group	Sociologistic

** Also called individualistic fallacy by some authors*

Figure 2 Within- and between-place relationships of opposite sign (modified from: Jones & Duncan, 1995)



Conversely, the topic of interest may also lie on studying group level associations based on observations at the individual level, and these may not always hold at the group level. The atomistic fallacy (Alker, 1969), which is the individual level counterpart, refers to the fallacy of drawing inference at the aggregate level based on individual-level data.

Two other fallacies are also possible according to table 3. Firstly, ignoring relevant group level variables whilst studying individual-level associations may give rise to the psychologistic fallacy, whereas ignoring individual level variables in a study of group level associations may give rise to the sociologistic fallacy (of which Durkheim may have committed in his book *Le suicide*). Both these two types of fallacies can essentially be thought of as special cases of confounding where relevant variables pertaining to other levels are excluded from the model (Diez Roux, Schwartz, & Susser, 2002).

In the context of the present thesis where the aim is to study outcomes at the individual level, it is hence necessary to avoid two possible fallacies; the ecological and the psychologistic fallacy. It is evident from table 2 that a third type of study design is possible; the multilevel statistical framework. This statistical framework accommodates both individual as well as ecological predictors on individual level outcomes. Importantly, it is *not* subject to the ecological fallacy, nor to the psychologistic fallacy (or more generally; the individualistic fallacy).

Besides these fallacies, it is important to emphasise that aggregate studies are incapable of answering the canonical question of whether *place makes a difference to the individuals' health* – this can, essentially, only be achieved within a multilevel statistical framework (Jones & Duncan, 1995; Jones, Gould, & Duncan, 2000; Jones, Duncan, & Twigg, 2005; Moon et al., 2005). Previous research working on a single level has essentially conflated levels of analysis and inference (Jones & Moon, 1993).

The multilevel statistical framework is outlined in the following section. The argument is that we need to *simultaneously* examine individuals at one level while at the same time accounting for the ecologies, or contexts, in which they are located. As some influential researchers within health geography has put it in the introductory section of their textbook;"What we argue for...is a greater recognition that both social *and* geographical factors influence different health outcomes in different ways. It is teasing out the relative influence of these factors that remains an exciting research challenge" (Graham, Boyle, Curtis, & Moore, 2004 p. 6 *authors emphasis*). This is precisely what this thesis aims to do in the enclosed empirical articles (articles 2 to 4); to tease out the relative influence of geography, and ultimately model contextual (i.e. geographical) features on individuals' health. It is argued that multilevel models represent a major advancement compared to research conducted on a single level.

Multilevel modelling

In the past 20 years multilevel modelling has emerged as an important approach within public health and in the social sciences. The statistical framework allows the simultaneous consideration of composition and context, and consequently, the definitive assessment of so-

called area effects on health-related outcomes (Moon et al., 2005). One of its big advantages is that it is conceptually realistic since it can handle the micro-scale of composition and the macro-scale of contexts within one model simultaneously.

In the social-, as well as in the medical sciences, multilevel, or hierarchically structured populations usually are the norm. Such hierarchical populations can be individuals within households within areas, or patients in wards within hospitals. Such hierarchies are often described in terms of clusters of level 1 units nested within each level 2 unit etc. As already noted, a common criticism of using standard statistical models to analyse quantitative data in the social sciences, as well as in epidemiology, is that these methods usually place too much attention on the individual, and very little on the social and institutional contexts in which the individuals are located.

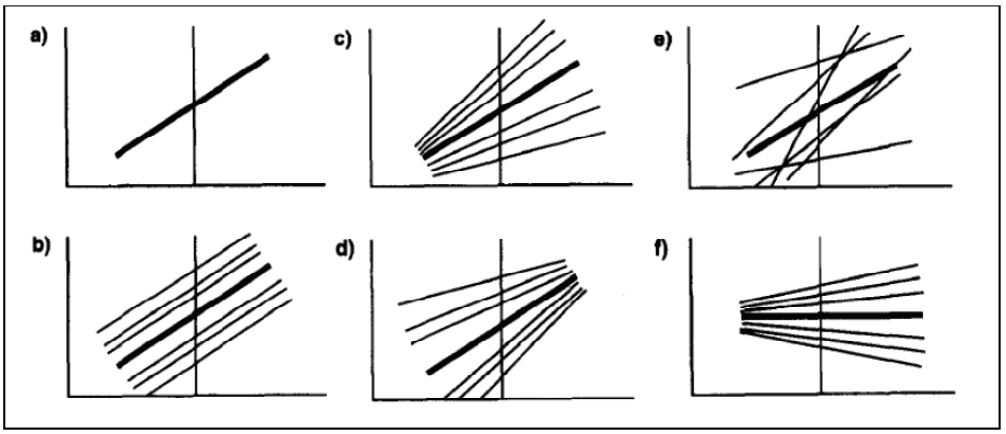
Multilevel models counter this imbalance by modelling processes at all levels of organisation. By paying attention to the various levels in the population, multilevel modelling enables us to understand *if*, *where*, and *how* such effects are occurring. Crucially, fitting a model which does not recognise the existence of clustering within higher level units creates serious technical problems, since clustering in general will cause the standard errors of the regression coefficients to be underestimated. Correct standard errors will only be estimated if variation at the higher levels of organisation is allowed for in the analysis. Multilevel modelling provides an efficient, if not the only way, of accommodating this. Importantly, multilevel models make it possible to model and investigate the relative sizes and effects of higher level characteristics on individual outcomes, as well as those of individual characteristics such as age, sex, socioeconomic status etc.

Importantly, by distinguishing between different levels of which the health determinants may operate, multilevel models treat these higher level contexts as if they are coming from a larger underlying population. Utilising this random sample of contexts, the procedures make inference about the variation among all contexts in this population, and consequently, these contexts are not treated as fixed but rather as a random property from a larger population.

A graphical introduction to multilevel models

In the following sections, a simple example is used, where individuals are nested within communities/places. We start by consider a hypothetical situation in which the probability of reporting ‘long term illness’ measured on a continuous scale (response variable) is a function of the predictor variable individual age (centred on its mean). An analysis using single-level simple regression would generate the relationship shown in figure 3 (a), whereby the relationship between long term illness and age is shown on a straight line. In general, older people are more likely to report being sick, but the context in which this occurs is neglected.

Figure 3 Varying relationships between long term illness (y-axis) and age (x-axis) (From: Duncan, Jones, & Moon, 1998)



Model 3 (b) on the other hand, shows a multilevel model (two-level) in which individuals are nested within six different communities. This 'random intercept' model shows a similar positive association, but here each community has its own illness/age relationship represented by a separate line. The single thicker line in the middle represents the average relationship across all communities. The parallel line for each community shows that the illness/age association is similar in all communities but, importantly, some communities have consistently higher rates of illness than others. This model is hence context specific.

In models 3 (c) and 3 (d) additional complexity is added as the steepness of the lines varies from place to place. From 3 (c) we notice that for younger individuals it seems like place makes little difference for long term illness whereas for the older individuals there is a high degree of between-place variation in long term illness. In 3 (d) the situation is reversed with high between-place variation for young individuals and smaller for the older.

In fig 3 (e) the relationship is even more complex, where in some places the young individuals have relatively high rates of long term illness and in others it is the elders. Figure 3 (f) is rather unlikely in terms of the present example. Here we see that across all communities there is no relationship between long term illness (thick line) and age, but in some specific communities there is a distinct relationship. Models 3 (c to f) are commonly referred to as random slopes models.

As briefly mentioned earlier, a key feature of multilevel models is that these communities are treated as a sample drawn from a population, and their potentially different intercepts and slopes are treated as coming from distributions at a higher level. A multilevel analysis hence summarises these higher level distributions in terms of two parts; a *fixed* part that is unchanging across communities, and a *random* part that is allowed to vary. The fixed part gives the mean value of each distribution (the average slope and intercept across all communities shown by the thick lines in fig 3). Whereas the random part consists of variances that summarises the degree to which the community specific slopes and intercepts differ from these averages.

It is important to note that this graphical introduction is a rather crude simplification. Firstly, the number of higher level units (only six communities) is far too small for a proper multilevel random effects analysis (Rabe-Hesketh & Skrondal, 2008b). Secondly, and importantly, one might get the impression that separate lines are fitted to each particular community in figure 3. This would be the equivalent to procedures based on single-level Ordinary Least Squares (OLS) regression in which the fixed part of the model is expanded to include a slope and intercept term for each community and this would involve (in this example) fitting 12 parameters. This would require a very large sample size to get reliable estimates, and such an approach would be highly inefficient.

In contrast, multilevel techniques involve estimating the statistical characteristics of the higher level intercept and slope distributions for the population using the communities as a sample. Consequently, it is the random part of the model that is expanded. Considering the example in figure 3 (c to f), we only have to estimate two fixed part terms; the average intercept and average slope across all communities, and three random terms summarizing the variability between specific communities. If the sample of communities were expanded to include for instance 200 communities, we would still be estimating only 3 random terms. It should be noted that although place-specific intercepts and slopes are unknown in multilevel models, it is possible to obtain very accurate predictions of them (Jones & Bullen, 1994).

From graphs to equations

In this section the basic two level random intercept and random slope models are outlined. The previous long term illness/age example where individuals are nested within communities/places is continued.

The response is the continuous long term illness and the main explanatory variable is continuous age (centred on its grand mean). We can write the standard simple regression model relating long term illness to age as:

$$y_i = \beta_0 + \beta_1 x_i + e_i \qquad \text{Eq.1}$$

This gives us a single prediction line as shown in fig 3 (a). Once we make this a multilevel model we can have a different prediction line for each community and two models are possible:

The random intercept model:
Here communities differ in terms of their intercept only, which gives rise to a set of parallel lines as shown in figure 3 (b).

The random slopes model:
Here communities vary in terms of both their slopes and their intercepts, which gives rise to the set of lines depicted in figure 3 (c to f).

The random intercept model

Equation 1 can be extended to represent the random intercept model (the microequation):

$$y_{ij} = \beta_{0j} + \beta_1 x_{ij} + e_{ij} \qquad \text{Eq.2}$$

y_{ij} is the illness score for the *i*th individual in the *j*th community

β_{0j} is the intercept for the *j*th community

β_1 is the slope coefficient for the age variable

x_{ij} the age value for the *i*th individual in the *j*th community

e_{ij} is the departure of the *i*th individual in the *j*th community from the community's predicted line

The intercept for the *j*th community (β_{0j}) is expressed as (the macroequation):

$$\beta_{0j} = \beta_0 + u_{0j} \qquad \text{Eq.3}$$

Where β_0 is the average intercept for all communities in the sample and u_{0j} is the random departure for the *j*th community.

Substituting equation 3 into equation 1 we have:

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + u_{0j} + e_{0ij} \qquad \text{Eq.4}$$

In this basic multilevel model we assume:

$$u_{0j} \sim N(0, \sigma^2_{u0})$$
$$\mathbf{e}_{0ij} \sim N(0, \sigma^2_{e0})$$

Equation 4 exemplifies one of the key differences between multilevel models and standard multiple regression. The model now has two random variables; $\mathbf{e}_{0ij}$, the individual level random variable and u_{0j} , the community level random variable. A simple regression model only has one random variable, often called the error term. As multilevel models become more complex they often contain many random variables.

In this model four parameters are estimated; β_0 and β_1 which are like standard regression coefficients. They give the average prediction line from which the *j*th community's line is offset by a random departure u_{0j}. These regression coefficients (β_0 and β_1) are called *fixed*

parameters. We also estimate $\boldsymbol{\sigma^2_{u0}}$, the variance of the community level intercept departures and $\boldsymbol{\sigma^2_{e0}}$, the variance of individuals long term illness score around their community's summary line. The variances of these random departures at the individual and community levels are known as the *random parameters.*

In the current model there are two levels, individuals are the lower level units, and usually this lowest level is called level 1. Individuals are nested within the higher level units, communities, and generically the higher level is called level 2. In the presence of a four-level nested population structure, for example individuals within families within wards within municipalities, then individuals would be level 1, families would be level 2, wards level 3 and municipalities would be level-4. This can be expressed as (equation 5):

$$y_{ijkl} = \beta_0 + \beta_1 x_{ijkl} + f_{0l} + v_{0kl} + u_{0jkl} + e_{0ijkl} \qquad \text{Eq.5}$$

With assumptions:

$$f_{0l} \sim N(0, \sigma^2_{f0})$$
$$v_{0kl} \sim N(0, \sigma^2_{v0})$$
$$u_{0jkl} \sim N(0, \sigma^2_{u0})$$
$$e_{0ijkl} \sim N(0, \sigma^2_{e0})$$

Returning to the two-level example: The correlation between two individuals in the same community, which is referred to as the "intraclass correlation coefficient" (ICC) can be expressed as:

$$\rho = \frac{\sigma^2_{u0}}{\sigma^2_{u0} + \sigma^2_{e0}}$$

The higher the value of this correlation, the more similar two individuals from the same community is, compared to two individuals chosen at random from the population. This is also the between-community variance over the total variance, usually referred to as the variance partition coefficient (VPC). As the effect of clustering increases (or alternatively; the between-community variance) it becomes more important from both a *technical* as well as a *substantive* viewpoint to use multilevel techniques.

The technical issue is that standard multiple regression assumes that the observations are independent. Clearly, in the presence of clustering (i.e. non-independence), this assumption is false. This results in the standard errors of the regression coefficients produced by multiple regression being underestimated, which can lead to incorrect inferences. Consequently one will be inferring relationships that appears significant when in fact they are not. The existence of a non-zero intraclass correlation coefficient hence means that traditional estimation

procedures, such as 'ordinary least squares' (OLS), are inapplicable (Goldstein, 2003). It is possible to obtain corrected standard errors for the fixed effects estimates by means of so-called marginal-, or population-average models such as the generalized-estimating equation (GEE) (Diez Roux, 2000). But unlike multilevel models such procedures treats the within-unit clustering as a 'nuisance' that is to be corrected and not something of intrinsic substantive interest (Subramanian, Jones, & Duncan, 2003).

The substantive issue is hence, that in the presence of high amounts of clustering, much of the total variability is between higher level units and therefore it becomes important to explore the nature of this variability. Multilevel models hence provide an excellent framework for teasing out the *relative* contribution of geographical (or any other sources of nesting) from individual factors on individuals' health and subsequently *explaining* geographical and individual variability.

The random slopes model

The random intercept model can be extended to allow for the possibility of communities having different slopes by allowing the slope coefficient, $\boldsymbol{\beta_1}$, to vary randomly at the community level (as illustrated in fig. 3 c to f):

$$\boldsymbol{y_{ij} = \beta_{0j} + \beta_{1j} x_{1ij} + e_{0ij}}$$

$$\boldsymbol{\beta_{0j} = \beta_0 + u_{0j}}$$
$$\boldsymbol{\beta_{1j} = \beta_1 + u_{1j}}$$
$$\begin{bmatrix} u_{0j} \\ u_{1j} \end{bmatrix} \sim N(0, \Omega_u) \quad \Omega_u = \begin{bmatrix} \sigma^2_{u0} & \\ \sigma_{u01} & \sigma^2_{u1} \end{bmatrix}$$
$$\mathbf{e_{0ij}} \sim \boldsymbol{N(0, \sigma^2_{e0})}$$

Eq.6

$\boldsymbol{u_{0j}}$ and $\boldsymbol{u_{1j}}$ are random departures at the community level from $\boldsymbol{\beta_0}$ and $\boldsymbol{\beta_1}$. They allow the *j*th community's summary line to differ from the average line in both its slope and intercept. Both $\boldsymbol{u_{0j}}$ and $\boldsymbol{u_{1j}}$ follow a multivariate Normal distribution with mean 0 and covariance matrix Ω_u. In this model there are two random variables at level 2 so Ω_u is a 2 by 2 covariance matrix. The elements of Ω_u are:

var($\boldsymbol{u_{0j}}$) = $\boldsymbol{\sigma^2_{u0}}$ (the variation across the community summary lines in their intercepts)
var($\boldsymbol{u_{1j}}$) = $\boldsymbol{\sigma^2_{u1}}$ (the variation across the community summary lines in their slopes)
cov($\boldsymbol{\sigma^2_{u0}}$, $\boldsymbol{\sigma^2_{u1}}$) = $\boldsymbol{\sigma_{u01}}$ (the community level intercept/slope covariance).

Individuals scores on long term illness depart from their community summary line by the amount; $\mathbf{e_{0ij}}$.

A significant value for $\boldsymbol{\sigma^2_{u1}}$ would be evidence that the effect of age varies across communities. In addition, a positive covariance between intercepts and slopes would give rise to the pattern found in figure 3 (c); suggesting that communities with higher intercepts have steeper slopes (a 'fanning-out' pattern). Conversely, a negative covariance would give the pattern shown in fig. 3 (d).

One may further add other individual-level explanatory variables to the model such as gender, marital status, socio-economic status etc, just as in ordinary multiple regression. As well as estimating an average effect, all the coefficients of these variables can be made random at the community level to see if these average effects indeed vary across communities. Importantly, characteristics of communities, so-called level-2 explanatory variables can readily be included in the model as well as interactions within and between individual and community characteristics.

The very important ICC (or VPC) statistic is, however, no longer available in a random slope (or random coefficient) model. This is because the variance at level-2 is no longer constant, or similar, for all individuals as in the random intercept model. Rather, there is now a variance function at level 2 that is related to the individual predictor at level 1. This can be exemplified by considering figure 3 (c) with the 'fanning out' pattern. It is obvious, that the intercept variance is rather small for young individuals whereas the intercepts varies much more amongst the oldest. The higher level variance is dependent on the age predictor and wee se that the between-community heterogeneity is an increasing function of age. Instead of a summary ICC, or VPC, the variance in a random slopes model is usually graphically depicted (for an eloquent illustration, see: Merlo, Asplund, Lynch, Råstam, & Dobson, 2004).

Potentialities, challenges, limitations and pitfalls in multilevel modelling

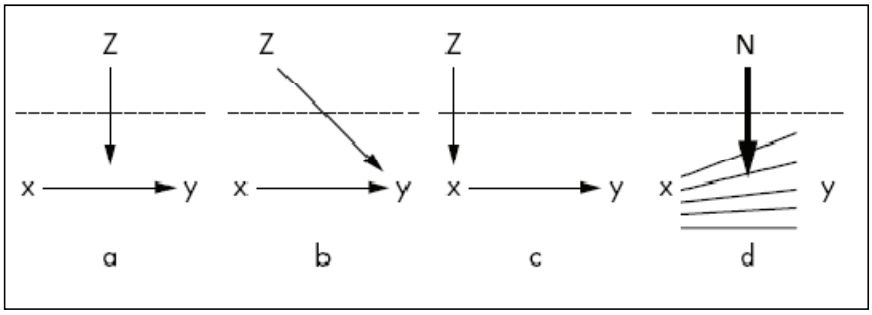
It is argued that the multilevel modelling framework is both conceptually realistic, it is parsimonious and it is efficient. From a theoretical and epistemological point of view it is a useful method when applying a structurationist view of the world (Duncan et al., 1996) since it, at least on the surface, gives due weight both to individual agency (at the individual level) and broader structural determinants (at higher levels) (Gatrell, Berridge, Bennett, Bostock, Thomas, Popay et al., 2004). There are a number of advantages worth highlighting, and some of them have briefly been mentioned already. Specifically, the two major advantages concerns corrected standard errors for the fixed effects parameters in the presence of within-unit correlation (non-independence). In addition, and importantly, we get a good assessment of the relative importance of context, or place, for individuals' health. And also; whether place matters more for the health of some categories of individuals and less for others?

Most of the existing accounts of multilevel methods have been largely restricted to two-level structures, typically individuals at level 1 nested within neighbourhoods at level 2. There is however, no reason to expect that these two levels are the only important levels. Indeed, there may be a multiplicity of levels, and the failure to account for this may in some cases attribute variability to the wrong levels (Tranmer & Steel, 2001). The multilevel framework can principally handle multiple numbers of levels, but limitations in terms of power may rapidly be imposed. There is really no consensus on sample size in multilevel modelling, because it is dependent on numerous issues relating to types of models, estimation techniques, and in particular which parameters (fixed or random) one is primarily interested in. It is however important to note that in addition to an adequate number of individuals, the number of higher level units is equally important, especially if primary interest is the variance/covariance components.

While the running example so far has used a continuous response variable, multilevel models can handle a wide range of responses. So-called generalized multilevel models exists to deal with both binary responses (with logit-, log-log-, and probit link functions), multiple category responses (multinomial and ordered categories) as well as counts (poisson and negative binomial). While these models work by assuming a non-normal distribution for the random part on the individual level, they do maintain normality assumption at higher levels (Blakely & Subramanian, 2006). Another attractive feature worth mentioning is that it is possible to model multiple outcomes simultaneously. The key feature with such multivariate multilevel models (which has a distinctly different meaning here than in traditional multivariate regression) is that a set of outcomes is nested within individuals. In other words, it is possible to conceive the outcomes as the lowest level and individuals at a second (higher) level.

Just like in ordinary regression modelling, it is possible to model interactions or effect modifications between fixed part predictors. Such interactions can be specified between predictors coming from both levels in a multilevel model. As illustrated in figure 4 (a), a contextual predictor may modify the association between an individual level covariate and the outcome (as figure 4 also shows, the contextual effect may also be direct (b), or indirect (c)). An illustrative example relates to the health effects of living in a place with a high or a low unemployment level (a contextual characteristic) whilst being unemployed; is it more health detrimental for the individual to be unemployed in a place with a high or a low unemployment rate?

Figure 4 A typology of macro-micro relations (cross level effects and cross level interactions) (Source: Merlo, Chaix, Yang, Lynch, & Råstam, 2005)



Besides modelling interactions between various levels in the fixed part of a model, it is also possible to do this in the random part. As previously illustrated in fig 3 (c), the between-community variance can differ according to age by allowing age to vary across communities in the random part at level 2. A random slope analysis can also be conceived of as a cross level interaction effect. But unlike in the fixed-effect interaction example we do not specify an interaction term between two predictors. Rather, we allow a lower level predictor to vary randomly across higher level units by specifying a variance function at level 2. Consequently, interactions can be specified both in the fixed and random part of a multilevel model. The difference is that in a fixed approach, one specifies how two *characteristics* jointly influences the outcome, whereas in a random approach we see how one characteristic at the individual level varies according to the sample of neighbourhoods as a *whole* (see fig. 4 (d)).

In addition to allowing age to vary across communities, it is in fact also possible to allow age to vary at the individual level, i.e. to check for non-constant variance (heteroskedasticity) (Jones, 1991). This is important in multilevel modelling because there may be confounding across levels – what appears to be contextual heterogeneity (variance at a higher level) may be due to between-individual heterogeneity (Bullen, Jones, & Duncan, 1997). So apart from modelling the average effect of age (in the fixed part), it is possible to model the variance (in the random part) as a function of age at all levels in the model.

In multilevel models, residual variation in the response is partitioned into components that can be attributed to different levels of analysis. The Intraclass correlation coefficient (ICC) or the variance partition coefficient (VPC) is often used interchangeably to describe this clustering or between-higher level heterogeneity. As previously mentioned they can be given two interpretations. Firstly, it can be seen as an expression of how similar two individuals within a higher level unit is, compared to two randomly chosen individuals from the population (Goldstein, 2003). The second interpretation is that it corresponds to the percentage of variation which is attributable to a higher level (Subramanian et al., 2003). While the ICC and the VPC are similar in a random intercept model, their definitions and usage should ideally be more specific. One way of viewing them could be to use ICC when the primary interest is to adjust the standard errors for the fixed effect estimates for non-independence/clustering, and the VPC when there is a substantive interest in the higher level variability.

Depending on the size of clustering, the standard errors for the fixed effect estimates may have to be corrected either through so-called 'population averaged models' or within a multilevel model (Diez Roux, 2000, 2002a). This is particularly important for higher level fixed-effect estimates, but this has importance for lower level associations as well (Snijders & Bosker, 1999; Hox, 2002; Rashbash, Steele, Browne, & Goldstein, 2009). If interest is mainly concerned with fixed effects, both methods will suffice. The major difference however, is that in multilevel models the VPC is important because it has *substantive* interest, it is not treated as a 'nuisance', or merely something to 'correct for' (Goldstein, Browne, & Rasbash, 2002a; Merlo, 2003; Merlo, Ohlsson, Lynch, Chaix, & Subramanian, 2009). Stated differently, it allows us to make an assessment of the relative influence of geographical factors on individuals' health. In addition, as previously described, the VPC cannot be expressed as a summary-measure of between-higher-level-variability in a random slopes model, because the variance is a function of one or more individual level predictors.

While the calculation of the VPC for continuous outcomes in straightforward, it is more cumbersome when the response is non-linear (Goldstein, Browne, & Rashbash, 2002b). In the case of binary responses with a logit link function (logistic multilevel regression), there is no single VPC measure since the level 1 variance is a function of the mean, which depends on the explanatory variables in the model. Furthermore, the higher level variance is on the logistic scale whereas the individual variance is on the probability scale (Merlo, Chaix, Ohlsson, Beckman, Johnell, Hjerpe et al., 2006).

To solve these technical difficulties some alternative approaches has been suggested in the case of logistic regression (Goldstein et al., 2002b). Two of these methods are; (1) the simulation method and (2) the latent variable approach. The first converts the area level variance to the probability scale whereas the latter convert the individual level variance from the probability scale to the logistic scale. In the latter, supported by (Snijders & Bosker, 1999) the VPC is only a function of the area level variance and does not depend on the prevalence of the outcome/the individual level variance. Another measure to describe between-higher-level variation is the Median Odds Ratio (MOR). The MOR translates the higher-level variance to the odds ratio scale and has been put forward as a complementary measure to the VPC (Larsen, Petersen, Budtz-Jorgensen, & Endahl, 2000; Larsen & Merlo, 2005; Merlo et al., 2006; Merlo et al., 2009).

Some authors have most rightfully warned against undue focus on the random variation observed at higher levels (Blakely & Subramanian, 2006). A usual interpretation of insignificant and/or small higher-level variation is that there is no need to model explanatory variables at that level. Two comments may be warranted in this respect. Firstly, many studies may be underpowered in terms of the number of level 2 units, hence, they are really not suited to make such assessments (Diez Roux, 2000, 2001). Secondly, it is quite possible to find moderately strong and significant fixed higher level effects even in the absence of statistically significant between higher-level unit variation (Merlo, Lynch, Yang, Lindstrom, Ostergren, Rasmusen et al., 2003; Diez Roux, 2007b). Although intuitively strange, these are not contradictory facts because fixed and random effects give different and complementary information (Merlo, 2003).

So while a given study may have insufficient power to detect variability between higher level units (in the random part of the model) it may well have power to detect fixed effects of a specific higher level attribute (i.e. a neighbourhood attribute). A test of the association of a specific higher level attribute will often have more power than the diffuse test that the higher level variability is zero (Diez Roux, 2004a). Hence, there is no direct correspondence between amount of variation at a higher level and the extent to which explanatory variables may be found at that particular level. Indeed, higher level covariates may play an important etiological role even when there is virtually no between-higher-level variation (Bingenheimer & Raudenbush, 2004). It should however be stressed that measures of higher level variance (e.g. ICC's) and traditional measures of association (regression coefficients) *taken together* yield important information. This applies both to etiological importance as well as in prevention (see appendix 1 for a crucially important illustration in terms of area based interventions).

The context-composition framework may at first glance seem straightforward in a multilevel model; there are individuals and there are places (or other contexts). A common modelling specification strategy is to fit a basic variance component model with no covariates to get an initial assessment of the magnitude of the higher level variability (a so-called 'nullmodel'). Thereafter several individual level covariates are introduced in a second stage. This yields a new estimate of the level 2 variance component, now after accounting for the composition of individuals. Depending on the size of the remaining level 2 variance (and a statistical test of the null hypothesis that the level 2 variance is zero) the investigator may declare either composition or context. The ultimate advancement will be to move to a third stage and try to explain and model eventually remaining level 2 variation with contextual (level 2) explanatory variables.

Although this analytical strategy has intuitive appeal it is in fact attached with rather thorny inferential problems. Firstly, etiologically important individual level covariates may be omitted or those included may be measured with error, and, as a result, the prime argument of 'misspecification of the model at the individual level' (i.e., confounding) is put forward together with arguments of 'overestimation' of higher level variation. Secondly, and essentially a counterargument, the omission of level 2 covariates may cause the contribution of the level 1 covariates to be overstated. This will be the case whenever some level 2 covariate is omitted that is correlated with the dependent variable and simultaneously correlated with one or more of the individual level compositional variables at level 1. This will ultimately cause the higher level variation to be underestimated because some of the variance associated with the level 2 covariate has been inadvertently removed (Duncan, Jones, & Moon, 1999).

Thus, separating context and composition is indeed a difficult specification problem. On the one hand, omitting important individual level covariates causes the between higher-level variation (unobserved heterogeneity) to be overestimated. On the other hand, the inclusion of individual level covariates that are correlated with a higher level covariate may cause the higher level variation to be underestimated. Both these issues will apply in most applications.

The core issue, in substantive terms, is that context and composition are deeply confounded (Bingenheimer & Raudenbush, 2004).

A closely related issue in model specification of contextual, higher level effects, is the role of individual level constructs in terms of confounders and mediators. While disease is expressed in individuals, higher level effects will, at some point, have to be mediated through individual level processes. In a strict sense, group level attributes cannot affect individuals independently of all individual level attributes, but this does *not* however, imply that group level constructs are reducible to individual level constructs (Diez Roux, 2003a). The extent to which an individual level variable is conceptualised as a confounder or as a mediator depends on the particular research question, and importantly, on its underlying theoretical model. It may be difficult to differentiate between individual level covariates as confounders or mediators. If it is the latter (cf. fig. 4 c), then 'adjusting' for an individual covariate will lead to overlooking indirect cross level effects (Blakely & Woodward, 2000). A side issue worth mentioning is of course the possibility of confounding purely within the ecological level (Chaix, Leal, & Evans, 2010).

Another nagging and related problem concerns the sorting of individuals into different contextual units (Oakes, 2004). This selection issue is perhaps *the* key problem in observational studies of 'neighbourhoods and health' (Diez Roux, 2004a). Individuals may be sorted into neighbourhoods according to individual characteristics, and these individual characteristics may be related to the outcomes (Diez Roux, 2001). Hence, selection processes may place individuals with certain characteristics affecting the outcome towards particular neighbourhoods, and this will, to varying degrees, confound the association between a neighbourhood characteristic and the health outcome (Chaix, 2009). Given that the large majority of so-called 'area effects studies' are cross-sectional, such processes will continue to hamper published results. Longitudinal studies with repeated measurements of individuals as well as neighbourhoods may partially be able to circumvent these difficulties (Blakely & Subramanian, 2006), but such datasets are not widely available.

Turning to quite a different issue; a very useful outcome from multilevel models worth mentioning is that of multilevel residual mapping. While a multilevel model estimates the variances, it is possible to estimate posterior (place-specific) residuals at each contextual level (Subramanian et al., 2003). These are not the 'raw' residuals but so-called 'shrunken' residuals that take account of the clustering within units as well as the number of individuals within each unit. Consequently, neighbourhoods with few individuals and/or the presence of a small degree of clustering (low ICC) will pull their neighbourhood residuals towards zero (the mean for the total sample).

The purpose of such residual mapping is not just methodological, but clearly substantive and practical. For instance, such residual mapping has been used to assess health service performance (Duncan et al., 1998). Furthermore, in studies with 'places' as the higher level, such residuals may highlight and expose 'unusual' places. There is hence, potential for some bridge-building with researchers doing more 'intensive' research (Sayer, 1992) as they would be more able to study the distinctiveness of these places (Jones, 1993). Such insights may

again have feed-back value for the further advancement in this field of study. Crucially, a further advancement within the, broadly termed, area of 'group-level determinants and health' is contingent on a combination of intensive and extensive research (Diez Roux, 2001).

More fundamentally, multilevel models have been described as a technical means to a theoretical end – the 'place' they capture is (merely) that of a higher-level measurement employed in a particular model (Kearns & Moon, 2002). These may have little sociological significance and may say more about data availability than the realities of place effects. Nevertheless, as Kearns & Moon also note, there is indeed an aspect of 'place awareness' in studies employing multilevel modelling. These models give clear recognition to the idea of hierarchies (i.e. levels), and the nesting of people, and by allowing for considerable complexity they are more faithful to the external reality than studies conducted on a 'single level'.

In summary and given the above discussion, it is apparent that multilevel models have some clear advantages over previous single-level methodologies, be they performed on individuals or at aggregate levels. There are indeed various challenges, limitations and pitfalls, and as one of the pioneers in this area have stated; multilevel models are not a panacea (Goldstein, 2003). After going through various phases from the initial unbridled enthusiasm, followed by critical reflection, and finally more realistic assessments of the techniques value, multilevel modelling is now part of the research community's armamentarium (Duncan et al., 1998).

The core analytical value of multilevel modelling is neatly and compactly summarised by Diez Roux, and according to her, it allows: "(1) a simultaneous examination of the effects of group-level and individual-level predictors, (2) the non-independence of observations within groups is accounted for, (3) groups or contexts are not treated as unrelated, but are seen as coming from a larger population of groups, and (4) both interindividual and intergroup variation can be examined as well as the contributions of individual-level and group-level variables to these variations. Thus, multilevel analysis allows researchers to deal with the micro-level of individuals and the macro-level of groups or contexts simultaneously" (Diez Roux, 2000 p. 174).

Consequently, and with relation to the theoretical framework outlined earlier, the multilevel statistical model can differentiate between, and model, what Geoffrey Rose termed; 'the causes of within-population variability (causes of cases) and the causes of between-population variability (causes of incidence)'. The key point is that factors at multiple levels may be important to understand causes of variability within a level. For instance, both individual level and group level factors are important to understand the causes of between-population differences in disease rates. Likewise, population level and individual level factors are important to understand causes of individual disease (Diez Roux, 2004b). Also, if we reiterate figure 1, the multi-layered contextual influences, ultimately causing individual disease, can be modelled by extending the statistical framework to include various levels of influence and interactions between them, as well as interactions between the various levels of influence and individuals.

An important side-effect in substantive terms is that multilevel modelling has challenged researchers to articulate theories of disease causation that brings together factors beyond the individual level and to consider various levels of causation. It highlights the upstream causes of disease by drawing attention away from individuals' bad behaviours, and this may ultimately have bearings on prevention as well as on social and health policy (Diez Roux, 2002b). More fundamentally, and according to the paradigm-shifts in public health (Pearce, 1996), this is a reorientation back to the 'old' public health, chiefly preoccupied with how the structures of society causes disease and adverse health (Frohlich et al., 2001; Macintyre et al., 2002). This is in fact no minor achievement.

6. Fundamental issues and critique

The critique levelled against the current line of inquiry, both in terms of theories, concepts and modelling approaches is profound. In particular, the critiques from within the social sciences have been rather fundamental. The partitioning of variance components and subsequent declarations of context (place matters) or composition (individuals matters more) is quite bluntly refuted by researchers with a different epistemological hue (e.g. Shaw, Dorling, & Mitchell, 2002). The context-composition distinction is arguably artificial, and there have been calls from some notable authors some while ago (Macintyre et al., 2002; Macintyre & Ellaway, 2003), and more recently (Cummins, Curtis, Diez-Roux, & Macintyre, 2007) to completely abandon this distinction. While it may seem somewhat absurd to partition variation from a statistical model and 'declare' either contextual or compositional effects, it is nevertheless argued, that in some instances this distinction within a multilevel modelling framework may still be useful.

Macintyre and colleagues, while calling for the dismissal of the false context-composition dichotomy, introduces yet another distinction to the explanatory vocabulary; collective explanations (Macintyre et al., 2002). According to the authors, collective explanations draw our attention to socio-cultural and historical features of places and emphasises the importance of shared norms, traditions, values and interests. Collective properties of residents in a place are not seen as separate from contextual features, but the authors nevertheless find it sensible to draw attention to features of collective social functioning and practices. The general idea is to expand psychosocial constructs like social capital and social cohesion to include aspects like; ethnic, regional or national identity, religious affiliation, political ideologies and practices, legal and fiscal systems, shared histories, kinship systems, domestic division of labour, as well as age and gender roles. A number of other authors have theorised along similar lines, in particular with respect to constructs like health lifestyles (Cockerham, 2005) and collective lifestyles (Frohlich et al., 2001; Frohlich, Potvin, Chabot, & Corin, 2002; Frohlich, Potvin, Gauvin, & Chabot, 2002).

There are some additional challenges, both theoretical as well as methodological, that need to be highlighted. Many of them are closely linked to the need to develop theoretical models of

which constructs defined at different levels influence individual-level outcomes. The nesting of individual into higher level units ultimately requires some form of delineation of these higher level units. While such delineations may be obvious and straightforward for some levels of nesting (e.g. countries) they are highly problematic in others. The definition of neighbourhoods is perhaps most controversial and easily contested, and as Massey points out; they are not simple areas you can easily draw a line around (Massey, 1991). While different criteria may be used (including historical criteria, geographical criteria, residents perceptions or administrative boundaries) the key issue concerns operationalising areal units based on the *underlying process* presumed to operate on health (Diez Roux, 2003a).

Most studies of neighbourhood effects on health have used administrative boundaries and arguably as a result of convenience (Haynes, Daras, Reading, & Jones, 2007). The effect has been that widely varying neighbourhood definitions have been used, and interpretations correspondingly difficult. As previous ecological analysis has shown, the modifiable area unit problem (MAUP) can lead to different results depending on various areal definitions (Openshaw & Taylor, 1979). A systematic comparison of alternative sets of neighbourhood units in the UK showed that it does indeed matter where you draw such boundaries in terms of area effects on health (Flowerdew, Manley, & Sabel, 2008). It is hence, crucially important, to define areal units according to the hypothesised mechanism linking place to health (Kawachi & Subramanian, 2007). Neighbourhood and health studies are often underpinned by the assumption that only the 'local' matters in terms of health-damaging and health-promoting features, but as Cummins note, this may indeed be questioned (Cummins, 2007).

Closely related to this is of course the identification of area level constructs, which again should be based on a theoretical model and the specific hypothesis being tested. This essentially concerns developing an operational definition and measurement of the relevant area level construct (Diez Roux, 2003a). Here it is possible to distinguish measures between "true" area level constructs (termed either as 'integral', 'primary' or 'global') from those derived from individuals (termed either as 'derived', 'analytical' or 'aggregate') (Diez Roux, 2003a). While many researchers have used available 'off the shelf' secondary-data proxies for their constructs, there has been some advancements in this area the last few years (Cummins, Macintyre, Davidson, & Ellaway, 2005; O'Campo & Caughy, 2006). Both technical advancements in terms of ecometrics (Raudenbush & Sampson, 1999; Raudenbush, 2003; Mujahid, Diez Roux, Morenoff, & Raghunathan, 2007), but perhaps more importantly, theoretical frameworks have been put forward that conceptualise the nature of neighbourhoods as they relate to the production of health inequalities (Bernard, Charafeddine, Frohlich, Daniel, Kestens, & Potvin, 2007).

As already noted, the issue of health selective geographical migration is a difficult issue with respect to place effects. Using place deprivation as an example, a central question becomes whether a relationship between deprivation and health indicates that living in a deprived area causes individuals' health to deteriorate. Alternatively; are those in poor health more likely to migrate towards, and less likely to move away from a deprived area, than those in good health? We cannot always be sure that the environment in which they lived at the time of illness or death was the one that caused health deterioration (Boyle, 2004; Boyle & Duke-

Williams, 2004). This is especially important given the growing recognition of life course influences whereby the health of individuals may be influenced by various factors at different time points in life (Kuh & Ben-Shlomo, 2004). The problem so far has been that the majority of studies have ignored the simple fact that individuals move around.

It has been acknowledged for quite some time that migration is not a random process (Boyle, Halfacree, & Robinson, 1998). Individuals' propensity to migrate may vary according to demographic and socioeconomic markers as well as those of health. Studies from the UK have shown that migrants in general are healthier than non-migrants, although those moving short distances tend to have higher mortality than those moving long distances (Boyle, Norman, & Rees, 2002) an elderly people are more likely to move if they are ill than if they are well (Bentham, 1988). Studies of adolescents in Norway have shown that rural-to-urban migration has a clear social class structure (Rye, 2006). Given our knowledge of the relationship between health and social class this migration will, by its very definition also be health selective, although it should be noted that the SES-health-gradient, in general, are less pronounced for adolescents than for middle aged individuals.

Just as health selective *social mobility* may widen (or narrow) the health gradient between social classes, *geographical mobility* may widen (or compress) geographical inequalities. Migration thus complicates research in two distinct ways; firstly, and in a descriptive sense, by continuously redrawing the map of geographical health variations, and secondly, in a causal or etiological sense, by obfuscating any relationship between place of residence (i.e. place deprivation) and the health of individuals. As Diez Roux have noted on this second issue; selective geographical mobility is *the* major problem in neighbourhood effects studies (Diez Roux, 2004a).

Smith and Easterlow have taken issue with this last statement and have put forward a rather wide-ranging and fundamental critique of area effects research and contextual explanations in general (Smith & Easterlow, 2005). They contend that health geographers studying place effects on health has produced a strangely one-sided geography, and that the geography of health has come to hinge almost exclusively around the question of whether "place matters for health". Furthermore, that there is undue and somewhat unwarranted focus on "context", while "composition" is neglected or seen as a mere nuisance (as the quotation by Diez Roux suggests). Smith and Easterlow argue for more attention on the compositional aspects, where the aim is to;"direct attention to the way that health histories and conditions are powerfully entangled with people's trajectories into, within, and out of, different spaces and places" (Smith & Easterlow, 2005 p. 185). They contend that health inequalities in society arises through various discriminatory practices associated with selective-, entrapment, exclusion and displacement. Hence there is also another side of health geography, a side where socially structured and institutionalized processes accumulated across the life course are systematically differentiating populations according to health histories and prospects.

Besides the issue of migration, another complicating matter in area effects research relates to how places themselves are continuously transformed. Not only do people move around, but places in which people live may also change and consequently can individuals experience

different levels of e.g. place deprivation without migrating at all (Boyle, Norman, & Rees, 2004). This is partly a consequence of the histories of places, how they relate to each other, as well as to how places relate to broader macro-structural factors (Tunstall et al., 2004). Much previous research has treated places like fixed entities with enduring characteristics which they obviously are not.

Closely related, it is also important to acknowledge the time-lag from an area exposure to the health outcome. Many studies are cross sectional, and as Blakely points out, this implies a zero lag-time between exposure and outcome and such an instantaneous effect is usually implausible (Blakely & Woodward, 2000). If the ecological exposure is stable through time this is less of a problem, but given the possibility that places can change through time it is likely that such exposures (along with other sources of bias) may render ecological exposures that are grossly misspecified (Diez Roux, 2008). While not addressing the problem of ecological time-lags specifically, a study from the UK provides some circumstantial evidence. Curtis and colleagues found in a longitudinal study that those who grew up in disadvantaged areas had higher relative risk of reporting illness and experience death, even after adjusting for current place of residence (Curtis, Southall, Congdon, & Dodgeon, 2004). Leyland and Næss did similar investigations with Norwegian data, and found current area of residence to be most influential for the mortality in the youngest age cohorts (Leyland & Næss, 2009).

Consequently, just as the relevant spatial scale may differ depending on the outcome, also the time frame may differ for various outcomes (Diez Roux, 2007b). To exemplify; whereas the impact of neighbourhood conditions on cardiovascular disease may require a long exposure to adverse neighbourhood conditions, the effects of the built environment on physical activity may occur much more rapidly because of the ability of behaviour to change in response to exposures.

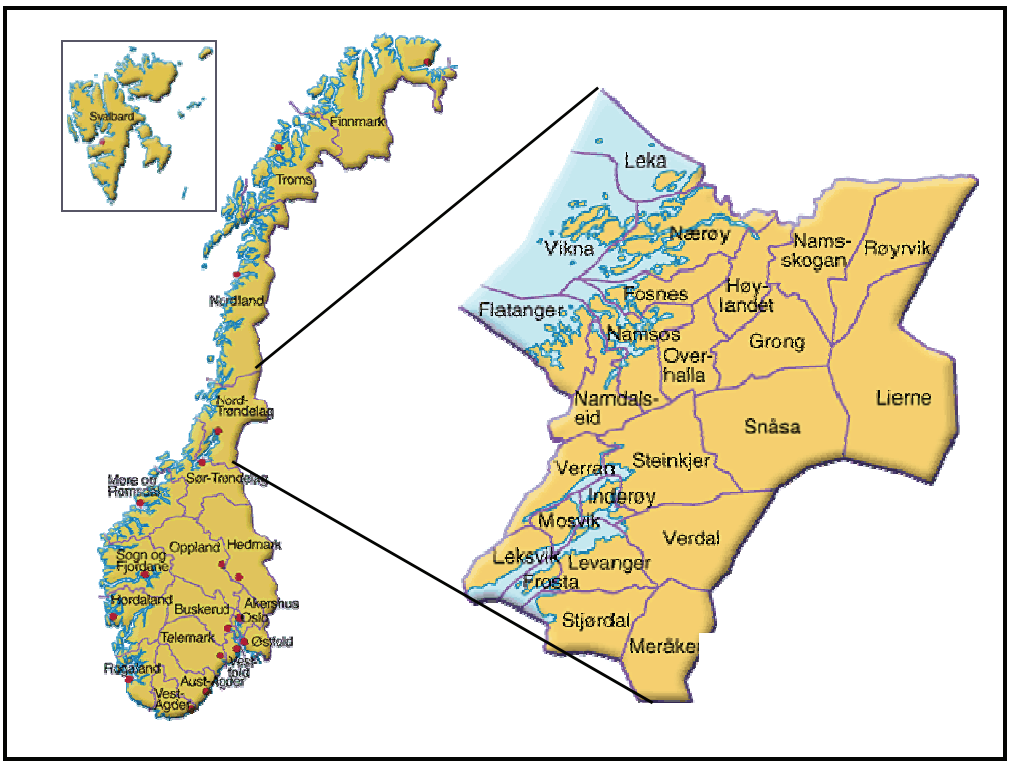
Two final issues should be mentioned. Firstly, the neighbourhood constitutes only one of multiple contextual units from which individual health may be influenced. For some groups of individuals it may make a difference whereas for others there may be other more important settings in which the health is influenced. Secondly and fundamentally, the pathways linking group level constructs to individual health are complex and involve reciprocal causation and feedback loops (Diez Roux, 2004b). Indeed, such dualism between individuals (agency) and society (structures) is a cornerstone in the proposed ontological framework. This clearly underscores the artificial, or simplistic distinction between composition and context because while neighbourhood features may influence individual health, neighbourhood features are simultaneously being shaped by the individuals (Diez Roux, 2008). The introduction of ‘complexity theory’ in health geography may be a promising avenue in which further advancements within this field can be made (Gatrell, 2005; Curtis & Riva, 2009). This is recently acknowledged within epidemiology as well (Diez Roux, 2007a; Galea, Riddle, & Kaplan, 2010). But as Gatrell notes, whether complexity theory constitutes an advancement over previous ‘systems theories’ still remains to be seen (Gatrell, 2005).

7. Study area, data material and ethics

Study area

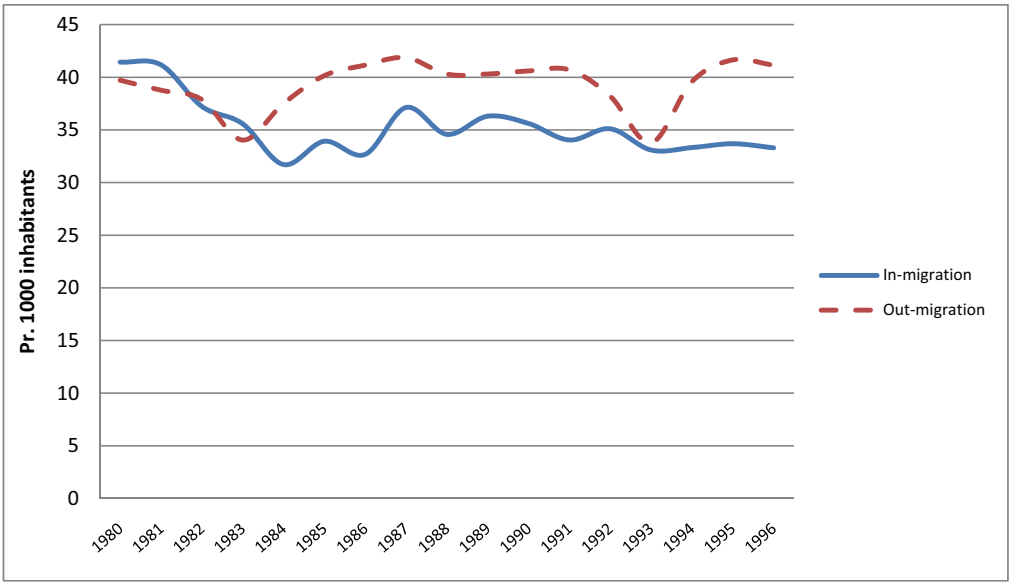
As one of 19 counties in Norway, Nord-Trøndelag is located in the central part and consists of 24 municipalities (Fig 5). The population size has been quite stable in the period 1984-1997 with about 127 000 inhabitants. With some exceptions concerning ethnic homogeneity, no major cities, and income and educational levels slightly below the average for the country, Nord-Trøndelag is *generally* considered representative for the whole of Norway (Krokstad, 2004).

Figure 5 Norway and the Nord-Trøndelag County



Geographical patterns of mobility in Nord-Trøndelag are illustrated in figure 6. The figure shows average migration rates for the 24 municipalities during the period 1980 to 1996 (data extracted from the NSD's 'regional database' maintained by Norwegian Social Science Data Services). At the start of the period an average of 40 per 1000 inhabitants migrated out of their municipality and approximately the same rate migrated into the municipalities. At the end of the period in 1996 the out-migration patterns were rather similar whereas in-migration was somewhat lower than in the beginning of the period. These between-municipality mobility patters are similar to that for Norway (see e.g. Østby, 2002).

Figure 6 Migration rates 1980 to 1996 in Nord-Trøndelag (annual averages for 24 municipalities)



Data material – the Nord-Trøndelag Health Study (HUNT)

The Nord-Trøndelag health study (HUNT) is a population based health survey and has been carried out in three waves; HUNT 1 in 1984 to 1986, HUNT 2 in 1995 to 1997 and HUNT 3 in 2006 to 2008. Data from the last survey were not available during the course of the current project and are not described here. All inhabitants aged 20+ years were invited to the two first surveys and among eligible respondents, 74, 977 (88.1 %) and 66,140 (71.2 %) attended HUNT 1 and HUNT 2 respectively. Attendance rates varied considerably with age and women had better attendance than men in both surveys (Holmen, Midthjell, Bjartveit, Hjort, Lund-Larsen, Moum et al., 1990; Holmen, Midthjell, Krüger, Langhammer, Lingaas Holmen, Bratberg et al., 2003).

A two-step procedure for data collection was used, whereby an invitation letter for a clinical examination including a self administered questionnaire (Q1) was sent to each eligible individual. Participants attending the clinical examination were subsequently given a second questionnaire (Q2) to be returned by mail. Similar procedures were used in both surveys, although HUNT 2 was more comprehensive than HUNT 1 and also had somewhat tailored questionnaires for different age groups as well as for gender. Some 45 000 individuals participated in both surveys. A more comprehensive description of HUNT 2 is attached below (appendix 2; Holmen et al., 2003), and questionnaires for both surveys is available for download at the HUNT website (http://www.ntnu.no/hunt/english/data/que). HUNT data was linked to national registry data in the current project (article 3 & 4). This linkage is made possible through the unique Norwegian 11-digit personal identity number and was performed by the HUNT research centre, and the current project utilised anonymous data. The national

registries contain longitudinal information on numerous sociodemographic features and are described extensively elsewhere (Akselsen, Lien, & Sivertstøl, 2007).

Ethics

The HUNT study has been approved by the Regional committee for medical research ethics. The exception is HUNT 1 in which this institution was not yet established. Approval of data linkages to national registries was given by the Norwegian Data Inspectorate. Participants in HUNT 2 provided written consent for the usage of data in medical research and have a standing opportunity to withdraw this consent at any time. The current project used anonymous data and the protocol was approved by the Regional committee for medical research ethics (REK).

8. Summary of articles

Social capital: The glue that keeps public health together? (Paper 1)[1]

The concept 'social capital' concerns social relations and is usually described as the 'glue' that holds a network or a society together. This glue is made of both trust and the shared norms which develop in such networks. In common with other forms of capital, such as economic, cultural and human capital, social capital is regarded as a *resource* for both individuals and societies. However, this resource is not linked to individuals in the way that more traditional research on networks understands it. Rather, theories on social capital form the basis for a more holistic understanding of how individuals develop and are influenced, socialize and obtain support. This occurs through dynamic interactions between different actors, cultures, environments, and societies. Thus, in order to understand social capital as distinct from social network theory, a system theoretical approach is advantageous as it works on the basic assumption that the whole is more than the sum of its parts (Bø & Schiefloe 2007).

The concept of social capital is used in various disciplines in order to understand different phenomena and is probably better described as a perspective rather than a theory. In common with other 'new' concepts, social capital is the subject of debate, and this is both a methodological as well as a normative debate.

It is claimed that there are several positive effects of social capital in a society, including a well functioning democracy, a high level of welfare, low levels of criminality, economic development, and a good public health. The aim of this chapter is to examine this phenomenon with a particular emphasis towards public health. What is social capital? How does it affect health? What do we know about empirical relations between health and health behaviour? What value does social capital have regarding efforts to improve public health and reduce social inequalities in health?

[1] The text is perhaps better described as an *introduction* to the book section rather than a traditional summary

The influence of social capital on self-rated health and depression – The Nord-Trøndelag health study (HUNT) (Paper 2)

Introduction: The article examines the relationship between neighbourhood social capital and two health outcomes: self-rated health and depression. A total of 42,571 individuals aged 30–67 years participated in a cross-sectional total population health study in Nord-Trøndelag in 1995–1997 (HUNT II) and were investigated using multilevel modelling. The first aim was to investigate potential area effects after accounting for the characteristics of individuals in the neighbourhoods (N = 155). The second aim was to explore the relationships between contextual social capital (the level of trust at the neighbourhood level and the level of local organizational activity), and the two health measures.

Results: Models with stepwise inclusion of individual level factors attenuated the ward level variance for both self-rated health (PCV: 41%) and depression (PCV: 43%). The inclusion of the two contextual social capital items attenuated the ward level variance for both self-rated health and depression to varying degrees. At the individual level, contextual social capital was associated with both self-rated health and depression. Individuals living in wards with a low level of trust experienced an increased risk of 1.36 (CI: 1.13-1.63) for poor self-rated health compared to individuals in wards with a high level of trust. For depression, this effect was even stronger (OR: 1.52, CI: 1.23-1.87). The associations with the level of organizational activity were inconsistent and weaker for both health outcomes.

Conclusion: Geographical variations in self-rated health and depression are largely due to the socio-economic characteristics of individuals. Nevertheless, contextual social capital, expressed as the level of trust, was found to be associated with depression and self-rated health at individual level.

Individual, family, and area predictors of BMI and BMI change in an adult Norwegian population: Findings from the HUNT study (Paper 3)

The global obesity epidemic is a major public health concern and there is strong evidence that the drivers are varied and operate via diverse pathways. Taking a systems approach allows the contextual influences operating upon the individual to be identified and quantified. We adopt such a perspective in this study, where longitudinal data from a cohort of 24,966 settled individuals participating in two major health surveys, the Nord-Trøndelag Health Study (HUNT 1 and 2) in the county of Nord-Trøndelag, Norway, were used to investigate associations between individual, family and area characteristics and two outcomes: body mass index (BMI) at follow-up and BMI change over an 11 year period. Linear multilevel models were fitted, with individuals nested in 17,500 families, 447 wards and 24 municipalities. A range of putative individual, family, and area predictors were tested. We found both outcomes were strongly associated with individual characteristics, with higher BMIs generally being amongst males, unmarried participants, non-smokers, those of lower education and those undertaking physically demanding work but participating in less physical activity outside work. The characteristics of those in the sample exhibiting higher BMI gain were rather similar except that women gained more and those with no employment income gained less.

Contextual influences were also found to be important; although just 1 % of the unexplained variance was located on the neighbourhood and municipality levels respectively, and hence suggesting small environmental influences, between 10-13 % could be attributed to families, highlighting the importance of the familial contextual environment. Rather little is known about the manner by which family influences may operate on bodyweight hence further work is needed to understand likely mechanisms and guide future interventions.

The impact of family and place of residence on smoking behaviour – the Nord-Trøndelag Health Study in Norway (Paper 4)

Tobacco control initiatives have steadily lowered the smoking prevalence in developed societies but further reductions seem increasingly difficult to achieve. A clear socioeconomic gradient in smoking is also evident in societies in the so-called matured stage of the smoking epidemic. The aim of the present study is to highlight new avenues for future tobacco prevention by investigating the sociodemographic, family and geographical distribution of smoking among adults in Norway. We utilise cross-sectional total population health data from the Nord-Trøndelag Health Study (N=50,535), and deploy multilevel models of individuals nested in families, wards and municipalities. We found that smoking was strongly correlated within families (ICC=39.1%, MOR=4.11) whereas the correlation within wards (ICC=1.07, MOR=1.26) and municipalities (ICC=1.12, MOR=1.27) was small. Smoking was strongly and inversely patterned by education with an odds ratio of 5.5 (95% CI 4.81 to 6.40) between highest vs. lowest category. Smoking was more prevalent among those unemployed/unfit to work, retired/on social security compared with being self employed and among divorced/separated compared with married individuals. There was a tendency towards higher risk of smoking in lower income families and relatively deprived municipalities. We conclude that families and households are important contextual units to understand smoking and this should be acknowledged in future tobacco prevention and policy. Secondly, the strong educational gradient in smoking suggests that future tobacco control efforts should have an explicit equity perspective.

9. Discussion

Main findings

A number of conclusions arise from the four papers in this dissertation. Firstly, for the outcomes investigated, the amount of variance associated with administrative units in Nord-Trøndelag is rather small. This applies both to the municipality level and more surprisingly to the ward level. It was expected, a priori, to discover somewhat larger amounts of variance at the ward level than at the municipality level. These higher level variances, whilst being small, were partly explained by the contextual constructs of social capital and place deprivation. The exception is for BMI where both constructs were unrelated to the outcome. Secondly, the amount of variance associated with the family level varied considerably in magnitude; a fairly modest portion was associated with BMI whereas a substantial amount was associated with daily smoking. The indicator of family SES was unrelated to BMI, but for smoking some of this family variance was explained. Thirdly, the outcomes in all the papers was inversely related to individual level education, particularly so for smoking. Fourthly, in the theoretical/review paper of the social capital constructs health-promotive salience, it is found that the Scandinavian empirical evidence of meso-level associations is not convincing. These findings have been substantiated in a later systematic review of social capital and health studies (Sund, 2010). Furthermore, social capital's diverse theoretical underpinnings lend the construct to widely varying interpretations, and in effect, to very diverse policy responses.

A comparison with international findings

It is not entirely straightforward to compare the current findings with international results. This is particularly true with respect to the unexplained variance at the municipality and ward levels. As noted in some of the most recent reviews of area effects: area effects on health are significant in most studies – but they depend on the outcomes under study, the area construct used, and importantly, on the spatial scale at which associations are studied (Riva et al., 2007). Another issue making comparisons difficult is the fact that many studies do not report clustering or between-higher-level variability (Merlo, 2003; Merlo et al., 2009). In two recent reviews, a consistent finding from societies with a high degree of egalitarianism was that they reported small higher level variances (Kim et al., 2008), typically with ICC's ranging from 0-2% (Islam et al., 2006). The findings presented in this thesis are very much in accordance with these two reviews with respect to fairly small variances associated with place of residence. With respect to the family level variance, there is a scarcity of studies to make comparisons with as this is somewhat of an emerging strand within social epidemiology (Merlo, 2010). There is most likely a multitude of studies from within sociology and psychology using similar approaches to study family influences on individual health, but this research area is admittedly not rigorously reviewed here.

Limitations

The cross sectional design in two of the papers is a weakness, hence the direction of causality is impossible to determine. In other words, for some of the variables, it is difficult to discern whether the explanatory variable affects the outcome or the other way around. Furthermore, the study area may be too geographically homogenous to appropriately study area effects on health. It is a *sine qua non* in any field of study that to detect an effect there must be sufficient variation in the exposure (and the dependent variable). So while the study population *in general* is considered representative, there may also be certain aspects, like small geographical heterogeneity, absence of highly urbanised and segregated areas that makes the study area less suited to study area effects. Related, and with respect to the municipality level, the analyses may be slightly underpowered as the number of higher level units is small (N=24). It is also important to reiterate one of the core assumptions in multilevel modelling described earlier; that the higher level units is treated like a sample coming from a population of higher level units. Also crucially important; delineating neighbourhoods through the usage of administrative boundaries is far from ideal, although this limitation is shared by the large majority of the research community within this field of study. Neighbourhoods are generally grossly misspecified in most, if not all studies (Diez Roux, 2008), and may partly explain modest and small portions of variance associated with this level.

Moreover, the operationalisation of the neighbourhood level constructs may be challenged. Improving the measurement of higher level constructs is greatly needed for this research area to make further advancements. Also, the issue of confounding (a mixing of effects whereby the association of an exposure with an outcome is distorted by an extraneous factor) both within and across levels is always an issue with observational data. As discussed above, it is particularly difficult to differentiate between confounders and mediators in multilevel research.

Finally, the migration issue poses interpretational challenges with respect to all the levels utilised in the empirical papers. A longitudinal study from the UK found increased variability at the household level when accounting for changes in household membership whereas the opposite was true for the between-area migration and the area variability (Chandola, Clarke, Wiggins, & Bartley, 2005). Evidence from experiments, that accounts for the issue of selective migration through randomization, and hence allow firmer causal inference, suggests that mental, but not physical outcomes, is associated with neighbourhood poverty (Kling, Liebman, & Katz, 2007). Some have noted that there are important limitations attached to this experiment (Diez Roux & Mair, 2010), but the study nevertheless provides important circumstantial evidence of causal neighbourhood effects.

Strengths

What the study area may lack in geographical heterogeneity is strongly outweighed by the data material used in this thesis. HUNT is one of the largest health surveys in the world; it has high participation rates, and is *in general* considered representative for the larger society. Linked to national registry data there are not many researchers internationally equipped with

similar high quality data. As a recent inventory has shown; some 50 PhD theses and hundreds of publications have been based on this material (HUNT, 2009).

The multilevel statistical framework, comprehensively described above, is a clear advancement compared to previous studies conducted on a single level. It allows both the exploration, description and, to some extent, explanation of the importance of higher level factors on individual health. It has both substantive as well as methodological advantages, and as some of the multilevel modelling pioneers have noted; this statistical framework is more 'true to the social reality' (Jones, 1993). Recent developments in structural equation multilevel modelling (see e.g. Rabe-Hesketh & Skrondal, 2008a; Muthén & Asparouhow, 2010) are likely to advance this field of study even further (Curtis & Riva, 2009).

Policy implications and further research

Policy implications

Targeted area-based initiatives according to administrative boundaries do not seem especially warranted in Nord-Trøndelag. This statement does however urgently need some clarification. Firstly, it applies to the particular outcomes investigated in this thesis. Secondly, these findings must also be interpreted in time as well as in space – they do not preclude area based initiatives in other rural parts of Norway, in more urban settings, or in the years to come. There are, however, several caveats attached to such targeted area-based initiatives. As Curtis (2004) notes, areas with the largest proportion of people in poverty and poor health do not contain the majority of people who are deprived. Hence, initiatives focused on particular areas will miss a large proportion of those experiencing adverse wealth and health. Indeed, such initiatives may in fact divert attention away from the fundamental structural factors in the political economy that is continuously reproducing a socioeconomic health gradient between social classes. As Hayes notes; while leading causes of death have changed, gradients have remained (Hayes, 2004) – gradients are rooted in societies modes of production.

Furthermore, area based initiatives may simply produce an appearance of action in a limited number of areas where the adversities are most pronounced, and at a lower cost that would be necessary to fully eradicate poverty (Curtis, 2004). Some authors see such initiatives as convenient for 'third way' social policies (Powell & Moon, 2001) and some argue that the social capital construct fits nicely in such programmes (Muntaner, Lynch, & Smith, 2000; Navarro, 2003). The bottom line is that targeted area based initiatives should in general be treated with caution (Shaw, Dorling, Gordon, & Davey Smith, 2001).

It is however crucially important to underline that health promotion or prevention may still be most appropriately issued according to areas or administrative units, but they should not be in the form of targeted interventions. This is an important distinction – administrative units like municipalities still constitutes convenient units for preventive efforts.

While targeted area based initiatives may have limited value in disease prevention and health promotion in this particular study area, some of the findings suggest that the family or household may be an important unit for health intervention. The simple fact that e.g. health behaviour is clustered in families or households should urgently be acknowledged in research as well as in preventive efforts. It should however be noted, that targeting families is a so-called 'high risk approach', and this raises a number of ethical aspects.

Further research

Despite many decades of neighbourhood and health research, it seems like this area is still trying to mature. The theoretical underpinnings are indeed present, but as should be evident by now, the inbuilt complexities are huge indeed – reality is after all messy. In order to advance this field further, it may be salient to utilise theories and associated mechanisms that are more specific rather than "catch-all" concepts like social capital (usually very eclectic operationalised). In order to illustrate; obesity has been linked to the concept of social capital and those may very well be associated (Kim et al., 2008). It would perhaps, be more transparent and replicable to test more specific opportunity structures related both to diet (Brug, 2008; Brug, Kremers, Lenthe, Ball, & Crawford, 2008) and physical activity (Stafford, Cummins, Ellaway, Sacker, Wiggins, & Macintyre, 2007). As others have noted, a specific approach is one that conceptualise, operationalise, and measure associations between specific health outcomes and specific area exposures – across specific spatial units (Riva et al., 2007). Such approaches will provide the basis for more precise area definitions and measures of ecological exposures and improved delimitations of area contours.

Families and households constitute an important area of research in years to come. This socio-relational contextual unit (or level) have received scarce attention within social epidemiology and this is surprising (Merlo, 2010). There is obvious statistical dependence attached to this level, and the failure to address such clustering may produce erroneous results. Importantly, one may also miss important matters of substance. As Merlo notes (2010); it is very likely that many individual differences in risk factors for disease can be explained by the general genetic and shared environmental backgrounds of families. The three consecutive HUNT studies are very well suited for such longitudinal family-based multilevel studies since they allow the disentanglement of genetic from shared and non-shared environmental components of variance. This avenue of research is perhaps not to be found within health geography, but may prove to be of great importance for the further cross-disciplinary understanding of the socioeconomic health gradient.

The major finding in this thesis, that place of residence seems to be of very modest importance for individuals' health, is a somewhat depressing finding from a geographers point of view. After all, we would like to argue that place matters for health. That said, this finding is also encouraging because it suggests that all the efforts laid down in regional policies has been successful in terms of securing equal opportunities to lead a healthy life.

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Paper 1

Social capital: The glue that keeps public health together?

(translated from Norwegian)

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Social capital: The glue that keeps public health together?

The concept 'social capital' concerns social relations and is usually described as the 'glue' that holds a network or a society together. This glue is made of both trust and the shared norms which develop in such networks. In common with other forms of capital, such as economic, cultural and human capital, social capital is regarded as a *resource* for both individuals and societies. However, this resource is not linked to individuals in the way that more traditional research on networks understands it. Rather, theories on social capital form the basis for a more holistic understanding of how individuals develop and are influenced, socialize and obtain support. This occurs through dynamic interactions between different actors, cultures, environments, and societies. Thus, in order to understand social capital as distinct from social network theory, a system theoretical approach is advantageous as it works on the basic assumption that the whole is more than the sum of its parts (Bø & Schiefloe 2007).

The concept of social capital is used in various disciplines in order to understand different phenomena and is probably better described as a perspective rather than a theory. In common with other 'new' concepts, social capital is the subject of debate, and this is both a methodological as well as a normative debate.

It is claimed that there are several positive effects of social capital in a society, including a well functioning democracy, a high level of welfare, low levels of criminality, economic development, and a good public health. The aim of this chapter is to examine this phenomenon with a particular emphasis towards public health. What is social capital? How does it affect health? What do we know about empirical relations between health and health behaviour? What value does social capital have regarding efforts to improve public health and reduce social inequalities in health?

1. The social capital concept in health inequality research – a brief history

Social capital is often described as a new concept and it has been used increasingly since the beginning of the 1990s. The ideas that it builds on are nevertheless not new, and the concept has a long history. Well-known but very different social scientists, such as Durkheim, Marx, Weber, Tönnies, Granovetter and Toqueville, have described phenomena which are to varying degrees consistent with the social capital concept. In recent years, this primarily concerns the sociologists Pierre Bourdieu and James Coleman, and in particular the American political scientist Robert Putnam, following the publication of *Bowling Alone* (Putnam 2001a). In *Bowling Alone*, Putnam gives a powerful and above all metaphorical account of the trends taking place in North American society. Almost all indicators of social capital point in the same direction: less civil engagement, diminishing trust between people, and waning support for voluntary organizations. Individualization is the keyword, and according to Putnam the reason for this is the rising generations' lack of collective orientation. Further, he adds that television shares part of the blame for lack of civil engagement. Urbanization has also contributed by breaking down the links between workplace and local communities to the extent that previously closely integrated communities have disintegrated.

In order to understand why the concept has gained support within research and politics we have to return to the publication of the book *Making Democracy Work* by Putnam and colleagues (Putnam et al. 1993). Based on research in Italy, Putnam et al. found large regional variations in the democratic traditions, whereby the northern regions fared better than the southern ones both politically and economically. The authors explained these variations in terms of differences in civil society: in the north, societies are characterized by traditions of voluntary organization and social networks which nurture the development of trust and common norms, consequently they have large stocks of social capital, while the opposite is the case in the south.

The 'social capital' concept was introduced into the public health debate especially by the British social epidemiologist Richard Wilkinson. Based on both his own research and that of other researchers (e.g. Putnam) Wilkinson (1966) claimed that in the Western world the countries with the most egalitarian distribution of income have the best public health records, not necessarily countries displaying the highest level of affluence (measured, for example, in terms of GDP). According to Wilkinson there are two main reasons for this. First, relative poverty makes the psychosocial stresses of living in a society with great differences in people's status worse, not just for marginalized groups but also for the population at large. The second reason he put forward is that the level of income inequality determines the level of social capital in a country. In contrast to Putnam, who predominantly pointed to civil societies' varying ability to work together, Wilkinson claims that social capital (which is defined here as *social cohesion*) in partly a consequence of the unequal distribution of material resources within a country. His core message being that large inequalities in income break down the social structures.

Consequently, when it comes to understanding *the causes* of geographical variations in social capital, it is clear that the respective authors take quite different approaches. While they fundamentally agree on what constitutes the components of this social glue, they differ greatly on what constitutes the solvent. Against this background, we can now look more closely at selected definitions from the perspective of several disciplines.

2. Definitions and related concepts

Currently, there is no general agreement on the definition of social capital as a concept, and this is partly due to social capital being regarded as both an individual-level factor (micro-level) and a system-level factor (meso- and macro-level). Somewhat simplified, we can therefore start by saying that social capital at micro-level refers to resources which are available to each individual in a network. On a system level, social capital are those relationally embedded resources which are important for a *system*'s quality and ability to function. It is, of course, problematic that several different definitions exist, but nevertheless two distinct traditions, or directions, can be identified based on the work of the sociologists Bourdieu (Bourdieu 1986; Bourdieu & Wacquant 1992) and Coleman (Coleman 1988; 1990).

For Bourdieu, social capital is primarily linked to *the individual*, and together with other forms of capital (economic, cultural, and symbolic) they determine the position of individuals in different 'social fields'. Bourdieu emphasizes that these forms of capital are interchangeable, and hence they cannot be understood as independent of each other (Portes 1998). By this statement Bourdieu claims that individuals who are in possession of social capital will more easily have access to other forms of capital and the ultimate result is an unequal distribution of economic capital.

Bourdieu's definition of the concept (Table 1) can be broken down into two elements: first, *social relations*, which make it possible for individuals to access resources from others in a network; and second, the *reserves* and *quality* of these resources. Further, when individuals have access to resources through others it can also be as a consequence of a form of investment (conscious or non-conscious) made earlier. Part of the core of Bourdieu's understanding is also *the mutual dependence* between different forms of capital. Further, he claims that *unequal access to all forms of capital* contributes to the reproduction of socioeconomic inequalities and power differences. If one has much of one form of capital, one also has (access to) many of the other forms of capital. Bourdieu's theory and analyses therefore appear to have a clear power perspective, where social capital exists within socially homogenous groups.

Table 1 Definitions social capital

Author	Definition
Pierre Bourdieu, 1986	'Social capital is the sum of resources, actual and virtual, that accrue to an individual or group by virtue of possessing a durable network of more or less institutionalized relationships of mutual acquaintance and recognition.' (p. 119)
James Coleman, 1990	'Social capital is defined by its function. It is not a single entity, but a variety of different entities having two characteristics in common: They all consist of some aspect of social structure, and they facilitate certain actions of individuals who are within the structure. … [S]ocial capital is productive, making possible the achievement of certain ends that would not be attainable in its absence.' (p. 302)
Robert Putnam, 1993	'Social capital … refers to features of social organization, such as trust, norms, and networks, that can improve the efficiency of society by facilitating coordinated actions.' (p. 167)

Sociologist James Coleman sees social capital as a resource which lies within *social structures*, or more specifically in the relations between the actors (Table 1). These resources originate from social relations and can be beneficial for *both* individuals and society alike. Coleman's definition implies that the resources, which also occur on different levels, can be used to achieve different goals. Defined in this way, social capital also has a productive element, it gives returns: 'Just as physical capital and human capital facilitate productive activity, social capital does as well' (Coleman 1988, p. 101). When social capital is available, individuals can make achievements which otherwise would not be possible in its absence, and at meso- and macro-level the returns will be manifested in the form of a more secure and well-functioning society. Also, different types of social capital exist depending upon what structures are studied: '(Social capital) is not a single entity … A given form of social capital that is valuable in facilitating certain actions may be useless or even harmful for others' (Coleman 1988, p. 98). We will return to this point later in the discussion.

It is not a simple matter to compare Bourdieu and Coleman – in fact, they have widely different aims regarding social capital as a concept. One of Coleman's main points is that by using this concept one has the ability to identify the link between individuals' rational actions and their participation in wider social structures (Bø & Schiefloe 2007). As such, Coleman's theory has resemblance with an ontology, and this is somewhat surprising given that Coleman definitely falls within a rational actor paradigm. However, Coleman's intention is also to transcend the traditional actor–structure dichotomy, and he basically tries to combine (or fit) his individualistic methodology with social structures in order to be able to explain social action. This in turn opens up the possibility for defining social capital on different levels, namely individual-, meso- and macro-levels. Consequently, Coleman has far greater theoretical ambitions for his social capital concept than Bourdieu. For Bourdieu, social capital merely forms a part of a much larger concept apparatus (in fact, social capital constitutes a fairly subordinate part).

Coleman's definition is criticized for being too wide (Portes 1998; Hjellbrekke 2000). He is also accused of not fully clarifying the processes which transform social resources into social capital, and consequently the concept is not very well suited for differentiating between which are social capital relations, and which are not (Hjellbrekke 2000). Another important objection is that Coleman's concept inadequately covers how social networks can both exclude and include – as such, it would be rather weak if one wanted to see how this form of capital is distributed within and not least between groups on the basis of power, status, economic resources, ethnicity, etc. With respect to Bourdieu, it is somewhat paradoxical that, given his hard attacks against economists' rational choice models, he himself writes about 'strategies' and 'strategic behaviour' and about forms of capital which are 'invested' in ways of acting in social life. Nevertheless, and regardless of the strengths and weaknesses of the respective authors' work, it is predominantly Coleman's concept and definitions of social capital which is developed further.

Putnam defines social capital on the basis of Coleman's definition and he focuses especially upon *networks* and *trust* as important building blocks (Table 1). These represent respectively the structural and collective (also called the cognitive) components of the social capital concept, and it is primarily the structural component that is emphasized in Putnams's work. Putnam considers social networks to be a prerequisite for individual's ability to develop trusting relationships and there is a mutual relationship between the two. At the societal level, social capital will be reflected in the form of generalized reciprocity where individuals make contributions without necessarily expecting something in return. Such 'altruism' thus acts a lubricant in economic activities because trust reduces the risk in transactions (i.e. trust implies less risk). Additionally, the ability to reach collective solutions is easier in societies which are characterised more by trust than mistrust. In sum, social capital makes society more robust and capable of functioning (Bø & Schiefloe 2007).

Putnam also makes a distinction between *bonding social capital* and *bridging social capital*, which are different *types* of social capital. These two types are not mutually exclusive but exist together in varying degrees. *Bonding social capital* refers to social capital that exists between individuals in a socioeconomically homogenous group, such as between family members or between close friends and neighbours. These forms of social relations are also described as bonds. *Bridging social capital* describes the type of social capital which connects different social groups or geographical areas. This differentiation is also found in Granovetter (Granovetter 1973) when he distinguishes between strong and weak relations, e.g. between 'thick' and 'thin' trust. Putnam has emphasized that trust is not part of the definition of the social capital concept but that it can be regarded as an indicator of social capital (Putnam 2001b). He is especially concerned with networks which develop in voluntary organizations and the trust that is created within them: 'the more we connect with other people, the more we trust them, and vice versa' (Putnam 1995, p. 665). Through the contact that is established in such organizations are developed common norms, trust and understanding, and in turn this forms the basis for cooperation and common action. Thus, for Putnam it is especially bridging social capital (also referred to as 'open networks') which is important when he argues for the

importance of the social capital concept for accounting for variations in democracy and economic development.

More recent theoretical developments have added a third type of social capital which is a further refinement of bridging social capital, namely *linking social capital* (Szreter & Woolcock 2004). This refers to the social capital which exists (vertically) between social institutions and groups of individuals. However, Putnam has expressed doubts as to whether such differentiation is productive (Putnam 2004), yet it can be claimed that it is this type of nuancing that is needed – an important part of the charges against social capital is that (with the exception of Bourdieu's definition) it neglects power relations, stratification and social polarization. However, issues of power and material distribution are far more integrated in the related concepts which are briefly described in the following.

Related concepts

The concept '*social cohesion*' partly overlaps the social capital concept. It is understood as a wider characteristic of society and is normally measured on a higher geopolitical level – initially as a system characteristic of countries or states (Kawachi & Berkman 2000, Sampson 2003). In contrast to the social capital concept, only the quality and quantity of social relations are emphasized and not the resources which these give rise to (Stafford et al. 2003). The working hypothesis of Wilkinson, who introduced the concept, stems from his observation that material inequalities in a society are both divisive and socially destructive or corrosive. In contrast to social capital theoreticians, he claims that the concept cannot yet be defined because it presupposes that we already have considerable knowledge of the phenomenon. Accordingly, for Wilkinson social capital is an empirical question – he suggests that material inequalities (with marked hierarchy, social polarization, stratification) are destructive for solidarity between people in society and he attempts to provide evidence of this (Wilkinson & Pickett 2006). It is apparent that Wilkinson's social cohesion concept is influenced by Putnam, and in recent publications he also fairly consistently uses social capital to describe social structures which he previously referred to as social cohesion (Wilkinson 2005). However, there are also some distinct differences, especially concerning how the phenomenon develops and breaks down. We will return to this in the section which examines the relations between social capital and health.

The concept '*collective efficacy*' is even more broadly defined than social cohesion. In addition to social cohesion, elements such as social control and the ability to mobilize action for a common purpose are stressed (Sampson et al. 1997). Collective efficacy is, to a greater extent than social cohesion, often understood as more locally founded social systems and has been operationalized at neighbourhood level (meso-level) to explain, for example, geographical differences in criminality. The concept is rarely used within public health.

3. The geography of social capital

Social capital is not only an economic, sociological and political concept, but can also be understood as a geographical concept – as a characteristic of *places*, *regions* or *countries*. If social capital occurs as a result of interaction between actors it can be expected that the quality of these relations will influence and be affected by the contexts in which individuals live. Putnam stresses that participation in voluntary organizations is an important prerequisite in this respect – through participation the basis for the development of trust and social capital is formed. At the same time, we know that such participation varies according to age, class, ethnicity, and gender. It can therefore be expected that, as a minimum, social capital varies in geographic space simply because different areas can have different compositions of individuals (i.e. there are *compositional* reasons for geographical variations). However, it has been shown that even after accounting for the composition of people, geographical variations in social capital is still to be found (Subramanian et al. 2003). This is in line with the proposition that social capital is a contextual phenomenon – and consequently a feature embedded in systems (a system characteristic).

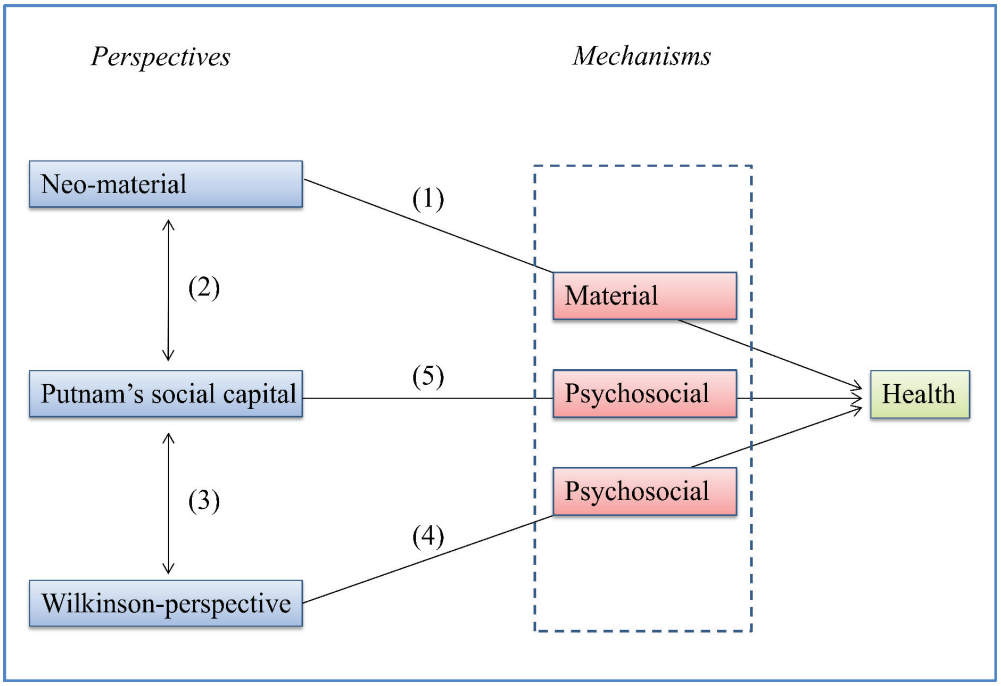
It follows that an unresolved question is what constitutes a relevant geographic scale or level in the measurement of social capital (assuming that it has a foundation in geographical space). To date, research has pointed to a very pragmatic approach to this problem. There is little doubt that accessibility to existing data has been decisive for what geographical levels such research has been carried out on, not leading theoretical considerations. The geographical levels which social capital has been applied to have varied considerably, and it is variously described as a system feature of countries, states, and down to local society (community). We will not give an exhaustive explanation of the concepts 'place', 'space', and 'region' here, but instead highlight the need for a greater acknowledgement of the fact that places are dynamic and more than just a static frame for social interaction. Social activities mean that places are continually changing – places and individuals influence one another through social practice and social capital can be understood as a part of such social practice (Mohan & Mohan 2002). The relationship between an individual and their place of residence is complex and the usual distinction of composition–context is to a large extent an artificial one (Macintyre et al. 2002), it is after all the relations between individuals which form the basis for the quality found at system level.

4. The relation between social capital and health

It is primarily Putnam's understanding of social capital which has attracted attention in health inequality research, and in the following account we will refer to this as Putnam's social capital perspective. In contrast, Wilkinson's hypothesis on income inequalities and social cohesion will be referred to as the Wilkinson perspective. In addition, we will also add a third perspective, one which deals with political economy and which is usually referred to as the neo-material perspective. Those who argue in favour of the neo-material perspective support

Wilkinson's hypothesis that income inequality is an important population health determinant but are very reluctant in terms of the connection with social cohesion (Lynch et al. 2001). Instead, supporters claim that countries with large differences in income systematically underinvest in public infrastructure and health services – this perspective thus represents a reorientation back to the importance of the welfare state for public health. A simple modified model based on Putnam (2004) can serve to illustrate the three main perspectives of the corresponding mechanisms (Figure 1).

Figure 1 Model of perspectives and mechanisms in the health inequality debate



Arrow (1) refers to the direct effect that welfare state schemes can have on health. There is little doubt of the importance of this effect, rather the debate largely concerns the degree of importance relative to other factors. Arrow (2) points to the reciprocal relationship between state and civil society. Arrow (3) relates to Wilkinson's hypothesis that inequalities in income are decisive for social capital in a society. The arrow shows that the relationship can also function in the opposite direction. For example, Putnam claims that it is equally likely that the opposite is possible – that an egalitarian distribution of income may be a consequence of social capital and cohesion (solidarity). Arrow (4) represents the direct effect of social inequalities in a society, including relative poverty. It is especially on this point that the conflict between Wilkinson and advocates of the neo-material perspective rests – namely, the extent to which the mechanisms are psychosocial or (neo)material. Arrow (5) points to the direct effect of social capital on health. The relationships represented by arrows 3, 4 and 5 are especially described and discussed in the section on the mechanisms operating between social capital and health.

Mechanisms between social capital and health

There are extensive epidemiological studies of mechanisms and relations on how social relations at individual level influence health (Berkman & Glass 2000). Stress-related reactions may be counteracted through the social support which is found in good social relations. At the same time, the absence of social relations can clearly constitute a risk factor. Such mechanisms are in many ways intuitively comprehensible, especially in relation to mental health. Yet, how can social phenomena, understood as a contextual phenomenon at the level of local society (meso-level), influence biological processes – how do they get 'under the skin' and ultimately become manifested as 'hard' health outcomes such as mortality? According to Kawachi and Berkman, there are at least three reasonable explanations for this (Kawachi & Berkman 2000). One is linked to health-related behaviour in local society, the second points to local society's ability to maintain 'access to services and other goods', and the third explanation implies psychosocial processes. We will examine these explanations more closely in the following.

According to Kawachi and Berkman social capital can affect *health behaviour* through two mechanisms. The first relates to how health information spreads faster and also increases the likelihood of citizens developing a set of norms for health promotion. Second, and related to the first mechanism, social capital can function as a control mechanism for health promoting behaviour. The latter mechanism is supported by findings in criminology where it is speculated that communities with a lot of social capital (here named 'collective efficacy') will prevent violations of prevailing norms of behaviour (Sampson et al. 1997).

The second mechanism concerns the local society's ability to (continue to) maintain public services and prevent the loss of existing provisions. It is well documented, for example from the UK, that there are systematic geographical variations in local societies' access to welfare services (Macintyre et al. 1993). Given such inequalities it is entirely understandable that societies which are able to form a common front for their local communities will have an advantage. In the long run, this may also contribute to demographic stability which subsequently makes it possible for social relations to have time to develop.

The third mechanism, which is controversial, postulates that psychosocial processes have a direct effect on health. This mechanism refers to the way that local societies with a lot of social capital will be able to provide their citizens with various forms of social support, self-respect, and encourage a greater degree of reciprocity between them. At neighbourhood level, empirical support for this mechanism is especially provided by studies which have reported that socially isolated individuals in societies with a lot of social capital have better health than corresponding individuals in societies with little social capital (Karachi & Berkman 2000). With regard to health, it is primarily the work of Wilkinson which has actualized the mechanism: he goes quite far in asserting (albeit with some reservations) that psychosocial factors are in fact clearly more important than material conditions in modern Western societies. His starting point is that the health of individuals is affected partly by social and economic circumstances and partly through their subjective understanding of their own

position relative to the social hierarchy in which they live. Based on this self-understanding, individuals will experience differing degrees of chronic stress and this is the key to understanding the direct biological effects on somatic health. It is continual exposure to chronic stress that makes individuals vulnerable to a number of diseases of the cardiovascular and immune systems (Marmot 2004). In addition, it is claimed that it is easier to adopt unhealthy practices if one has relatively low status in the social hierarchy (Wilkinson 1996). Thus, in addition to the directly postulated effects, lifestyle and health behaviour as a mechanism have an indirect effect.

However, the claim that psychosocial mechanisms are the most important factor behind the observed variations in health is perhaps more difficult to accept. Nevertheless, as Elstad have noted, psychosocial explanations certainly enriches our understanding of social causes in relation to health and health inequalities (Elstad 1998).

5. Empirical studies of social capital and health in Scandinavian countries

In addition to Wilkinson's work, a large number of earlier studies dating from around the mid-1990s which examined the relation between social capital and health were carried out by researchers at the Harvard School of Public Health in Boston, USA. These researchers used American data and operationalized social capital as a contextual characteristic of the states in the USA. Strong links were found between the level of trust and mortality in the American states (Kawachi et al. 1997). However, the studies suffered from several methodological weaknesses which rendered them vulnerable to criticism. Later research from this research group nevertheless showed that the links were (are) still valid, even when the initial methodological criticisms were taken into account (Subramanian et al. 2002; Subramanian & Kawachi 2006).

To date there has been a rapid increase in the number of studies examining the links between social capital and health. Several systematic literature reviews have been carried out on somatic health (Kim et al. 2008), mental health (Almedom 2005; De Silva et al. 2005; Whitley & McKenzie 2005) and health behaviour (Lindström 2008).

A number of studies have been conducted in a Scandinavian context and these are briefly summarized below. This overview is limited to peer-reviewed work which operationalizes social capital as a contextual area characteristic, where the geographical scale (or level) is lower than that of nation, and where adequate methods have been used, primarily multilevel analyses. It should also be emphasized that where we limit ourselves to studies which have measured social capital at meso-level, this is solely because it is this particular research area that we are most familiar with, and in which we hold a good overview.

Table 2 Associations between contextual social capital and health and health behaviour (Scandinavian studies).

Study (reference)	Design/ Analysis	Contextual Social Capital	Health and Health Behaviour Outcome	Result (fixed effects)
Lindström 2003 (Lindström et al. 2003)	Cross-sectional study in Malmø in 1994 3861 individuals (20–80 years) Neighbourhoods (N = 74)	Proportion out-migrants from neighbourhoods	Physical activity	No association
Martikainen 2003 (Martikainen et al. 2003)	Longitudinal study in Helsinki 1990–1995 251,509 men (25+ years) Residential areas (N = 55)	Index comprising: proportion that co-habited with partner, voted in 1988 election and out-migration	Cause specific mortality	Low social capital associated with mortality due to accidents, violence, alcohol, and respiratory disease
Blomgren 2004 (Blomgren et al. 2004)	Longitudinal study in Finland 1990–1996 1,100,000 men (25–64 years) Regions (N = 84)	Index of family patterns and voting in 1988 and 1992	Alcohol-related death	Low social capital associated with increased alcohol-related death
Johnell 2004 (Johnell et al. 2004)	Cross-sectional study in Malmø in 1991–1996 15,456 women (45–73 years) Neighbourhoods (N = 95)	Proportion with low participation in social activities	Use of medication for anxiety	Low social capital associated with increased use of medication for anxiety
Islam 2006 (Islam et al. 2006)	Longitudinal study in Sweden 24,419 individuals Local authority districts (N = 275)	Voting controlled for socio-demographic reasons	Health-related life quality	Low social capital associated with reduced health-related life quality
Johnell 2006 (Johnell et al. 2006a)	Cross-sectional study in Sweden in 2000 20,362 individuals (18–79 years) Local authority districts (N = 78)	Proportion with low participation in social activities	Ordinary prescriptions not followed-up	No association

Table 2 (continued)

Study (reference)	Design/ Analysis	Contextual Social Capital	Health and Health Behaviour Outcome	Result (fixed effects)
Johnell 2006 (Johnell et al. 2006b)	Cross-sectional study in Malmø in 1991–1996 12,156 women (45–73 years) Neighbourhoods (N=95)	Aggregated data on participation in social activities	Disability benefits	Low social capital associated with increased receipt of disability benefits
Lindström 2006 (Lindström et al. 2006)	Cross-sectional study in Skåne in 2000 13715 individuals (18–80 years) Neighbourhoods (N = 33)	Aggregated data on participation in social activities	Lack of access to permanent GP	No association
Sundquist 2006 (Sundquist et al. 2006)	Longitudinal study in Sweden 1998–2000 2,800,000 individuals (45–74 years) Neighbourhoods (N=9967)	Proportion that voted in local election in 1998	Coronary heart disease	Low social capital associated with increased risk of coronary heart disease
Sundquist 2006 (Sundquist & Yang 2006)	Cross-sectional study in Sweden in 2000–2002 11,175 individuals (25–64 years) Residential areas (N = 740)	Proportion that voted in national election in 2000	Self-rated health	Low social capital associated with increased risk of self-rated health being low
Lofors 2007 (Lofors & Sundquist 2007)	Longitudinal study in Sweden 1997–2000 4,500,000 individuals (25–64 years) Residential areas (N=729)	Representative of voting in local election in 1998	Admission for psychosis or depression	Low social capital associated with increased risk of psychosis but not of depression
Sund 2007 (Sund et al. 2007)	Cross-sectional study in Nord-Trøndelag 1995–1997 42,571 individuals (30–67 years) Electoral wards (N = 155)	Aggregated data on trust and participation in organizations	Self-rated health and depression	Level of trust associated with both outcomes No association with participation in organizations

Table 2 (continued)

Study (reference)	Design/ Analysis	Contextual Social Capital	Health and Health Behaviour Outcome	Result (fixed effects)
van der Wel 2007 (van der Wel 2007)	Cross-sectional study in Oslo in 2000 11,807 individuals Boroughs/districts (N = 25)	Aggregated data on trust and participation in voluntary organizations	Self-rated health	No association
Chaix 2008 (Chaix et al. 2008)	Longitudinal study in Skåne in 2000–2004 7791 individuals (45+ years) Neighbourhoods (N = 1533)	Aggregated data on social cohesion	Death caused by heart disease	Low social capital associated with death caused by heart disease
Engström 2008 (Engström et al. 2008)	Cross-sectional study of Stockholm in 2002 31,182 individuals (18–84 years) Residential areas (N = 92)	Aggregated data on social trust, political trust, participation in organisations, and elections	Self-rated health	No association

We have included a total of 15 studies carried out in the Scandinavian countries, of which 11 come from Sweden, 2 from Finland, and 2 from Norway (Table 2). Six of the studies had a longitudinal design while the remainder were cross-sectional studies. Ten of the studies report on associations between contextual social capital and measures of health or health behaviour in the expected direction. Hence, these studies find that social capital as a contextual feature and operationalized for geographic units, have an effect (cross-level) on the health or health behaviour of individuals. These effects are relatively moderate, and the studies which calculated the amount of variance that can be attributed to place of residence (by reporting the Intraclass Correlation Coefficient, ICC) find that individual circumstances are clearly the dominant factor.

It is also worth noting that some of the studies have examined interactions between contextual social capital and socioeconomic status without finding any significant associations. The exception is the study conducted by van der Wel (2007) from Norway, which showed an interesting interplay between contextual social capital and the income levels of individuals. For high income individuals, the social capital of the place of residence was unrelated to how they rated their health, while the health of those with low incomes improved by higher levels of social capital.

However, the studies vary quite widely in several respects. For instance, they differ in the way social capital is operationalized: some have used indicators on just one aspect of social capital (structural social capital) while others have attempted to operationalize both aspects (i.e. structural and cognitive social capital). Almost all of the studies have used indicators of social capital based on individual responses which were later aggregated, yet this approach is generally not considered to be ideal. Only two studies operationalized social capital after adjusting for underlying sociodemographic composition (Islam et al. 2006; Chaix et al. 2008). Further, we find there is large variation in the size of the geographical units (geographical levels) used in the studies: some have focussed on neighbourhoods while others have looked at large functional regions. A side-issue worth mentioning is that studies have been undertaken which have examined non-geographical contextual forms of social capital in relation to health, including two Finnish studies which report on associations between social capital at the workplace and self-rated health (Oksanen et al. 2008) and depression (Kouvonen et al. 2008).

Another essential aspect is the way in which confounding factors are adjusted for, at both individual level and ecological level. In this respect, there are large variations between the studies for several reasons, and these may account for the differences between those which find associations and those which do not. Studies of place effects introduce a number of new problematic factors in addition to those which ‘classical’ individual-level studies contain (Blakely & Woodward 2000). Migration is especially problematic for various reasons, one of them being that there is a risk of misclassifying the social capital ‘exposure’ for large proportions of the sample.

The Scandinavian countries are often portrayed as societies where the effect of contextual social capital on health (measured at meso-level) is modest or weak (Kim et al. 2008).

Further, we find that the relative importance of place of residence for the health of individuals is relatively minor in the Nordic countries compared to, for example, the USA – place of residence thus seem to be of greater importance within countries with large social inequalities and pronounced segregation. These discrepancies are often explained by the structure of the Scandinavian welfare model, which with its welfare schemes, social security system, and provisions relating to employment help to prevent social inequalities from becoming too great (Kim et al. 2008). It is interesting that the Scandinavian welfare model is put forward as an explanation for why social capital has less importance for health in the Scandinavian countries. It is particularly interesting in that it is advanced by those who are perhaps the leading supporters of the salutary effect of social capital, because it is precisely the same argument that the neo-materialists use as an argument *against* the relevance of social capital for health. We will not go into this interesting discussion more closely here, but will limit our attention by highlighting that in this regard, studies from the USA have very limited value in terms of transferability to a Scandinavian context.

6. Investing in social capital, a possible approach in public health work?

Norway is possibly among the countries in the world with the highest levels of social capital, where participation in voluntary organizations is greatly supported (Sivesind 2007), and its citizens show a great degree of trust in their fellow human beings (Ringdal 2004). While Norway evidently has a lot of social capital, does it necessarily follow that it is a nation of 'social capitalists'? This may be the general case, but in the same way as averages have concealed inequalities within public health, variations in social capital may be concealed in a similar way. On various questions relating to social capital, previous studies do indeed find large differences between groups of individuals (Rogstad 2007; van der Wel et al. 2007) and consequently we might also expect to find differences between geographical areas.

There is a high level of social capital, but it is unevenly distributed. Findings from Norway indicate that participation in voluntary organizations will soon become a feature of the middle classes (Wollebæk 2000). If trends in social capital over time show that it is about to become an elite phenomenon (cf. Bourdieu's capital forms and 'positions in social fields') we can no longer refer to social capital as a resource; on the contrary, it will be an exclusion mechanism (Wollebæk & Selle 2005). Is it the case that the bonding social capital within socially homogenous groups increases, while at the same time the (presumed more important) bridging social capital diminishes? These are crucial questions to shed light on in the years to come. With regard to health, it is relevant because there is speculation that bonding social capital can in fact have negative effects on the health of individuals (Ziersch & Baum 2004), while bridging social capital is assumed to be health promoting for individuals and society.

Thus, it cannot be denied that social capital can have negative effects. First, it is disputed that societies with a lot of social capital have better health status (Kaplan & Lynch 1997). Second, several of the indicators which have been used to describe social capital may reflect a high degree of social control – yet it is impossible to understand obligatory participation and rigid norms for behaviour as particularly health promoting. Another aspect that was touched upon earlier concerns bonding social capital. It is clear that close bonds within a group will not necessarily benefit those who fall outside the group, and in this respect social capital can also have an excluding effect. Further, it may also be the case that individuals within certain close groups can be subject to strong social control, which in extreme cases may develop in the form of violent acts and psychological terror, which clearly may constitute risk factors for disease.

Social capital is a relatively young concept. There are dissenting views on the definition, operationalization, measurement, analytical value, and its potential in policy making. In common with all new concepts it will take time to develop a more precise concept apparatus and the social capital concept is in a phase where much can appear complex and difficult to grasp. However, this is something which the social capital concept has in common with the early history of other concepts such as class, ethnicity, and gender – the social sciences are often concerned with phenomena which are difficult to observe. However, an apparently big paradox is why almost all health inequalities researchers have adopted Putnam's version of the social capital concept. One would assume that Bourdieu's work, which was fundamentally concerned with systematic social inequality and patterns of stratification, would have more to offer this field of research.

With certain reservations, we are positive towards the concept's importance in relation to public health. Where we have some reservations, they are linked to several factors, especially concerning what perspective is adopted concerning how social capital develops, and also the sources of social capital. As mentioned, Putnam sees social capital as a result of processes of socialization which occurs in voluntary organizations, consequently he has a bottom-up perspective. Social capital in *civil society* thus becomes a premise for a vigorous democracy.

On the other hand, from an *institutional perspective* one can equally argue the opposite case, i.e. that a viable democracy is a condition for social capital to occur and develop. Understood in this way, the focus shifts from the break-up of norms at the individual level and the concept is placed within a political context. Such a perspective is supported by macro-level studies which have found that it is countries with comprehensive welfare schemes which have the highest levels of social capital, in contrast to societies based on means-tested welfare programmes (Rothstein & Stolle 2003; Kumlin & Rothstein 2005). This is in good agreement with Wilkinson's hypotheses and, we would add, it addresses much of the criticism against the social capital concept in relation to health. In this critique it is claimed that recent interest in social capital represents a depoliticization of social inequalities in health and that investing in social capital becomes an exercise in social engineering that does not necessitate any redistribution of economic resources (Navarro 2002).

The civil society perspective and the institutional perspective are very different and consequently, it is not surprising that the social capital concept has been embraced along the entire political spectrum. There is agreement on the importance of trusting relations in the wider as well as in the local society, although the policy implications to nurture such relations will differ with respect to the perspective adapted. As such, the concept is by no means value-neutral. Our own understanding of the social capital concept is that we regard it as a consequence of public policy in the same way as social inequalities are. As such, a more equitable distribution of welfare and resources would be an investment in social capital – and in health.

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Paper 2

The influence of social capital on self rated health and depression – The Nord-Trøndelag health study (HUNT)

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Abstract

Introduction: The article examines the relationship between neighbourhood social capital and two health outcomes: self-rated health and depression. A total of 42,571 individuals aged 30–67 years participated in a cross-sectional total population health study in Nord-Trøndelag in 1995–1997 (HUNT II) and were investigated using multilevel modelling. The first aim was to investigate potential area effects after accounting for the characteristics of individuals in the neighbourhoods (N = 155). The second aim was to explore the relationships between contextual social capital (the level of trust at the neighbourhood level and the level of local organizational activity), and the two health measures.

Results: Models with stepwise inclusion of individual level factors attenuated the ward level variance for both self-rated health (PCV: 41%) and depression (PCV: 43%). The inclusion of the two contextual social capital items attenuated the ward level variance for both self-rated health and depression to varying degrees. At the individual level, contextual social capital was associated with both self-rated health and depression. Individuals living in wards with a low level of trust experienced an increased risk of 1.36 (CI: 1.13-1.63) for poor self-rated health compared to individuals in wards with a high level of trust. For depression, this effect was even stronger (OR: 1.52, CI: 1.23-1.87). The associations with the level of organizational activity were inconsistent and weaker for both health outcomes.

Conclusion: Geographical variations in self-rated health and depression are largely due to the socio-economic characteristics of individuals. Nevertheless, contextual social capital, expressed as the level of trust, was found to be associated with depression and self-rated health at individual level.

Introduction

The proposition that 'where you live' matters for health and longevity has been advanced by epidemiologists, demographers and geographers (1). Geographical variations in health were initially attributed to differences in the socio-demographic characteristics of individuals. However, features of the areas themselves can also contribute, independently of the individuals involved, and these are referred to as contextual explanations or contextual effects (2, 3). Also, observations of social class gradients in health and mortality in Western societies have received increasing attention. Regardless of type of indicator used to operationalize socio-economic position (SEP), the inverse and graded association between SEP and health have been found in virtually all populations (4). Both in the area-effects debate and the related health-inequality debate, the concept of social capital has re-emerged as an essential concept. It is typically regarded as a part of societal structure and a key determinant of the health of populations (5). In an attempt to combine both spatial and social elements, social capital might be the key concept to explain both geographical inequalities and health inequalities between social positions in society (6).

The main aims of this article are twofold. The first aim is to investigate whether geographical differences in health are a compositional artefact or due to contextual influences. Are geographical differences merely spatial manifestations of social inequalities in health? The second aim is to test the social capital and health relationship with two different health measures at a very low geographical level. The ward level used in these analyses is possibly in good correspondence with an individual's perception of what constitutes their neighbourhood. The underlying idea for this approach is that place makes a separate and distinct contribution to both spatial and social health inequalities.

Social capital, health and area effects

In recent years, the notion that some disease determinants cannot be conceptualized at the individual level has received attention (7–9). The idea that group level factors are important to the health of individuals is not new. In his seminal paper 'Sick individuals and sick populations', Rose (10) discusses the importance of detecting group level factors (or population-level factors as he terms them). By focusing on what distinguishes sick from healthy individuals *within* a population, studies may miss important health determinants, but by comparing different populations these factors may be detected. Hence Rose suggested that the causes of disease in individuals may be distinguished from the causes of differences in disease rates between populations (groups), and of great importance for disease prevention and particularly health inequalities (11). As Schwartz and Carpenter explained: 'When the causes of the rate differences between populations and the causes of interindividual variation within a population are different and the question is about rate differences, this discrepancy

results in what has been called a type III error – providing a right answer for the wrong question' (12, p. 1175).

The resurgence of interest in the social determinants of health has contributed to highlighting the importance of group level factors, with Wilkinson as an exponent in revitalizing this perspective (13–15). Summarizing some complex debates, he has argued that beyond a certain level of development, the most egalitarian societies have the best health standing. Wilkinson contends that egalitarian societies possess a higher level of social capital – simply stated, they are more cohesive. The social capital concept differs among theorists. Broadly speaking, it relates to social interaction and network membership, and their consequences (16). Wilkinson further argues that living in an equal society reduces the level of psychosocial stress resulting from pronounced social hierarchies. Partly as a result of Wilkinson's work, a separate body of research has emerged which is less focused on income inequalities and more oriented towards social capital, the 'stand-alone' social-capital-and-health hypothesis.

Social capital is a multidimensional concept, and following Putnam's definition it consists of 'features of interpersonal trust, norms of reciprocity, and social engagement that foster community and social participation' (17). The concept is usually further refined into structural (e.g. participation) and cognitive (e.g. trust) components, i.e. different *forms* of social capital. These can be described as 'what individuals do' as opposed to 'what individuals feel' (18). The beneficial effects to individuals include the provision of affective support and a source of self-esteem and mutual respect. Further, social capital can facilitate access to services and amenities, promote healthy behaviour, exhibit social control over deviant health-related behaviour, and ease the transmission of health information (19).

According to Wilkinson's view on social capital as an outcome of hierarchical societies, there are also direct physiological effects (embodiment) on individuals' psychoneuroendocrine systems through stress, physical hardship and emotional difficulties (13). This has led Wilkinson and others to suggest that psychosocial mechanisms are the key element (20, 21), and one type of critique of proposed mechanisms has been expressed from the 'neo-materialists' (22, 23). They claim the social capital (and psychosocial) literature obscures underlying political, administrative and economic determinants. The critique is directed against Wilkinson's income-inequality-and-social-capital hypothesis as well as the stand-alone social capital literature. However, the criticism is much sharper towards the latter because it allegedly omits structural economic inequality and political conflict (24). Nevertheless, as exponents for both sides in this vigorous debate acknowledge, separating the material from the psychosocial is not sustainable (24, 25). Both sides in the debate are rooted in the common perception that inequality is divisive and socially corrosive. It should also be noted that there are differences between Putnam's and Wilkinson's notion of social capital, especially regarding what kind of processes influence social capital formation and decline.

Studies of the importance of the social environment have shown that living in an area with a high level of social capital or related concepts is associated with a number of social phenomena: the quality of democracy (26), voting patterns (27), well-being (28), and crime

(29, 30). Health associations have been demonstrated with regard to mortality (31, 32), coronary heart disease (33), obesity and physical inactivity (34), and a number of other health outcomes. Of particular relevance for the current study are two reviews of the social capital and mental health relationship (35, 36). While Whitley and McKenzie conclude on inadequate evidence supporting social capital's influence on individuals' mental health, Almedom finds that social capital is salutary for adults' mental health. In relation to self-rated health studies are both supporting (37–39) and dismissing an association (40, 41). The variety of definitions, conceptualizations, operationalizations, and health outcome under study can possibly explain this discrepancy. Regarding the latter, there are a number of justifications for the current study's choice of health outcomes.

Depression is a highly prevalent disorder in the general population, and constitutes a major public health problem (42). The economic burden for society at large imposed by mental health problems is huge, both in terms of disability expenditure and loss of productivity. Approximately one-third of disability pensions awarded in Norway are based on mental health problems and are increasing both in absolute and relative terms (43). Depression has been found to be a robust predictor for disability pension awards (44).

Self-rated health is one of the most common health measures in health research. It is considered a valid and reliable indicator and a very cost-effective means of individual health assessment that provides valuable additional information to other health measures (45). An individual's assessment of their own health thus represents a summary statement of perceived aspects of subjective as well as objective health. Self ratings of health have also proved to be an important predictor for survival (46) and use of health care (47). A study utilizing the same data material as the current study demonstrated the predictive power of self ratings for subsequent mortality (48).

Methods

Data material

In the Norwegian county of Nord-Trøndelag, two large-scale, total population (20+ years of age) health surveys have been conducted (HUNT). The present study analyses participants from the second survey (HUNT II) and includes individuals of 30–67 years of age, comprising a total of 42,571 males and females. The individuals were nested within 155 wards.

Definitions: individual level

Outcomes:

Self-rated health was dichotomized into poor health (poor and fair) and good health (very good and good) based on the first questionnaire item: How is your present state of health?

Depression was derived from the Hospital Anxiety and Depression Scale (HADS). This is a self-report questionnaire consisting of seven four-point Likert-scaled items for depression. The HADS-instrument has shown good case-finding properties (49). A cut-off value of 8 on the depression subscale has been found to give an optimal balance between sensitivity and specificity and was therefore utilized in the present study.

Independent variables:

Age was classified into the four age categories: 30–39, 40–49, 50–59, and 60–67.

Sex: female and male.

Civil status was dichotomized as: (1) married (plus registered partner) versus (2) not married (plus widowed, divorced and separated).

Education comprised three groups: (1) Low: 7–9 years (no high school) and 10–11 years (some high school), (2) Medium: 12 years (completed high school), and (3) High: >12 years (College/University).

Employed was derived from the question: What kind of work situation do you have? This variable was dichotomized into (1) Employed (paid work and self-employed in business) and (2) Not employed (full-time housework, education/military service, unemployed/certified unfit for work, and retired/receiving social security). The respondents could fill in one or more responses to this question – hence all respondents who put a mark against paid work and/or self-employed in business were classified as employed.

Definitions: neighbourhood level

Social trust is based on the questionnaire statement: 'One cannot rely on each other here' (completely agree, partially agree, not sure, partially disagree, completely disagree). These were dichotomized into: (1) Trust (partially/completely disagree) and (2) No trust (completely/partially agree and not sure). The respondents were requested to relate their answer according to their neighbourhood/area of residence. The proportion reporting trust in each ward represents the level of neighbourhood trust. The wards were further classified into four categories based on the mean and +/− 1 SD. Wards with less than 30 individuals were excluded from the analysis.

Participation is derived from the question: 'How often do you take part in social activities such as, for example, sewing circle, athletic club, political association, religious or other associations?' The responses were dichotomized into: (1) Participation (1–2 times a month, about once a week, more than once a week) and (2) No participation (never/a few times a year). The proportion of individuals reporting participation in each ward represents participation in the respective neighbourhood/ward. The variable was classified similar to the social trust variable, into four categories (based on mean and +/− 1 SD). As with the trust variable, we excluded wards with less than 30 individuals from the analysis.

Statistics

Multilevel logistic regression models (two-level variance components models with random intercepts) with individuals (first level) nested within wards (second level) were fitted to the two outcomes in separate analyses. The baseline (null-model) contained no predictor variables. In the next model, individual level covariates were included, and in the last the two models, ecological social capital variables were fitted separately. The models were estimated using MLwiN software (Version 2.02 (50)), based on the penalized quasi-likelihood approximation (PQL) of a second-order Taylor linearization procedure. The binomial logit function was used in the models with the dichotomous outcome being set to record whether or not each respondent reported poor self-rated health or depression and an odds ratio of reporting these outcomes being estimated for each of the explanatory variables. The odds ratios hence constitute the models' *fixed effects*.

Random effects is reported as between ward variance (with standard errors). In addition, the Variance Partition Coefficient (VPC) is calculated. The VPC is the proportion of the total variance associated with the wards (second level). The VPC is calculated with the latent variable method (51) as:

$VPC = V_N / V_N + V_I$; $(V_I = \pi^2 / 3)$

V_N is the neighbourhood variance (second level variance) and V_I is the variance between individuals. The VPC ranges between 0 and 1 and a high value informs that areas are very important whilst a VPC of 0 suggests that the wards (area level) are similar to random samples taken from the county. Put simply, the VPC is a measure of the extent to which people within groups are more alike than across groups.

Ward differences in health may be attributable to both context (place) and characteristics of individuals. By adjusting for individual characteristics and comparing different models, the Proportional Change in Variance (PCV) quantifies how much of the initial area effect (second level variance) can be explained by the characteristics of individuals. The PCV is calculated for the different models as (52):

$PCV = ((V_0 - V_1)/V_0) \times 100$

The V_0 is the neighbourhood variance in the initial null model and V_1 is the neighbourhood variance in the consecutive models.

Table 1. Descriptives for individual (N=42571) and area level variables (N=155)

Individual level variables		Number	(%)
Age			
	30-39	11571	(27.2)
	40-49	13468	(31.6)
	50-59	10499	(24.7)
	60-67	7033	(16.5)
	Total	42571	(100)
Sex			
	Male	20323	(47.7)
	Female	22248	(52.3)
	Total	42571	(100)
Civil status			
	Married/reg.partner	30506	(71.1)
	Not married	12064	(28.3)
	Missing	*1*	*(0.0)*
	Total	42571	(100)
Education			
	Basic/secondary	29446	(69.2)
	Junior college	2990	(7.0)
	University	8963	(21.1)
	Missing	*1172*	*(2.8)*
	Total	42571	(100)
Employed			
	Yes	32791	(77.0)
	No	9140	(21.5)
	Missing	*640*	*(1.5)*
	Total	42571	(100)
Self rated healh			
	Good	31478	(73.9)
	Poor	10755	(25.3)
	Missing	*338*	*(0.8)*
	Total	42571	(100)
Depressive symptoms			
	No	35515	(83.4)
	Yes	4155	(9.8)
	Missing	*2901*	*(6.8)*
	Total	42571	(100)

Area level variables	Mean (SD)	Range
Population	275 (345)	31-2503
Level of trust	60 (8.9)	29.6-80.0
Level of org.act	58 (8.5)	34.7-77.4

Results

The descriptives for the individuals and the wards are summarized in Table 1. Missing values on the covariates were negligible, while on symptoms of depression the proportion missing was more pronounced (7%). The mean population in the wards was 275, (range 31–2503. The level of trust varied from *c.*30% to *c.*80% (mean 60), and the mean level of participation was 58% (range 35–77).

Bivariate associations between the health outcomes and the covariates are shown in Table 2. One-quarter (25.5%) reported poor self-rated health. On symptoms of depression, the prevalence was 10.5%. For the ecological variables the tendencies are in the expected direction with better health in the wards defined as having much social capital, and then gradually decreasing. The level of trust in the wards shows the expected associations between health outcomes, and for the level of participation the prevalences are as expected, yet somewhat more modest. Most noteworthy are the associations between labour market participation (employed or not employed) and the health outcomes. The age group included in the analysis are all in the working age group and although the non-employees are a rather heterogeneous group (as defined in this analysis), the associations seem quite strong.

Table 2. Bivariate associations between self rated poor health, depression and individual and area factors.

		Number (Valid %) Poor self rated health		Number (Valid %) Depressive symptoms	
Age					
	30-39	1622	(14.1)	773	(7.0)
	40-49	2794	(20.9)	1259	(9.9)
	50-59	3377	(32.5)	1262	(13)
	60-67	2962	(42.5)	861	(14.1)
	Total	10755	(25.5)	4155	(10.5)
Sex					
	Male	4687	(23.2)	2075	(10.9)
	Female	6068	(27.5)	2080	(10.1)
	Total	10755	(25.5)	4155	(10.5)
Civil status					
	Married	7688	(25.4)	2854	(10.0)
	Not married	3067	(25.6)	1301	(11.8)
	Total	10755	(25.5)	4155	(10.5)
Education					
	University	1346	(15.1)	587	(6.8)
	Junior college	524	(17.7)	206	(7.2)
	Basic/secondary	8411	(28.8)	3252	(11.8)
	Total	10281	(25.0)	4045	(10.4)
Employed					
	Yes	6074	(18.7)	2687	(8.6)
	No	4410	(48.7)	1380	(17.0)
	Total	10484	(25.2)	4067	(10.3)
Level of trust in wards (%)					
	High (>69.1)	702	(22.9)	264	(9.1)
	Med high (60.2-69)	6382	(24.1)	2423	(9.7)
	Med low (51.3-60.1)	3046	(28.3)	1206	(12.1)
	Low (<51.2)	625	(32.1)	262	(14.5)
	Total	10755	(25.5)	4155	(10.5)
Level of participation in wards (%)					
	High (>66.2)	741	(24.1)	273	(9.5)
	Med high (57.6-66.1)	4269	(23.6)	1740	(10.2)
	Med low (49.1-57.5)	4579	(26.5)	1696	(10.4)
	Low (<49)	1166	(30.4)	446	(12.6)
	Total	10755	(25.5)	4155	(10.5)

Tables 3 and 4 show the individual- and area characteristics' fixed and random effects for self-rated health and depression. The models were built sequentially, starting with a baseline model with no predictor variables to assess whether multilevel models were necessary. The random effects were significant for both self-rated health (Wald test $p < 0.05$) and depression (Wald test $p < 0.05$), suggesting that place of residence matters for these health outcomes.

Starting with self-rated poor health in Table 3, the amount of variance associated with area or place of residence (model 1) was approximately 2% (VPC = 1.9, Variance: 0.063 (SE: 0.011)). By adding individual level predictors, we were able to assess the relative importance of these factors and the level 2 variance gradually decreased with adjustment for sociodemographic characteristics. The relative importance regarding area effects of these individual level covariates can be monitored by the proportional change in level 2 variance (PCV). We noted that 41% of the level 2 variance is accounted for after adjusting for the characteristics of individuals.

For the social capital measures we observed that the level of trust in the wards is associated with poor self-rated health. Individuals living in wards more than one standard deviation below the average on the level of trust scale (i.e. low trust), have an increased risk of approximately 1.36 (95% CI: 1.13-1.63) compared to individuals in high trust wards. For the other two categories only the medium-low category (wards between the average and one standard deviation below the average) are significantly different from the reference category (OR: 1.24, CI: 1.08-1.44). The association with structural social capital is substantially weaker; only the category termed low organizational activity is significantly associated with poor self-rated health in model 4. We also note that their contributions in explaining level 2 variance are different.

For depression (Table 4) the level 2 variance in the empty model was 0.051 (SE: 0.012), giving a variance partition coefficient of 1.5. This variance gradually diminished in the consecutive models, and the proportional change in level 2 variance gradually increased while adjusting for the characteristics of individuals and features of the area. Adjustment for individual factors explained 42% of the level 2 variance, and with the inclusion of the trust variable in model 3, 71% of the level 2 variance was accounted for.

Table 3. Multilevel logistic regression of poor self rated health, men and women 30-67 years HUNT II. 42571 individuals nested within 155 wards. Odds ratios (95% CI)

	Model 1 Empty		Model 2 OR	(95 % CI)	Model 3 OR	(95 % CI)	Model 4 OR	(95 % CI)
Fixed effects								
Individual level								
Age 30-39			1	Ref	1	Ref	1	Ref
40-49			1.55	(1.43-1.67)	1.54	(1.43-1.67)	1.55	(1.43-1.67)
50-59			2.44	(2.24-2.65)	2.43	(2.24-2.64)	2.44	(2.24-2.64)
60-67			2.87	(2.51-3.20)	2.86	(2.57-3.20)	2.87	(2.73-3.01)
Sex								
Males			1	Ref	1	Ref	1	Ref
Females			1.13	(1.08-1.19)	1.13	(1.08-1.19)	1.13	(1.08-1.19)
Civil status								
Married			1	Ref	1	Ref	1	Ref
Not married			1.10	(1.04-1.16)	1.09	(1.04-1.16)	1.09	(1.04-1.16)
Education								
University			1	Ref	1	Ref	1	Ref
Junior College			1.26	(1.12-1.42)	1.26	(1.12-1.42)	1.26	(1.12-1.42)
Basic,- Second. School			1.56	(1.46-1.67)	1.55	(1.45-1.66)	1.56	(1.46-1.67)
Employed								
Yes			1	Ref	1	Ref	1	Ref
No			2.60	(2.27-2.97)	2.58	(2.26-2.96)	2.59	(2.26-2.97)
Interaction age x Employed								
Age 30-39 x Not employed			1	Ref	1	Ref	1	Ref
Age 40-49 x Not employed			1.74	(1.46-2.09)	1.75	(1.46-2.09)	1.74	(1.46-2.09)
Age 50-59 x Not employed			1.60	(1.35-1.90)	1.60	(1.35-1.90)	1.60	(1.35-1.90)
Age 60-67 x Not employed			0.88	(0.74-1.05)	0.88	(0.74-1.05)	0.88	(0.74-1.05)
Ward level								
Cognitive social capital								
High Trust					1	Ref		
Medium high					1.10	(0.96-1.26)		
Medium low					1.24	(1.08-1.44)		
Low Trust					1.36	(1.13-1.63)		
Structural social capital								
High Org activity							1	Ref
Medium high							0.99	(0.86-1.14)
Medium low							1.07	(0.92-1.23)
Low org activity							1.19	(1.00-1.42)
Random effects								
Level 2 variance (SE)	0.063	(0.011)	0.037	(0.008)	0.030	(0.007)	0.033	(0.007)
VPC	1.9		1.1		0.9		1.0	
PCV	REF		41 %		52 %		48 %	

VPC=%

Table 4. Multilevel logistic regression of depression, men and women 30-67 years HUNT II. 42571 individuals nested within 155 wards. Odds ratios (95% CI)

	Model 1 Empty		Model 2 OR	(95 % CI)	Model 3 OR	(95 % CI)	Model 4 OR	(95 % CI)
Fixed effects								
Individual level								
Age 30-39			1	Ref	1	Ref	1	Ref
40-49			1.45	(1.30-1.62)	1.45	(1.30-1.61)	1.45	(1.30-1.61)
50-59			1.86	(1.66-2.08)	1.85	(1.65-2.07)	1.86	(1.66-2.08)
60-67			1.94	(1.66-2.28)	1.93	(1.65-2.27)	1.94	(1.66-2.27)
Sex								
Males			1	Ref	1	Ref	1	Ref
Females			0.85	(0.79-0.91)	0.85	(0.79-0.91)	0.85	(0.79-0.91)
Civil status								
Married			1	Ref	1	Ref	1	Ref
Not married			1.29	(1.20-1.39)	1.29	(1.19-1.39)	1.29	(1.20-1.39)
Education								
University			1	Ref	1	Ref	1	Ref
Junior College			1.07	(0.91-1.27)	1.07	(0.90-1.27)	1.07	(0.91-1.27)
Basic,- Second. School			1.46	(1.33-1.61)	1.45	(1.32-1.60)	1.46	(1.32-1.60)
Employed								
Yes			1	Ref	1	Ref	1	Ref
No			1.99	(1.64-2.41)	1.96	(1.62-2.38)	1.98	(1.63-2.40)
Interaction age x Employed								
Age 30-39 x Not employed			1	Ref	1	Ref	1	Ref
Age 40-49 x Not employed			1.46	(1.14-1.86)	1.46	(1.14-1.86)	1.46	(1.14-1.86)
Age 50-59 x Not employed			1.05	(0.83-1.32)	1.05	(0.83-1.33)	1.05	(0.83-1.32)
Age 60-67 x Not employed			0.68	(0.53-0.87)	0.68	(0.53-0.88)	0.68	(0.53-0.87)
Ward level								
Cognitive social capital								
High Trust					1	Ref		
Medium high					1.10	(0.94-1.29)		
Medium low					1.31	(1.11-1.54)		
Low Trust					1.52	(1.23-1.87)		
Structural social capital								
High Org activity							1	Ref
Medium high							1.09	(0.92-1.30)
Medium low							1.13	(0.95-1.35)
Low org activity							1.28	(1.04-1.58)
Random effects								
Level 2 variance (SE)	0.051	0.012	0,029	0,009	0,015	0,007	0,028	0,009
VPC	1.5		0.9		0.5		0.8	
PCV	Ref		43 %		71 %		45 %	

VPC=%

The inclusion of the ecological trust variable shows a similar, yet stronger, association with depression than with self-rated health. For individuals living in low trust wards the relative risk of depression is approximately 50% higher compared to the reference category, and residing in what we term medium-low areas exerts a 30% increased risk of poor self-rated health. In model 4 the inclusion of the organizational activity variable demonstrates incoherent associations; only the category termed low organizational activity was associated with depression (OR: 1.28, CI: 1.04-1.58).

Discussion

Summarizing the findings along with possible limitations in this study, we found in accordance with our first research question, that geographical inequalities in health were reduced when we adjusted for the characteristics of individuals. More than 40% of the second level variation is explained by adjusting for the characteristics of the individuals for both health outcomes. Hence we can claim that geographical inequalities in health are partially spatial manifestations of social inequalities in health at the individual level. Nevertheless, some 60% remains unaccounted for in these analyses. With the inclusion of the ecological level of trust variable, a total of 52% for self-rated health and *c.*70% for depression were accounted for of the second level variance. The level of participation in organizational activity explained some of this variance for self-rated health, but was almost negligible in relation to depression.

Regarding our second research question, we found that the level of trust in an individual's neighbourhood was clearly associated with the operationalized health outcomes. Individuals living in wards possessing a low level of trust experienced a 50% increased risk of depression compared to individuals in high trust wards. For self-rated health, the social capital effect was slightly weaker, with a 36% risk increase. Besides explaining some of the second level variance, this ecological construct also had a direct cross-level effect on individuals' health. The other social capital measure, participation in organizational activities, was found to be weak and gave inconsistent results.

Our first research question concerns a rather classical issue, often termed the composition/context debate. The fruitfulness of this dichotomy is debatable, and as has been argued, 'the distinction between "composition" and "context" may be more apparent than real' (53, p. 125). The discussion concerns the extent to which area effects have an independent effect on the health of individuals. A common argument to set aside area-effect studies is that the models are mis-specified and the apparent area effect emerges due to individual level factors having been omitted in the analysis. The corresponding counter argument is that none of the individual factors or compositional characteristics in an area have emerged randomly. People move from one place to another for a variety of reasons, and they settle for an equal number of reasons (or limitations). Some of these individual characteristics might just as well be regarded as mediating factors rather than risk factors or confounders. As

some geographers have stated: ‘Places form people as much as places are formed from peoples’ (54, p. 8). This study included individual level attributes in accordance with previous studies of area effects.

The second research question, relating to the first, concerns social capital as an alleged area effect. This has importance for geographical health inequalities as well as bearings on individuals’ health. The relationship poses problems in terms of interpretation. Regarding causality, the direction of this association could, in principle, go either way due to the interrelatedness between the outcomes at the individual level and our social capital predictors at the ecological level. Low trust could be the outcome of, as well as a determinant for, individual health (55). This poses a limitation especially for the association with depression – the varying degrees of trust can be a reflection of different prevalence’s of depressed individuals in the wards. It is also questionable whether social capital is adequately operationalized and whether it is strictly spatially bounded.

Our findings contradict the conclusions from a review article on the social capital and health relationship in egalitarian societies (56): first, that social capital was less salient for individuals’ health in egalitarian societies, and second, studies supporting this relationship applied a high geographical level, often using US states. Our study used the lowest available geographical level to measure social capital. The rationale behind this was that we would get more homogenous units intended to measure bonding social capital, i.e. the type of social capital between individuals with similar socio-economic status.

The social capital effect on individuals’ health is a cross-level effect and refers to the main (or direct) effects of higher level units on outcomes at a lower level. This effect can, in principle, be confounded in the same way as individual level analyses can be confounded. We cannot rule out the possibility that other features of the neighbourhoods may be of importance, and as Diez Roux has suggested, we can make a conceptual distinction between two types of area level variables (57). On the one hand, we have the so-called ‘integral’ area features (no analogues at the individual level), such as the availability of health and social care, population density, certain types of regulations, etc. On the other hand, we have ‘derived’ features (summaries of individual properties), such as socio-economic compositional characteristics, in/out migration, proportion single-person households, age/sex composition, etc. Related to the latter, the aggregation of individual level variables to represent an over-individual phenomenon can cause ambiguity of what this variable is actually measuring. A particular strength of this study is that the respondents were asked to relate their answer about trust to their immediate neighbourhood. This was not the case with the participation in organized activities, and may in fact explain our findings for this particular indicator. Furthermore, we would also have preferred adjustments for individual income, which is not available. It is not unlikely that some of the area variability and social capital effects could have been influenced. Ideally, we would have preferred to follow recent recommendations of using multiple measures of socio-economic position.

The underlying theme in this study concerns socio-economic disparities in health that we observe at the individual level, or more correctly between aggregated positions in terms of

stratification in society. The relation between social capital in individuals' neighbourhoods and social health inequalities may at first glance seem vague. Two apparently different perspectives may illustrate how features of the residential area may influence social health inequalities. The first is termed the 'neo-material', whilst the second is often termed the psychosocial. Briefly, the psychosocial explanation emphasizes the deteriorating health effects of relative social disadvantages and the accompanying feelings. The neo-materialists claim that the structural and material causes of inequalities are the main explanatory factors and not just perceptions of inequality and relative disadvantage. Applied to our study, the social capital and health relationship is considered a psychosocial interpretation, and the socio-economic inequalities in health in the wider society are partly caused by differing levels of social capital. These differing levels of social capital are expressed in extended social distance, differing levels of cohesion and solidarity, and is more stressful. The protective value of social capital for health thus varies according to neighbourhood. The neo-material interpretation, less occupied with perceptions of disadvantages, argues that these alleged area effects of social capital emerge because of differing levels of social investments in the neighbourhoods (e.g. in schools, health care). Our response to this discussion is that the latter interpretation, although highly relevant in other societies, has less value in the Norwegian welfare state. Variations in communities' provision of welfare services according to need are limited, and accordingly, local spending on welfare institutions is higher in disadvantaged communities. This redistribution effect makes the social capital and psychosocial interpretation more likely. Studies performed in Scandinavian welfare states may add pertinent knowledge to the debate of the relative magnitude of psychosocial versus neo-material factors in explaining socio-economic disparities.

Similar to other studies applying a cross-sectional design, a cross-sectional multilevel study not only introduces the possibility of reverse causation, it also implies a zero lag-time between the ecological exposure (social capital) and individuals' health (58, 59). A zero lag-time is usually impossible, although impaired if the ecological exposure is stable over time. In order to reduce this misclassification bias this study applied health outcomes that to a larger extent respond to current exposures than mortality or diseases. It is simply more intuitive to expect an association with psychological and subjective health measures.

Selection bias is another unavoidable feature in population health surveys. Nevertheless, a non-responder study suggested no significant selection according to health or demography (60). The slightly lower participation in the youngest age groups does not affect this study as only adults (30+ years) are included. Another crucial bias in cross-sectional studies is the complexity of migration. The proposed area effect on individual health may also be misclassified. The effect of migration on geographical inequalities can, in principle, go either way, but in terms of area effects on individual health this constitutes a serious limitation and calls for longitudinal studies.

Conclusion and policy implications

The study shows that over-individual characteristics, the level of trust associated with social capital in neighbourhoods, have significant impacts on self-rated health and depression, independent of individual-level factors. The findings give conditional support to assumptions that local social capital and neighbourhood still act as an influential framework or arena of life and health issues for people. The effects or presence or absence of such forms of local social capital or 'ties' may seem 'weak' (61) and marginal at first glance. Each effect, considered isolated, seems almost negligible and apparently disappears when their 'genuine' health effects are scrutinized. However, the sum effects of the captured protective factors or harmful factors may function as a 'sprinkling system'.

The area effect linked to the local physical environment and their design and arrangements will stimulate and substantiate a well-functioning social life. Meeting places, access to low threshold exercise activities and green areas are just a few examples. They may facilitate social capital formations which enhance the local level of thriving and health. At the same time, neighbourhood inequalities in the socio-material structure may foster health inequalities in the population.

Mounting evidence that neighbourhood qualities and forms of social capital matter for the population's health has public health implications. To the point formulated, '[i]f social conditions rather than individual behaviour cause the problem, then society must share some of the responsibility for solving it' (62, p. 340). In an era of increasing individualism, collective health promoting actions initiated or supported by public or non-public efforts should be revisited.

Despite moderate support for the protective value of social capital for the health of individuals in this study, we do not recommend some kind of 'social engineering' to enhance social capital in society unless the more fundamental causes (63) have been addressed first. Nevertheless, if social capital is understood as a 'societal-wide capacity for inclusiveness, human rights, social justice, and full political and economic participation' then public health should invest in social capital (23, p. 408).

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Paper 3

Individual, family, and area predictors of BMI and BMI change in an adult Norwegian population: Findings from the HUNT study.

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Abstract

The global obesity epidemic is a major public health concern and there is strong evidence that the drivers are varied and operate via diverse pathways. Taking a systems approach allows the contextual influences operating upon the individual to be identified and quantified. We adopt such a perspective in this study, where longitudinal data from a cohort of 24,966 settled individuals participating in two major health surveys, the Nord-Trøndelag Health Study (HUNT 1 and 2) in the county of Nord-Trøndelag, Norway, were used to investigate associations between individual, family and area characteristics and two outcomes: body mass index (BMI) at follow-up and BMI change over an 11 year period. Linear multilevel models were fitted, with individuals nested in 17,500 families, 447 wards and 24 municipalities. A range of putative individual, family, and area predictors were tested. We found both outcomes were strongly associated with individual characteristics, with higher BMIs generally being amongst males, unmarried participants, non-smokers, those of lower education and those undertaking physically demanding work but participating in less physical activity outside work. The characteristics of those in the sample exhibiting higher BMI gain were rather similar except that women gained more and those with no employment income gained less. Contextual influences were also found to be important; although just 1 % of the unexplained variance was located on the neighbourhood and municipality levels respectively, and hence suggesting small environmental influences, between 10-13 % could be attributed to families, highlighting the importance of the familial contextual environment. Rather little is known about the manner by which family influences may operate on bodyweight hence further work is needed to understand likely mechanisms and guide future interventions.

Introduction

The increasing prevalence of obesity has been described as an epidemic process (James, 2008) and is now a major driver behind rises in the prevalence of certain chronic diseases and disabilities worldwide. Recent projections from the World Health Organisation (WHO) estimate that globally 1.6 billion adults were overweight and at least 400 million were obese in 2005, with these figures expected to grow to 2.3 billion and 700 million by 2015 (WHO, 2006). In Europe current trends are expected to give rise to 150 million obese adults by 2010 (Branca, Nikogosian, & Lobstein, 2007). The annual rate of increase in childhood obesity in this region is a particular cause for concern, with the current prevalence being over 10 times higher than in 1970 and with 15 million children expected to be obese by 2015 (Branca et al., 2007). Indeed, some have forecasted that youths of today may, on average, live less healthy and possibly even shorter lives than their parents (Olshansky, Passaro, Hershow, Layden, Carnes, Brody et al., 2005).

In a recent WHO publication, Branca et al. (2007, p.xiii) state that "obesity presents Europe with an unprecedented public health challenge that has been underestimated, poorly assessed and not fully accepted as a strategic governmental problem with substantial economic implications". The authors further note that the prevention of obesity requires innovative environmental approaches. The term "obesogenic environment" refers to the role environmental factors may play in determining food intake and physical activity, both important determinants of bodyweight (Jones, Bentham, Foster, Hillsdon, & Panter, 2007). Swinburn and colleagues have defined the concept as "the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations" (Swinburn, Egger, & Raza, 1999 p 564). These authors have further described the environment in terms of micro and macro components, where micro-environments are defined as settings that influence peoples' interactions (e.g. home, school, workplace, and neighbourhood) which are themselves influenced by macro-environments (e.g. the education and health system, government policy and society's attitudes and beliefs).

The concept of an obesogenic environment is grounded in a systems perspective where health related behaviour is contextualised in the environment within which it takes place. The advancement of the concept has been driven by the development of ecological models which suggest that weight related behaviours, such as food intake and physical activity, arise as the result of the combined action of psychosocial, demographic, as well as physical environmental processes (Diez Roux, 2007; Van Dyck, Deforche, Cardon, & De Bourdeaudhuij, 2009)

Numerous different environmental factors at various geographical scales have been put forward as potential determinants of overweight and obesity (Black & Macinko, 2008). There is some evidence of an effect of income inequality, with Pickett and colleagues (Pickett, Kelly, Brunner, Lobstein, & Wilkinson, 2005) finding a positive association between income inequality and rates of obesity in 21 developed countries, whilst Holtgrave & Crosby (2006) found higher levels of social capital in US states to be associated with a lower prevalence of obesity. At the neighbourhood level, many studies in the US (e.g. Diez-Roux, Link, &

Northridge, 2000; Janssen, Boyce, Simpson, & Pickett, 2006; Rundle, Field, Park, Freeman, Weiss, & Neckerman, 2008), UK (e.g. Moon, Quarendon, Barnard, Twigg, & Blyth, 2007) and in Canada (e.g. Ross, Tremblay, Khan, Crouse, Tremblay, & Berthelot, 2007) have reported that high material deprivation levels are associated with elevated adult obesity prevalence.

A large amount of research evidence on the role of the physical environment is available. A recent review of predominantly US studies found that the majority reported an association between some aspect of the neighbourhood built environment and obesity, with associations with features such as the walkability of neighbourhoods and the accessibility of greenspaces being found (Papas, Alberg, Ewing, Helzlsouer, Gary, & Klassen, 2007). In addition to objectively measured features, the findings of a recent meta-analysis support the view that perceptions of the neighbourhood environment, such as those regarding safety and the accessibility of destinations, are also important (Duncan, Spence, & Mummery, 2005).

A contextual unit rarely acknowledged in the obesity literature is the family or household. Yet there is compelling evidence in support of interventions at the parent or family level in paediatric obesity research (Zeller, Reiter-Purtill, Modi, Gutzwiller, Vannatta, & Davies, 2007). Parental obesity has a strong predictive power in the development of child and adolescent obesity, arguably with a genetic component, but there are also studies suggesting that there are indeed modifiable determinants operating at this contextual level (Krahnstoever Davison, Francis, & Birch, 2005). These could include the availability of foods, and the provision of familial social support for physical activity, and weight management practices.

There is strong evidence that the drivers of the epidemic are varied and operate via diverse pathways. Systems approaches (see Bailey, 1994) to exploring health behaviour causation can be useful in such situations, as they allow the outcomes of individual actions to be examined within the social and environmental contexts within which the individual operates. We adopt such a perspective in this study, where the aim is to contribute to an understanding of how environments may directly and indirectly affect behaviour and how such behaviour is ultimately expressed in terms of bodyweight. This is done firstly by quantifying variation in body mass index (BMI) and changes in BMI associated with individual, family, and area characteristics. Secondly we try to explain how the composition of individuals in families and areas may account for this variation. Finally we test how contextual features like family and area socioeconomic status, and area deprivation and social capital may explain variability in the outcomes not associated with characteristics of individuals. The research is longitudinal, utilising anthropometric height and weight measurements from two time points amongst a large and settled adult population from the county of Nord-Trøndelag in Norway.

Methods

Data sources

The Nord-Trøndelag Health Study (HUNT) is one of the world's largest population health surveys. The first wave (HUNT 1) was carried out in 1984–1986. All residents of Nord-Trøndelag County in Norway aged 20 or above were invited to participate in the study which included a physical examination and self-completed questionnaires. Questionnaire 1 was attached to the invitation letter, and 74,977 (88.1%) of the 85,100 eligible persons completed and returned it when they attended. Of these, 74,332 (99.1%) had reliable height and weight measurements recorded with light clothes and without shoes by specially trained nurses. During the clinical investigation, participants were given a second questionnaire to complete and return by mail. This collected information on socio-demographic and behavioural issues (education, alcohol intake, smoking, lifestyle issues, and functional impairment) and was returned by 53,016 (70.7%) of the study members. More detailed descriptions of the methods in HUNT 1 are provided elsewhere (Holmen, Midthjell, Bjartveit, Hjort, Lund-Larsen, Moum et al., 1990).

The second wave of HUNT (HUNT 2) was carried out in the same geographic region as HUNT 1 in the period 1995 to 1997 (Holmen, Midthjell, Krüger, Langhammer, Lingaas Holmen, Bratberg et al., 2003). Similar methods for measuring height and weight were used. A total of 47,048 (72%) of the participants in HUNT 2 had also participated in HUNT 1. Data linkage between HUNT 1 and HUNT 2 was undertaken via the national Norwegian 11 digit unique personal identification number. This number was also used to link every individual to National statistics' registries from which we derived information on place of residence (ward number), family identification number, education and income data. These registries do not however, contain information on the entire follow-up period, but only for the four years preceding the second wave of HUNT. The exception is education for which there is information dating back to 1970. All data linkages were undertaken by a third party and preserved participant anonymity. The Norwegian Data Inspectorate and the Regional Committee for Ethics in Medical Research approved the protocols for HUNT 1 and HUNT 2. The protocol for this study was approved by the Regional Committee for Ethics in Medical Research and the HUNT Publication Review Board approved the manuscript.

The present study, carried out in 2009, includes men and women 25-64 years old at baseline (HUNT 1) and who participated in HUNT 2 eleven years later. Of the 47,048 participants attending both waves 24,966 were eligible for inclusion here. After excluding 7,605 individuals who were outside the age-range 25-64 years and 853 (2%) with missing weight or height measurements from any of the two waves we were left with 38,590 individuals. From these we excluded 9,447 individuals who had missing values on any of the covariates from the baseline survey (24%). In addition we left out 4,152 individuals (11%) who had changed municipality of residence between the waves (or changed ward number or family number in the period 1992 to 1995) and 25 individuals (~ 0.1%) who had an error in municipality and ward coding. Norwegian municipalities are administrative units with an average population in

1998 of 10,155. Wards are a lower level statistical unit nested within the municipalities, with an average of 322 inhabitants in 1998. The analysed subsample of 24,966 individuals constitutes 65% of the original eligible cohort. The main reason for missing values on the covariates is because of nonresponse to the second questionnaire in the baseline survey (HUNT 1) which was to be completed and returned by mail after the clinical examination. The 24,966 individuals were located in 17,500 families (Mean=1.4), 447 wards (Mean=55.9) and 24 municipalities (Mean=1040.3) respectively. Of the 17 500 families just 0.2% were three-person, 42.2% were two-person and the rest were singleton clusters (57.6%) in our data, although other family members may have been present but did not participate in HUNT.

Outcome

We fitted two sets of models; one examining BMI at the end of the follow-up in 1995, and one BMI change in the period 1984 to 1995. We calculated BMI as the ratio between body weight in kilograms (kg) and body height in meters (m) squared (kg/m^2). BMI is accepted by the WHO as the appropriate measure of assessing under- and overweight (WHO 1995). BMI-change in the follow-up period was modelled using the methodology of Vickers & Altman, (2001) by fitting BMI in 1995 as the outcome and adjusting for baseline BMI by including it as an explanatory variable in the model. This is the equivalent to the analysis of a change score.

Individual level explanatory variables

All individual level explanatory variables are baseline characteristics collected in 1984-85 (HUNT 1). Age in years and age-squared were modelled as continuous variables with both centred on their mean. Sex was modelled as a dummy variable with males as the reference category. Marital status was classified into four groups: Married (reference category), not married, widow(er), and divorced/separated. Occupational class was based on self reported responses about occupation and was classified into non-manual (reference), manual and a no-employment-income group. Education was derived from national statistics registries' and was the highest level of education achieved up until the first wave in 1984-85 (HUNT 1). We dichotomized this variable into high (completed a college or university degree) and low (primary or high school) education, with the former as reference category.

Estimates of physical activity level during work, including domestic activity amongst homemakers, were made based on responses to the question "Is your work physically demanding to the extent that you feel physically tired or exhausted at the end of the work day?" We reclassified the responses into 'yes' ("almost always" and "quite often") or 'no' ("rarely" and "seldom, hardly ever") with the latter as the reference category. Physical activity in leisure time was based on the questionnaire item: "How frequently do you exercise (give an average)? By exercise we mean, for example, going for walks, skiing, swimming or training/sport". We dichotomized the five category response into 'inactive' (never, less than once a week, once a week) and 'active' (2-3 times a week, nearly every day) with the former as reference category. Smoking was coded as either non (reference) or current smoker.

Contextual explanatory variables

Family income was based on the aggregation of individual level data (net income) derived from Statistics Norway registries' for the two years (1993 and 94) prior to the second wave of HUNT. We calculated the average for these two years for every individual, aggregated these averages to the family level using the family identification number, and using the methodology of recent OECD publications on income inequality (OECD, 2009) we divided by the square root of the number of family members (also derived from Statistics Norway registries data) to account for scale advantages in larger families. We then calculated the natural logarithm (Ln) of this measure of "equivalent family income". Family income, an indicator of family socioeconomic status (SES), is hypothesised to relate to BMI by describing microenvironments that may be more or less conducive to physical activity and healthy eating via mechanisms of ability to pay, and also in terms of knowledge (Giskes, van Lenthe, Kamphuis, Huisman, Brug, & Mackenbach 2009; Burke, Beilin & Dunbar 2001).

Ward education level, the only available indicator of ward socioeconomic status (SES) was measured as the proportion of individuals aged between 16 and 66 with a high level of education (college or university degree) in the wards. These ecological data were derived from a secondary data source: "Censuses on constituency level" maintained by Norwegian Social Science Data Services (NSD, 2008) for the year 1990. The rationale for including this predictor is previous studies showing that neighbourhoods with a high socioeconomic status have lower levels of obesity. Access to resources conducive to physical activity and healthy eating are likely mechanisms. In addition, neighbourhood SES may act as a surrogate measure for safety, as well as the presence of chronic neighbourhood stressors, (Mujahid, Diez-Roux, Shen, Gowda, Sánchez, Shea et al., 2008).

The municipality deprivation score employed was a summary measure developed by Statistics Norway that characterised the 24 municipalities in Nord-Trøndelag County in terms of material deprivation, or "level of living" in a Norwegian context, for the year 1989 (Tønseth, 1999). The variable is a composite of the following indicators: male unemployment rate, low educational level, general practitioners per 1000 inhabitants, physiotherapists per 1000 inhabitants, average income per inhabitant, person-years in home nursing care per 10 000 inhabitants, net-migration, supplementary benefit recipients per 1000 inhabitants, prevalence of disability pension claimants, percentage child care coverage, after-school care facility provision, traffic accidents per 1000 inhabitants, male mortality 45-66 years, female mortality 45-66 years, and municipality gross income per inhabitant. The index ranges theoretically from 16 (high deprivation) to 60 (low deprivation). The indicator of population change, a longitudinal indicator for area deprivation, was similarly derived from Statistics Norway and measures percentage population change between 1984 and 1995. Previous studies have shown that place deprivation is associated with obesity, and a number of mechanisms and pathways may be operating. These include the lack of infrastructure for physical activity or healthy eating, but also the presence of health damaging factors such as outlets providing energy dense food (Macdonald, Cummins & Macintyre 2007). Social capital was measured in terms

of election participation and was the pooled participation rate for the local political elections for the years 1983, 1987 and 1991. Election participation has been used as a measure of social capital in a number of previous studies (e.g. Sundquist & Yang, 2007). Likely mechanisms by which social capital could operate include the diffusion of knowledge about healthy lifestyles, maintenance of healthy norms through informal social control, promotion of access to services and amenities, and also psychosocial processes which may, for example, act to support physical activity behaviours (Kawachi & Berkman 2000). All municipality level variables were gathered from the Norwegian Social Science Data Services who administer Statistics Norway's data for researchers.

Statistical Analyses

Multilevel linear models (Goldstein, 2003; Hox, 2002; Rabe-Hesketh & Skrondal, 2008; Snijders & Bosker, 1999) were estimated to distinguish the individual, family, ward, and municipality-level sources of variation in BMI and BMI change. The multilevel statistical modeling framework allows the simultaneous examination of the effects of group-level and individual level predictors and it accounts for the non-independence of observations (clustering) within higher level units. In addition, both inter-individual (difference between individuals) and intergroup variation (differences between higher level units) can be examined as well as the contributions of individual level and group-level variables to these variations.

In the context of the analysis presented here, the multilevel techniques allowed estimation of the overall relationships between BMI and BMI change and both individual and contextual correlates, along with the variation between the higher level units (family, ward, municipality) that could not be accounted for by these factors. The substantive and technical relevance of multilevel models in public health is well described (Blakely & Subramanian, 2006; Congdon, Shouls, & Curtis, 1997; Diez Roux, 1998, 2000, 2004a, b; Moon, Subramanian, Jones, Duncan, & Twigg, 2005; Subramanian, Jones, & Duncan, 2003).

Four-level models were estimated, with the continuous responses, of BMI and BMI change for individuals (level 1) nested within families (level 2), wards (level 3) and municipalities (level 4). The model parameters were estimated using the maximum likelihood-based Iterative Generalized Least Squares (IGLS) algorithm and the models were fitted using the MLwiN v.2.11 package (Rasbash, Charlton, Browne, Healy, & Cameron, 2009). A modeling strategy was developed whereby a null (empty) model depicting the variation in outcome between the four levels were initially fitted (Model 1). This was followed by the introduction of individual level demographic (e.g. age and sex) predictors (Model 2), then individual socioeconomic variables (e.g. education and occupation) in Model 3, then individual behavioral variables (e.g. physical activity and smoking) in Model 4, followed finally by the contextual predictors (Model 5). Using standard convention, we divide the models into fixed effects (predictor variables, the effect of which does not vary between individuals, families, wards, and municipalities) and random effects (the variance in outcome at each of these levels that remains after controlling for the fixed effects). We report these random effects in terms of the intraclass correlation coefficient (ICC) for the higher level variances. The ICC is the

percentage of the total unexplained variance that is attributable to each level in the model. Likelihood ratio tests were used to determine the statistical significance of the ICCs (see Rasbash, Steele, Browne & Goldstein 2009).

Results

Table 1 describes the characteristics of the sample and the contextual variables. Mean BMI increased by nearly 2 kg/m^2 during the 11 years of follow-up, and the standard deviation increased from 3.47 to 3.92. Mean age at baseline was 43 years. Males and females are equally represented and the majority were married. The variables measuring individual SES indicate that most individuals reported working in manual occupations and had a low educational level. Nevertheless, the majority did not report being physically tired or exhausted at the end of the working day. A third of the individuals were physically active in their leisure time, and a similar fraction were daily smokers.

Table 1. Sample characteristics

Individual level (N=24 966)	*Mean*	*SD*
Body Mass Index (BMI) 1984	24.79	3.47
Body Mass Index (BMI) 1995	26.70	3.92
Age (years)	43.1	10.8
Sex	*N*	*%*
Males (ref)	12,569	50.3
Females	12,397	49.7
Marital status		
Married (ref)	20,905	83.7
Not married	2,811	11.3
Divorced/Separated	717	2.9
Widow (-er)	533	2.1
Occupation		
Non-manual (ref)	5,302	21.2
Manual	18,348	73.5
No employment income	1,316	5.3
Education		
High (ref)	3,358	13.5
Low	21,608	86.5
Physically demanding work		
No (ref)	12,556	50.3
Yes	12,410	49.7
Physical activity (leisure time)		
Inactive (ref)	17,321	69.4
Active	7,645	30.6
Current smoker		
No (ref)	16,335	65.4
Yes	8,631	34.6
Contextual levels	*Mean*	*SD*
Family variables (N=17500)		
Income (Ln) (Kroner per annum)	10.13	3.69
Ward variables (N=447)		
High education level 1990 (%)	11.07	6.88
Municipality variables (N=24)		
Deprivation index 1989 (score)	36.54	4.44
Population change 1984-95 (%)	-4.02	7.66
Election participation pr.1000 (1983-91)	712.92	48.46

Compared to those excluded from the analyses because of missing information on the covariates, the included sample were younger, (43 vs. 46 years, $p<0.05$), more likely to be male, (50 vs. 41 percent, $p<0.05$), to be married (84 vs. 80 percent, $p<0.05$) to have a high education (14 vs. 8 percent, $p<0.05$), to be employed in a non-manual occupation (21 vs. 14 percent, $p<0.05$) and to be physically inactive (69 vs. 64 percent, $p<0.05$). There was no difference in terms of smoking status or in reporting physically demanding work. Included women had a slightly lower BMI than excluded women in both HUNT 1 (24.3 vs. 25.1 kg/m^2, $p<0.05$) and HUNT 2 (26.6 vs. 27.2 kg/m^2, $p<0.05$), whereas included men also had a slightly lower BMI than excluded men in both HUNT 1 (25.2 vs. 25.4 kg/m^2, $p<0.05$) and HUNT 2 (26.8 vs. 26.9 kg/m^2, $p<0.05$).

Table 2. The predictors of BMI in the sample

	Model 1		**Model 2**		**Model 3**		**Model 4**		**Model 5**	
	β		β		β		β		β	
Fixed Effects										
Individual level (intercept)	26.851		26.879		26.560		26.858		26.826	
Demography										
Age (years)			0.240	**	0.257	**	0.252	**	0.253	**
Age (years)^2			-0.002	**	-0.002	**	-0.002	**	-0.002	**
Female (yes/no)			-0.086		-0.745	**	-0.906	**	-0.911	**
Not married (yes/no)			0.208	*	0.208	*	0.229	**	0.227	**
Widow (yes/no)			0.079		0.027		0.093		0.097	
Divorced/Separated (yes/no)			-0.396	**	-0.430	**	-0.280		-0.265	
SES										
Manual employment (yes/no)					0.044		-0.014		-0.048	
No employment income (yes/no)					1.004	**	0.969	**	0.914	**
Low education (yes/no)					0.299	**	0.309	**	0.268	*
Female*low education (interaction)					0.638	**	0.817	**	0.834	**
Behaviour										
Physically demanding work (yes/no)							0.303	**	0.288	**
Active in leisure time (yes/no)							-0.491	**	-0.482	**
Current smoker (yes/no)							-0.853	**	-0.850	**
Family level										
Family income, Kroner per annum (Ln)									0.002	
Ward level										
High educational level 1990 (%)									-0.026	**
Municipality										
Deprivation index 1989 (score)									0.032	
Population change 1984-95 (%)									-0.018	
Election participation per 1000 1983-91									-0.002	
Random Effects										
Variance components#										
Municipality level	0.103	(0.043)	0.109	(0.045)	0.106	(0.043)	0.095	(0.039)	0.080	(0.034)
ICC (%)	*0.67*		*0.72*		*0.71*		*0.65*		*0.55*	
Ward level	0.191	(0.035)	0.186	(0.034)	0.154	(0.031)	0.126	(0.028)	0.090	(0.025)
ICC (%)	*1.24*		*1.23*		*1.03*		*0.86*		*0.61*	
Family level	2.377	(0.175)	2.069	(0.171)	1.994	(0.171)	1.911	(0.168)	1.927	(0.168)
ICC (%)	*15.45*		*13.72*		*13.34*		*12.99*		*13.14*	
Individual level	12.717	(0.193)	12.720	(0.192)	12.695	(0.192)	12.580	(0.192)	12.568	(0.190)
ICC (%)	*82.64*		*84.33*		*84.92*		*85.51*		*85.70*	
-2*loglikelihood:	138667.178		138198.204		138011.517		137656.051		137621.214	

*** p<0.05, ** p<0.01, # = All higher level variances are significant at a 0.01 probability level (likelihood ratio test).**

ICC = Intraclass correlation coefficient

Table 2 reports the unstandardised coefficients and ICC values from the models of BMI at the end of the study regressed on explanatory variables from baseline. Model 1, the null model, shows that about 83 % of the variation in BMI was at the individual level, 15 % between families and 1.24 % and 0.67 % between wards and municipalities respectively. Adjusting for sample demographics (Model 2) showed a quadratic association with age, whereby BMI was highest in middle age and lower amongst younger and older study participants. Incorporating the SES-variables (Model 3) showed higher BMI amongst respondents reporting no employment income. For the effect of education, a significant interaction with sex was present; men with a low level of education had on average a higher (0.299 kg/m^2) BMI than men with high education, with a greater effect for women, whereby those with a low education had a 0.937 kg/m^2 (0.299 + 0.638) higher BMI than their more highly educated counterparts.

In Model 4 the addition of the behavioural variables showed that smokers and those reporting being physically active during leisure time had lower BMI whereas reporting of physically demanding work was associated with higher BMIs. Model 5, after full adjustment for contextual variables, showed no association with family income but BMIs of participants were gradually lower amongst those living in wards with more educated populations. None of the municipality level variables reached statistical significance. Compared to the null model, the proportions of variance in BMI at the municipality, ward, and family levels were somewhat attenuated after full adjustment, but all remained statistically significant.

In Table 3 we present the models examining change in BMI during the 11 year follow-up and the modelling structure is the same as that of Table 2. Model 1 shows that 89% of the variance in BMI change was at the individual level, with approximately 8%, 1% and 2.5% associated with family, ward, and municipality levels respectively. The addition of demographic variables showed a quadratic relationship with age, that BMI gain in general was greatest in females, and in widows. The inclusion of SES measures (Model 3) showed higher gains amongst those with no employment income. Educational effects for men were small, but for women low education was associated with higher BMI change. Model 4 shows that those with physically demanding jobs gained more weight whilst physical activity outside work and smoking showed no associations with BMI change. The fully adjusted model showed no effect of family income, ward level of high education or any of the municipality level variables. After adjustment of these factors the ICCs for all four levels were relatively unchanged and still statistically significant.

In order to test for any moderating effects of area type we also performed sub-group analyses by stratifying the total sample according to degree of urbanisation in the municipality (urban versus rural). The models, not reproduced for the sake of brevity, were very similar to those presented for the whole sample. One exception was in the relationship between BMI and marital status, whereby in the urban sample the divorced/separated group had a lower BMI (0.524 kg/m^2, $p<0.05$) than the married group, whilst divorced/separated participants in the rural sample had a higher BMI (0.472 kg/m^2), although this was not statistically significant. A further difference was that BMI change was associated with ward level SES in the urban

sample only, with increasing SES being associated with a lower level of BMI increase (0.008 kg/m^2 less per unit increase in SES, $p < 0.05$) in this group.

Table 3. The predictors of BMI change in the sample

	Model 1		Model 2		Model 3		Model 4		Model 5	
	β		β		β		β		β	
Fixed Effects										
Individual level (intercept)	26.714		26.381		26.455		26.444		26.447	
Baseline BMI	0.953	**	0.997	**	0.997	**	0.996	**	0.996	**
Demography										
Age (years)			0.050	**	0.048	**	0.048	**	0.048	**
Age (years)^2			-0.001	**	-0.001	**	-0.001	**	-0.001	**
Female (yes/no)			0.683	**	0.433	**	0.409	**	0.407	**
Not married (yes/no)			-0.008		-0.005		-0.003		-0.001	
Widow (yes/no)			0.219	*	0.214	*	0.217	*	0.223	*
Divorced/Separated (yes/no)			0.014		0.011		0.008		0.015	
SES										
Manual employment (yes/no)					-0.003		-0.020		-0.024	
No employment income (yes/no)					-0.201	**	-0.206	**	-0.212	**
Low education (yes/no)					-0.082		-0.103		-0.108	
Female*low education (interaction)					0.313	**	0.340	**	0.343	**
Behaviour										
Physically demanding work (yes/no)							0.084	**	0.083	**
Active in leisure time (yes/no)							-0.023		-0.022	
Current smoker (yes/no)							0.008		0.009	
Family level										
Family income, Kroner per annum (Ln)									0.003	
Ward level										
High educational level 1990 (%)									-0.003	
Municipality										
Deprivation index 1989 (score)									0.028	
Population change 1984-95 (%)									-0.008	
Election participation per 1000 1983-91									-0.002	
Random Effects										
Variance components#										
Municipality level	0.107	(0.035)	0.118	(0.038)	0.118	(0.038)	0.119	(0.038)	0.104	(0.034)
ICC (%)	*2.38*		*2.80*		*2.81*		*2.83*		*2.48*	
Ward level	0.042	(0.009)	0.029	(0.007)	0.029	(0.007)	0.028	(0.007)	0.028	(0.007)
ICC (%)	*0.94*		*0.69*		*0.69*		*0.67*		*0.67*	
Family level	0.356	(0.050)	0.440	(0.047)	0.438	(0.047)	0.437	(0.047)	0.438	(0.047)
ICC (%)	*7.93*		*10.45*		*10.42*		*10.40*		*10.46*	
Individual level	3.984	(0.059)	3.622	(0.054)	3.619	(0.054)	3.618	(0.054)	3.618	(0.054)
ICC (%)	*88.75*		*86.05*		*86.08*		*86.10*		*86.39*	
-2*loglikelihood:	107681.039		105951.556		105922.524		105911.255		105906.363	

*** p<0.05, ** p<0.01, # = All higher level variances are significant at a 0.01 probability level (likelihood ratio test).**

ICC = Intraclass correlation coefficient

Discussion

In this study we have examined the correlates of BMI and BMI change in a large longitudinal sample of adult individuals. We found that both outcomes were associated with individual characteristics, with higher BMI most often found amongst males, unmarried participants, non-smokers, those of lower education and those undertaking physically demanding work but participating in less physical activity outside work. The characteristics of those in the sample

exhibiting higher BMI gain were rather similar except that women gained more and those with no employment income gained less.

The counter-intuitive finding, that those who reported physically demanding work gained more weight than those who did not, is not easily interpreted as we adjusted for a large number of confounders. It may be that this association is due to lower leisure-time physical activity along with less favourable eating habits outside the workplace for manual workers. As others have noted, it is also possible that relative weight gain is due to increased muscle mass for those reporting physically demanding work (Guitiérrez-Fisac, Guallar-Castillón, Díez-Gañán, García, Banegas, & Artalejo, 2002).

While previous studies have reported rather consistent and inverse associations between BMI change and both occupation (Martikainen & Marmot, 1999) and education (McLaren, 2007), we found modest relationships for BMI change. Taken together SES variables may relate to BMI and BMI change in different ways, where education is important in terms of knowledge of nutrition and exercise whilst occupation may have consequences for energy expenditure during the work day. Income may have an impact in terms of costs for healthy food and exercise opportunities. These associations are also likely to vary according to age, as well as between cohorts over time. In addition, as others have suggested, the SES-BMI pathway may be different for men and women and also dependent on childhood circumstances (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007). In this respect our results are generally consistent with previous reviews that have reported the strongest association with education, and an effect that is stronger for women (Sobal & Stunkard 1989, Ball & Crawford 2005, Mclaren 2007).

Although we only had information on one member of almost 60% of families in our sample, the fact that the sample size was large, and therefore had good power, meant that we were able to examine the effects of family, something which is rarely possible. Although family income was not associated with either outcome, we did find that between 10-15% of the unexplained variance was at the family level, suggesting that some unmeasured characteristics of families may be important. The fact that BMI is correlated within families suggests the presence of a complex interplay between a possible genetic susceptibility to weight gain and characteristics of the family environment such as shared eating and physical activity habits.

The random variance in both outcomes at both the ward and municipality levels was small but statistically significant, and of a similar magnitude that found in other studies (e.g. Lebel, Pampalon, Hamel &Therieult, 2009). The fact that the variability associated with the municipality level is larger for BMI change than for BMI is interesting, and worthy of further investigation. It suggests that some characteristics of either the social or physical environment of areas may be particularly important in driving the trends in weight gain, and also that previous cross sectional studies using a single time point may have underestimated the magnitude of environmental influences behind increasing obesity rates. Furthermore, none of the municipality-level explanatory variables were significantly associated with our two BMI outcomes. Previous studies have reported significant associations with both deprivation indices (van Lenthe & Mackenbach, 2002) and area social capital (Veenstra, Luginaah,

Wakefield, Birch, Eyles, & Elliott, 2005). However, the low number of municipalities in our study (N=24) does make it difficult to establish significant associations which may require large variation in exposure.

The neighbourhood SES variable (high educational level in the wards) was associated with BMI in the total sample and with BMI change in our urban subsample in the stratified analysis. These associations may be related to norms and practices around both diet and exercise in these neighbourhoods and may be viewed as components of collective lifestyles (Frohlich, Corin, & Potvin, 2001). These are not merely the behaviours individuals engage in, but also concern the relationship between individuals' social conditions and their social practices and they hence may vary according to different neighbourhood socioeconomic context.

Families, neighbourhoods and municipalities are not fixed quantities with enduring characteristics, but are dynamic settings or systems. To some extent our study has met recent calls to utilize a "relational" perspective in area effects studies (Cummins, Curtis, Diez-Roux, & Macintyre, 2007). A relational perspective means firstly the abandonment of the context – composition dichotomy, and favours a systems view. It also clearly recognizes the importance of scale or multiple levels. We have nevertheless still used the concepts of context and composition in order to be able to make a distinction between obesity-related determinants within and outside the individual here.

In the modelling of BMI and BMI change we followed a conventional approach by first fitting a variance component model and then adding individual level variables incrementally. This allows us to adjust the models for the composition of higher level units in order to assess the remaining variation between them. Like any other observational study these are difficult issues and separating context from composition is a vexing specification problem. On the one hand there is a risk of omitting important covariates or confounders. On the other, one may over-fit the models and include mediating covariates that are on the causal pathway between the contextual exposure and the outcome. Since primary key interest here was the ecological explanatory variables (and variances) we tested different versions of compositional adjustments (results not shown). It can be argued that the behavioural characteristics in model 5 may be mediating variables rather than confounders. This is especially true for physical activity in leisure time because we implicitly assume an indirect ecological effect (Blakely & Woodward, 2000) via obesity related behaviour on BMI. The conclusions regarding the fixed higher-level effects, and the higher level variances, will however remain the same regardless of model preference.

Our study has a number of strengths and weaknesses. Strengths include the availability of data from a large, well characterized cohort of individuals. We used anthropometric height and weight measurements to calculate BMI which is preferable. The large sample size meant we had the power to examine the effects of family, ward, and municipalities and by correct assignment of individuals to these various contextual units both in time and space, we were able to achieve a stable contextual exposure through eleven years. Other researchers have noted how it is important to recognise the various levels in a population, and the exclusion of

one level will affect the estimates (variance components) at other levels (Tranmer & Steel, 2001).

In terms of weaknesses, our family and ward level consists of data for the period 1992 to 1995 and not for the entire follow-up period; hence there is a possibility of incorrect assignment of individuals into these units where individuals have moved within municipalities or where family circumstances have changed. The characteristics of our sample are also less varied than the broader Norwegian population. In particular, the cohort is drawn from a single county and hence the heterogeneity in the physical environment is rather limited, which might explain our lack of findings regarding area effects. Furthermore, the study area has a very equal income distribution, even in an egalitarian Norwegian context (Elstad, Dahl, & Hofoss, 2005). It is also noteworthy that our exclusion criteria meant that a substantial proportion of the total HUNT cohort did not form part of our analysis. Although a comparison of included and excluded individuals did not reveal strong differences, those included tended to be younger, male, with a high education, and in a non manual occupation. Included individuals also had a lower BMI than their excluded counterparts. These differences may affect the generalisability of our findings. We excluded individuals who changed their area of residence in order to provide a stable sample for estimating area effects, although movers are in themselves an interesting group as they allow the effects of change in context to be examined. Whilst beyond the scope of this article, we are currently analysing change amongst this group.

Conclusion

In conclusion, we found that a statistically significant proportion of the variance in BMI and BMI change can be attributed to families, in addition to the individual. We were able to quantify the family components that determined our outcomes but insight from elsewhere does suggest that processes governing physical activity and nutrition in families are extremely complex (Kegler, Escoffery, Alcantara, Ballard, & Glanz, 2008). The small amounts of variance attributable to the ward and municipality levels show that features of the wider social and physical environment are likely to be important, yet relatively minor determinants of BMI in our study. This may in part be due to features of the Scandinavian welfare model, both in terms of social security arrangements and regional policy, which act to prevent strong geographical inequalities in health, although the magnitude of our findings is similar to that for others. There is thus a need for more research to examine both more specific pathways between contextual environments and bodyweight (Stafford, Cummins, Ellaway, Sacker, Wiggins, & Macintyre, 2007) in different macrosettings characterized by different institutional arrangements.

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Paper 4

The impact of family and place of residence on smoking behaviour in Norway – results from the Nord-Trøndelag Health Study

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Abstract

Tobacco control initiatives have steadily lowered the smoking prevalence in developed societies but further reductions seem increasingly difficult to achieve. A clear socioeconomic gradient in smoking is also evident in societies in the so-called matured stage of the smoking epidemic. The aim of the present study is to highlight new avenues for future tobacco prevention by investigating the sociodemographic, family and geographical distribution of smoking among adults in Norway. We utilise cross-sectional total population health data from the Nord-Trøndelag Health Study (N=50,535), and deploy multilevel models of individuals nested in families, wards and municipalities. We found that smoking was strongly correlated within families (ICC=39.1%, MOR=4.11) whereas the correlation within wards (ICC=1.07, MOR=1.26) and municipalities (ICC=1.12, MOR=1.27) was small. Smoking was strongly and inversely patterned by education with an odds ratio of 5.5 (95% CI 4.81 to 6.40) between highest vs. lowest category. Smoking was more prevalent among those unemployed/unfit to work, retired/on social security compared with being self employed and among divorced/separated compared with married individuals. There was a tendency towards higher risk of smoking in lower income families and relatively deprived municipalities. We conclude that families and households are important contextual units to understand smoking and this should be acknowledged in future tobacco prevention and policy. Secondly, the strong educational gradient in smoking suggests that future tobacco control efforts should have an explicit equity perspective.

Introduction

Smoking is currently responsible for more than 5,4 million annual deaths worldwide (WHO, 2009). Although the prevalence of smokers is decreasing in high income societies, it is increasing in low income countries and constitutes a public health problem along the entire advantageous-disadvantageous continuum at the global scale (Ezzati & Lopez, 2004). While the decision to smoke is an individual act there is reason to believe that smoking is contingent on memberships in various social contexts (Poland, Frohlich, Haines, Mykhalovskiy, Rock, & Sparks, 2006). Neglecting these contexts may cause preventive efforts to be limited. There is hence a need to assess to what extent context matters in terms of smoking.

Smoking is disproportionally higher among lower socioeconomic groups, at least in a number of western societies (Cavelaars, Kunst, Geurts, Crialesi, Grotvedt, Helmert et al., 2000; Huisman, Kunst, & Mackenbach, 2005a). This aspect is to some extent responsible for social inequalities in health in western societies. The contribution of smoking to social inequalities in mortality does however vary widely between populations (Mackenbach, Huisman, Andersen, Bopp, Borgan, Borrell et al., 2004; Van der Heyden, Schaap, Kunst, Esnaola, Borrell, Cox et al., 2009) and over time (Blakely & Wilson, 2005). Whilst previous, predominantly population level interventions obviously have had success, there are now difficulties in attaining further reductions in some western societies. Given the increasingly strong socioeconomic smoking gradient (Giskes, Kunst, Benach, Borrell, Costa, Dahl et al., 2005), a suggested policy response in Norway and other countries in the matured stage of the smoking epidemic (Lopez, Collishaw, & Piha, 1994) is to target the most disadvantaged groups (Huisman, Kunst, & Mackenbach, 2005b; Lund & Lund, 2005). This is done, firstly, in order to reduce the incidence of smoking-related diseases and premature mortality in the population, and secondly to narrow the health gap between socioeconomic groups in society. This is the high risk approach seeking to reach individuals and may be appropriate given such a distribution (Rose, 1992).

What this high risk approach may neglect however, is that these individuals still form parts of smaller social systems (Diez Roux, 2007), and that the likelihood of smoking initiation, or smoking cessation, may be highly dependent on which contexts the individual resides within. These contexts can be spatially delineated, they can be relationally separated, or a mixture of the two. A number of previous studies utilising what is considered appropriate statistical frameworks (Subramanian, 2004) have investigated smoking in geographical contexts using administrative boundaries to describe the neighbourhood or place of residence. Place deprivation (Duncan, Jones, & Moon, 1999; Shohaimi, Luben, Wareham, Day, Bingham, Welch et al., 2003) and related concepts of area disadvantage (Chuang, Cubbin, Ahn, & Winkleby, 2005; Chuang, Li, Wu, & Chao, 2007; Datta, Subramanian, Colditz, Kawachi, Palmer, & Rosenberg, 2006; Diez Roux, Merkin, Hannan, Jacobs, & Kiefe, 2003; Gray & Leyland, 2009; Karvonen, Sipila, Martikainen, Rahkonen, & Laaksonen, 2008; Pearce, Hiscock, Moon, & Barnett, 2009; van Lenthe & Mackenbach, 2006) have perhaps been the most commonly selected area characteristics under investigation. The underlying assumption

is that deprived areas exert a contextual effect on smoking through social norms and psychosocial stress levels (Datta et al., 2006).

Another potentially influential contextual unit concerns the family or household. While the number of studies using multilevel modelling is sparse, there is good reason to believe there is behavioural conformity and that smoking practices is clustered in families. A study utilizing quitting smoking as the outcome did find that members of the same household had similar quitting behaviours and that the mechanisms was related to the household level (Chandola, Head, & Bartley, 2004). Similarly, two studies conducted in India found smoking to be strongly correlated in households in adjusted models (Subramanian, Nandy, Kelly, Gordon, & Davey Smith, 2004a; Subramanian, Nandy, Kelly, Gordon, & Smith, 2004b), and yet another study showed that smoking was correlated between spouses (Clark & Etile, 2006). In addition, there are a number of studies using individual-level methodology reporting associations between parents and adolescence smoking (Fagan, Brook, Rubenstone, & Zhang, 2005), smoking initiation (Greenlund, Liu, Kiefe, Yunis, Dyer, & Burke, 1995), and cessation (Farkas, Distefan, Choi, Gilpin, & Pierce, 1999), as well as between spouses and subsequent cessation (Monden, de Graaf, & Kraaykamp, 2003). A recent review of health and health related behavioural concordance within couples is also supportive of the notion that smoking must be contextualised (Meyler, Stimpson, & Peek, 2007).

The aim of the present study is to investigate how individual smoking may be contingent on membership in higher level units, or systems, like the family, neighbourhood and municipality, while simultaneously accounting for the demographic and socioeconomic characteristics of individuals within them. Rather than adopting a classical analytical approach that investigates the association between contextual characteristics (e.g., social capital, area deprivation) and smoking, we focused on measures of variance and clustering. These measures help to identify the relevant collective boundaries that influence individual propensity for smoking: a knowledge of high relevance for identifying the appropriate level of intervention in preventive public health strategies (Merlo, Ohlsson, Lynch, Chaix, & Subramanian, 2009).

Methods

Study population

Nord-Trøndelag is one of 19 Norwegian counties and in 1995-1997 all 92,936 inhabitants aged > 19 years were invited to attend a clinical examination as part of a general health screening program, the second wave of the Nord-Trøndelag Health Study (HUNT 2). Of these, 64,945 (71%) answered a first questionnaire and attended the physical examination and were subsequently given a second questionnaire to be returned by mail after the examination. An evaluation of randomly selected non-participants showed that lack of time, no need for a physical examination and serious physical illness were the primary reasons for not attending

(Holmen, Midthjell, Krüger, Langhammer, Lingaas Holmen, Bratberg et al., 2003). The county is considered representative for the Norwegian population with the exception of two sociodemographic features; there are no major cities and a very homogeneous population in terms of ethnicity.

The present study, carried out in 2009, utilises questionnaire information from the participants combined with public registry information made possible through the Norwegian identification number that is unique for each individual residing in the country. From various registries, assembled in a dynamic social security database, we obtained information on place of residence (ward and municipality), income and a unique family number. From those attending, we initially excluded 6,123 individuals outside of the age range 25-80. In addition we excluded 1,685 individual (2,6%) due to difference in municipality coding between HUNT-data and registry-data, 221 (0,3%) due to error in ward coding, 39 (~ 0%) because of missing family number, and 46 (~ 0%) because of cross classifications in higher level units (i.e. individuals in the same family had different ward or municipality numbers). Furthermore we excluded 3,279 (5,0%) because of missing information on the smoking outcome and on the following covariates: marital status: 19 (~ 0%), education: 2,058 (3,2%) and current job situation: 940 (1,4%). The final analysed sample consists of 50, 535 individuals, which represent 78% of the sample. These individuals were nested in 34,032 families (Mean family size: 1.5), 442 wards (Mean number of inhabitants: 114) and 24 municipalities (Mean number of inhabitants: 2106).

Assessment of variables

Outcome: We obtained information on current smoking status from the smoking items present in the questionnaire: Do you smoke? And the questionnaire item: Cigarettes daily (yes/no).

Explanatory variables at individual level: age and age squared (both grand mean centred), sex (reference are males). Self-reported marital status: married (reference category), not married, widow(-er), divorced/separated. Self-reported education: basic school, secondary school, junior college, college/university less than four years, college/university more than four years (reference group). Self-reported current job situation: salaried employee, self-employed (reference group), full-time housework, undergoing education/military service, unemployed/temporary redundant, retired/on social security.

Explanatory variables at household level were derived from register information. Equivalent family income: net annual individual income in 1995 (in NOK) aggregated to family units and divided by the square root of the number of family members. Negative and missing incomes were set to zero. We classified income into four groups based on quartiles (i.e., highest, medium high, medium low, and lowest) and used the group with highest income as reference in the comparisons.

Explanatory variables at municipality level: Municipality deprivation in 1995 is an index derived from Statistics Norway consisting of the following six indicators (recipients of social

security benefits 20-40 years of age per 100 inhabitants, age and sex adjusted mortality per 100 000 0-64 years of age (averages 1984-1993), disability pensioners/occupational rehabilitants per 100 inhabitants 35 to 55 years of age, crime rate per 10 000 inhabitants (average for 1992-1993), unemployment rate 16 to 74 years of age, transitional beneficiaries per 100 women 20-39 years of age. The index, theoretically ranging from 1 to 10, was dichotomized into deprived and non-deprived (reference category) municipalities based on the average for the 24 municipalities.

Analyses

Given the hierarchical structure of the data, we applied four level binominal logistic regression (Goldstein, 2003). For the present analysis this framework allows the estimation of (a) the relationship between smoking and individual sociodemographic markers across higher level units (fixed parameters), (b) the variance in smoking risk between higher level units that is not accounted for by individual predictors (random parameters), (c) the contextual effect of higher level predictors on the individual risk of smoking (fixed parameters) and finally (d) possible remaining variance unaccounted for by higher level predictors (random parameters). The principles and rationale behind multilevel modelling is well described both in public health (Blakely & Subramanian, 2006; Diez Roux, 2000; Moon, Subramanian, Jones, Duncan, & Twigg, 2005; Subramanian, Jones, & Duncan, 2003) and social epidemiology (Merlo, Chaix, Ohlsson, Beckman, Johnell, Hjerpe et al., 2006; Merlo, Chaix, Yang, Lynch, & Rastam, 2005a, b; Merlo, Yang, Chaix, Lynch, & Rastam, 2005).

Parameters for our binary outcome were estimated with a logit link function in the MLwiN package v. 2.14 (Rasbash, Charlton, Browne, Healy, & Cameron, 2009) using MCMC methods (Browne, 2009) and were run for 100 000 iterations. We used starting values from a RIGLS second-order penalised quasi-likelihood (PQL) procedure. Estimates for fixed effects are reported as odds ratios (OR) with 95% confidence intervals (95% CI). Random effects are reported as (1) variances (on the log odds scale) along with 95% Bayesian credible intervals (CrI), which limits corresponds with the 2,5% and 97,5 % percentiles of the posterior distribution, as (2) intraclass correlation coefficients (ICC) and finally (3) as median odds ratios (MOR). The intraclass correlation coefficients (ICC) were calculated as the proportion of variance on a given contextual level divided by the total variance. Here we used the latent variable approach (Snijders & Bosker, 1999) that considers the variance from a standard logistic distribution ($\pi^2/3 = 3.29$) as the individual level variance.

The ICC was calculated as:

For level-4: $V_{level\text{-}4}/(V_{level\text{-}4} + V_{level\text{-}3} + V_{level\text{-}2} + 3{,}29)$
For level-3: $V_{level\text{-}3}/(V_{level\text{-}4} + V_{level\text{-}3} + V_{level\text{-}2} + 3{,}29)$
For level-2: $V_{level\text{-}2}/(V_{level\text{-}4} + V_{level\text{-}3} + V_{level\text{-}2} + 3{,}29)$

Here the $V_{level\text{-}4}$ is the variance at the municipality level, $V_{level\text{-}3}$ is the ward-level variance and $V_{level\text{-}2}$ is the family-level variance. The individual level variance is set to 3.29 and all these variances constitutes the total interindividual variance: ($V_{level\text{-}4} + V_{level\text{-}3} + V_{level\text{-}2} + 3{,}29$).

The MOR was calculated as:

$$\exp\left[\sqrt{(2 \times \sigma^2)} \times 0.6745\right] \approx \exp\left(0.95\sqrt{\sigma^2}\right)$$

(σ^2 = higher level variance)

The MOR is (like the ICC) a measure of between-context heterogeneity but with an additional advantage in that it translates the higher level variance to an effect measure in terms of an odds ratio (Larsen & Merlo, 2005; Larsen, Petersen, Budtz-Jorgensen, & Endahl, 2000). To understand the rationale behind the MOR one can imagine that we compared all possible pairs of persons with similar covariates but residing in different contexts. By using the higher level residuals we could compute the OR for each pair of persons with the subject with the highest odds always placed in the numerator. All these possible pairs would then yield a distribution of odds ratios and the MOR is the median of this distribution (Merlo et al., 2006). Put more simply, if MOR is equal to 1 there is no higher level variation and conversely, the higher the MOR, the more important are the contextual units for understanding individual level outcomes. Since MOR is in the form of an odds ratio it can be compared with other (fixed effects) odds ratios in the analysis hence it is very useful in epidemiologic terms (Merlo et al., 2006). To compare models we report the deviance information criterion (DIC). This goodness of fit measure is simultaneously accounting for additional model complexity; hence a lower value means a better fit (Spiegelhalter, Best, Garlin, & van der Linde, 2002).

Table 1. Summary information for individuals 25-80 years in the 1995-97 Nord-Trøndelag Health Study (N=50 535)

Individual level variables		
Mean age (SD)	49.8	14.6
	N	%
Current smoker		
Yes	15339	30.4
No	35196	69.6
Sex		
Males (ref)	24187	47.9
Females	26348	52.1
Marital status		
Married (ref)	33639	66.6
Not married	9787	19.4
Widow (-er)	3339	6.6
Divorced/Separated	3770	7.5
Education		
University>4 years (ref)	3958	7.8
College/University<4 years	5981	11.8
Junior college	3942	7.8
Secondary school	17773	35.1
Basic school	18921	37.4
Current job situation		
Self-employed (ref)	7536	14.9
Salaried employee	27129	53.7
Full-time housework	3119	6.2
Education/military service	726	1.4
Unemployed/temporary redundant	1036	2.1
Retired/on social security	10989	21.7
Family level variable (N=34 032)		
Family income (equivalized)		
1 Highest (ref)	12632	25
2	12634	25
3	12635	25
4 Lowest	12634	25
Municipality level variable (N=24)		
Non-deprived municipalities (ref)	12670	25.1
Deprived municipalities	37865	74.9

Results

Overall smoking prevalence in Nord-Trøndelag during the period 1995-1997 was 30% according our sample (Table 1). Table 2 shows both crude (model 1) and adjusted (model 2) fixed effect estimates in terms of odds ratios (along with 95% confidence intervals) and measures of higher level variances/heterogeneity in four-level models. In the unadjusted

(empty) model we found that approximately 1.8, 1.4 and 44% of the total variance in the probability of smoking was located at the municipality, ward and family levels respectively. This corresponds to median odds ratios of: 1.38, 1.33 and 4.89. The ICC in the adjusted model for municipalities was 1.12% (MOR=1.27), for the wards; ~1% (MOR=1.26) and for the family level: 39% (MOR=4.11). All higher level variances were conclusive according to the Bayesian 95% credible intervals (CrI) in the adjusted model. There was a curvilinear association with age where the probability of smoking was high for younger age groups and increased up to the age of circa 40 years and then decreased rapidly by increasing age. Females were more likely to be smokers than males (OR=1.18). Compared with married individuals those who were not presented a higher odds of smoking and the association was strongest for the latter (OR=2.75). However, the odds of smoking steadily increased by decreasing educational level, being five times higher in the group with low than in group with high educational achievement (OR=5.55). For work situation, with self employed as reference category, we found that those in paid work and full-time housework had in excess of 30% higher odds of smoking whereas there was a twofold odds of smoking for those undergoing education/military service (OR=2.06) and those who were retired or on social security (OR=2). Unemployed or certified unfit for work was associated with nearly three times higher odds of smoking (OR=2.74). Low family income was associated with 32% higher odds of smoking compared with the highest income group, whereas the association for the medium low and medium high was 1.17 and 1.24 respectively. Municipality deprivation was also associated with smoking; individuals living in deprived municipalities had nearly 40% higher odds of smoking compared with individuals in non-deprived municipalities. We also performed an identical subgroup analyses by restricting our sample to families with multiple family members (N=31 977). The results, for both fixed and random effects, (not shown) were similar as those for the total sample.

Table 3 shows the results from three-level models, individuals nested in families nested in wards, stratified on municipality urbanicity (rural versus urban based on population size). The models are similar as in table 2 with the exception of the omission of the random term at the municipality level and the municipality deprivation predictor. The random part of the unadjusted models shows that there was larger ward heterogeneity in rural municipalities (ICC=3.45, MOR=1.55) than in urban municipalities (ICC=0.87, MOR=1.25). At the family level there was an opposite tendency with larger variability between urban families (ICC=47, MOR=5.2) than rural families (ICC=42, MOR=4.6). This variability were reduced in the adjusted models where the percentage reduction (PCV = Proportional Change in Variance ((variance in unadjusted model ÷ variance in adjusted model) / variance in unadjusted model) * 100) at ward level in rural municipalities were 29% whereas in urban municipalities the corresponding figure were 35% The reduction in between-family variability was 20% for both the rural and urban sample. Fixed effects associations for rural and urban residents were not very dissimilar. The smoking gradient by education were somewhat steeper in the rural sample and the increased odds of smoking (OR=1.4) for widows (-ers) in the urban sample were not found among the rural individuals.

Table 2. Odds ratios (95% CI) and random effects (variances, ICC and MOR) for current smoking in the total sample (50 535 individuals nested in 34 032 families in 442 wards and in 24 municipalities).

	Model 1 § (Crude/empty)		Model 2 # (Adjusted)	
	OR	95% CI	OR	95% CI
Fixed effects				
Age (centred)	1.14	(1.13 to 1.16)	1.20	(1.18 to 1.22)
Age squared (centred)	0.99	(0.99 to 0.99)	0.99	(0.99 to 0.99)
Sex				
Male	1		1	
Female	1.22	(1.17 to 1.27)	1.18	(1.11 to 1.24)
Marital status				
Married	1		1	
Not married	1.36	(1.28 to 1.45)	1.37	1.25 to 1.50)
Widow	0.72	(0.65 to 0.80)	1.20	(1.03 to 1.39)
Divorced/separated	3.10	(2.85 to 3.36)	2.75	(2.44 to 3.09)
Education				
University>4 years	1		1	
College/university<4 years	1.62	(1.42 to 1.84)	1.67	(1.44 to 1.95)
Junior college	2.49	(2.17 to 2.85)	2.55	(2.16 to 3.00)
Secondary school	3.43	(3.07 to 3.85)	4.12	(3.59 to 4.73)
Basic school	3.31	(2.95 to 3.70)	5.55	(4.81 to 6.40)
Work situation				
Self employed	1		1	
Paid work	1.34	(1.25 to 1.44)	1.38	(1.27 to 1.51)
Full time housework	1.13	(1.01 to 1.27)	1.33	(1.15 to 1.55)
Education/military service	1.57	(1.30 to 1.90)	2.06	(1.60 to 2.65)
Retired/social security	0.84	(0.77 to 0.91)	2.01	(1.77 to 2.28)
Unemployed/unfit for work	2.94	(2.50 to 3.44)	2.74	(2.24 to 3.36)
Contextual predictors				
Family income				
1 Highest income (25%)	1		1	
2 Medium high (25%)	1.33	(1.24 to 1.42)	1.24	(1.11 to 1.40)
3 Medium low (25%)	1.23	(1.15 to 1.32)	1.17	(1.05 to 1.32)
4 Lowest income (25%)	1.36	(1.27 to 1.46)	1.32	(1.12 to 1.55)
Municipality deprivation				
Non-deprived	1		1	
Deprived	1.37	(1.12 to 1.69)	1.37	(1.10 to 1.70)
Random effects				
Municipality variance (95%CrI)	0.114	(0.05 to 0.23)	0.063	(0.02 to 0.14)
ICC (%)	1.82		1.12	
MOR	1.38		1.27	
Ward variance (95%CrI)	0.088	(0.06 to 0.12)	0.060	(0.04 to 0.09)
ICC (%)	1.41		1.07	
MOR	1.33		1.26	
Family variance (95%CrI)	2.769	(2.53 to 3.03)	2.194	(1.98 to 2.42)
ICC (%)	44.23		39.13	
MOR	4.89		4.11	
DIC:	57383.509		55039.871	

§ Fixed effects=crude (from RIGLS second order PQL), Random effects=empty model (MCMC: 100 000)

Fixed and random effects from adjusted model (MCMC: 100 000)

CrI, Credible interval

ICC, Intraclass correlation coefficient

MOR, Median Odds Ratio

DIC, Deviance Information Criteria

Table 3. Odds ratios (95% CI) and random effects (variances, ICC and MOR) for current smoking in rural and urban municipalities.

	Rural sample				Urban sample			
	Empty model		Adjusted model		Empty model		Adjusted model	
			OR	95% CI			OR	95% CI
Fixed effects								
Age (centred)			1.21	(1.18 to 1.23)			1.20	(1.17 to 1.22)
Age squared (centred)			0.99	(0.99 to 0.99)			0.99	(0.99 to 0.99)
Sex								
Male			1				1	
Female			1.18	(1.09 to 1.28)			1.17	(1.09 to 1.26)
Marital status								
Married			1				1	
Not married			1.27	(1.13 to 1.43)			1.48	(1.31 to 1.67)
Widow			1.02	(0.86 to 1.22)			1.40	(1.17 to 1.68)
Divorced/separated			2.74	(2.35 to 3.20)			2.82	(2.45 to 3.25)
Education								
University>4 years			1				1	
College/university<4 years			1.83	(1.45 to 2.30)			1.59	(1.33 to 1.91)
Junior college			2.73	(2.15 to 3.47)			2.45	(2.01 to 2.99)
Secondary school			4.24	(3.47 to 5.19)			4.10	(3.47 to 4.84)
Basic school			5.89	(4.78 to 7.25)			5.36	(4.50 to 6.38)
Work situation								
Self employed			1				1	
Paid work			1.48	(1.31 to 1.66)			1.31	(1.15 to 1.48)
Full time housework			1.48	(1.23 to 1.78)			1.21	(0.98 to 1.49)
Education/military service			2.25	(1.58 to 3.19)			1.92	(1.40 to 2.63)
Retired/social security			1.94	(1.65 to 2.29)			2.09	(1.75 to 2.50)
Unemployed/unfit for work			2.86	(2.21 to 3.70)			2.64	(2.00 to 3.49)
Contextual predictors								
Family income								
1 Highest income (25%)			1				1	
2 Medium high (25%)			1.23	(1.09 to 1.39)			1.26	(1.11 to 1.42)
3 Medium low (25%)			1.20	(1.06 to 1.35)			1.16	(1.03 to 1.31)
4 Lowest income (25%)			1.30	(1.15 to 1.46)			1.34	(1.19 to 1.52)
Random effects								
Ward variance (95%CrI)	0.208	(0.15 to 0.28)	0.148	(0.10 to 0.21)	0.055	(0.03 to 0.09)	0.036	(0.01 to 0.07)
ICC (%)	3.45		2.71		0.87		0.63	
MOR	1.55		1.44		1.25		1.20	
Family variance (95%CrI)	2.533	(2.18 to 2.89)	2.025	(1.75 to 2.33)	3.009	(2.47 to 2.95)	2.417	(2.09 to 2.77)
ICC (%)	42.00		37.07		47.36		42.09	
MOR	4.56		3.89		5.23		4.41	
DIC:	27340.555		26189.636		30056.074		28847.849	

CrI, Credible interval
ICC, Intraclass correlation coefficient
MOR, Median Odds Ratio
DIC, Deviance Information Criteria

Discussion

This study has shown that the clustering of smoking in municipalities and neighbourhoods are small whereas smoking is strongly clustered in families. Furthermore we found strong independent effects of education which showed a graded inverse relationship with smoking. Being divorced/separated was strongly associated with the outcome as well as being retired/on social security and unemployed/unfit for work. The contextual predictors, family income and municipality deprivation showed moderate associations. These findings suggest, firstly, that the context-dependent nature of smoking in families or households should be acknowledged in future preventive efforts. Secondly, there is a need to identify population level interventions that can reduce the educational gradient in smoking. The clustering of smoking in families was similar to the effect of highest versus lowest educational category.

These findings must however be interpreted in light of a number of study limitations. Firstly and crucially, the partitioning of variance into between-family variability (or within-family clustering) for a non-continuous outcome is complicated. The sparse cluster sizes, many of which are single-person families do cause loss of power since these clusters do not contribute to the between-family variability. We did however fit identical subgroup-models restricted to multiple-person families and the results were practically identical for the between-family variability and the other estimates in adjusted and unadjusted models. In addition, a number of previous studies have done similar modelling by partitioning variance into family or household variance for dichotomous outcomes (Chandola et al., 2004; Subramanian et al., 2004a; Subramanian et al., 2004b). Similarly, reporting clustering or between higher level variability for dichotomous outcomes is not straightforward, and consequently we have reported both variances, intraclass correlation coefficients (ICCs) and median odds ratios (MORs) in order to achieve comparability across studies.

Secondly, we have analysed current smoking behaviour cross-sectionally, and we have been unable to incorporate important issues like smoking initiation or smoking cessation. We have merely shown that smoking is strongly correlated in families, and whether this correlation is due to so-called positive assortative matching (e.g. smokers tend to marry smokers) or due to a shared environment is beyond our current scope. Previous studies are nevertheless supportive of the notion that quitting smoking is indeed a family issue (Chandola et al., 2004). Similarly, given that smoking behaviour usually is taken up early in life (Jefferis, Power, Graham, & Manor, 2004), it also remains equivocal whether smokers are less likely to educate, if education prevents smoking, or if the gradient emerges because of differential rates of quitting.

We followed a conventional procedure in model fitting, by first adjusting for the composition of individuals in the higher level units and then adding family income and place deprivation. Although family income did not show a stepwise or graded pattern as for education, the municipality deprivation variable was related to smoking. Based on previous findings (Riva, Curtis, Gauvin, & Fagg, 2009),we also specified an indicator of ward socioeconomic status

(level of high education from a different stand-alone data source) as well as an indicator of municipality urbanicity but none of these were related to smoking. In the stratified analyses, we did find that the ward variance was larger in rural municipalities than in urban municipalities and this may suggest a possibility for area based initiatives in some of these wards. We were unable to illuminate on exactly which ward characteristics such initiatives should be based upon and this may warrant further examination.

The two major findings, the educational gradient and the clustering at the family level, can to some extent be interpreted together. Bourdieu has shown quite eloquently how class position is continually reproduced in families, across generations, and also how a whole range of lifestyle factors are symbolic for these positions (Bourdieu, 1984). Health behaviour, including smoking, is hence very likely to be clustered in families while simultaneously serving as a way of defining a lifestyle that is socially appropriate to ones family's social position and providing a demarcation towards other social groups. The bottom line, according to Bourdieu, is that lifestyles are to some extent also socially inherited.

Acknowledging the limitations, the findings do have some bearings on future smoking policy and prevention. Firstly, families and households, not only individuals, should be acknowledged as an important unit of prevention. Secondly, traditional tobacco control policies targeting individual smokers may widen the educational gradient. Further initiatives should also incorporate an equity lens.

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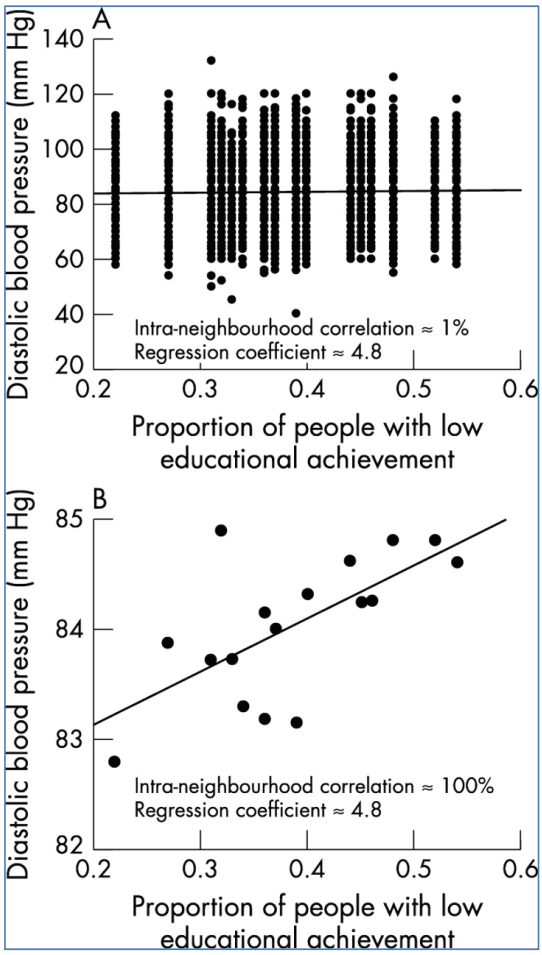
Appendices

Appendix 1 Illustration: how traditional measures of association may be misguiding area-based preventive efforts (from Merlo, 2003)



Figures (A) and (B) present two multilevel analysis showing the exact same association (regression coefficient, β = 4.8) between blood pressure and the proportion of people with low educational achievement in the neighbourhoods.

However, the size of the intra-neighbourhood correlation ranges from less than 1% (A) to 100% (B). In the first case (A) the neighbourhoods do not differ more than random samples taken from the whole population: the geographical environment has almost no effect on the individual outcome. In the second case (B), the clustering of persons in relation to blood pressure is total, and the geographical environment completely influences individual outcome.

Despite the large disparity in the size of the intra-neighbourhood correlation, the size of the regression coefficients is similar (that is β = 4.8) in both cases.

Comment: The figures underscore the importance of reporting measures of variance along with measures of association.

Appendix 2 Description of HUNT II (Holmen et. al. 2003)

Norsk Epidemiologi 2003; **13** (1): 19-32

The Nord-Trøndelag Health Study 1995-97 (HUNT 2): Objectives, contents, methods and participation

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ABSTRACT

The second Nord-Trøndelag Health Study in 1995-97 (HUNT 2) was partly a follow-up study of HUNT 1, conducted in 1984-86. HUNT 2 comprised, however, a larger scientific program. The large amount of information collected from each participant, and the large number of participants in a wide age range covering an entire county population, make HUNT one of the largest health studies ever performed. This paper describes the survey covering persons aged 20 years and older. In total, 66.7% of men (n=30,860) and 75.5% of women (n=35,280) participated, the highest participation was in age group 60-69 and the lowest among the young and the elderly. Data collected from several questionnaires and with blood and urine samples and various clinical measurements, some of them in sub-samples of the study population, comprise a huge database for research. All data for each person are linked, and data are also linked to various health registries; all data handling being supervised by The Data Inspectorate and The Regional Ethical Committee. Procedures for data access are established, and more than 100 researchers are currently working on HUNT data. A large number of scientific papers in various disciplines are published, among them 15 doctoral theses (June 2003). The research potential of the HUNT biobank is still not fully exploited, but initiatives are taken. In line with other population based studies both in Norway and abroad, there was a decline in participation rate from HUNT 1 to HUNT 2 (16.9%). This has raised concern about the validity of future population based health studies. However, the good local and national network and the support from the population, make up a good platform also for future health studies in Nord-Trøndelag.

Key words: Health survey, methods, participation, epidemiology, cardiovascular disease, hypertension, diabetes, lung disease, osteoporosis, depression, anxiety, hemochromatosis, hearing loss, headache, migraine, prostate, women's health

INTRODUCTION

The first large health survey in Nord-Trøndelag County, Norway (later called the HUNT 1 study) was conducted during 1984-86. It was primarily designed to cover four sub-studies, i.e. on hypertension, diabetes, lung diseases and quality of life. The main objectives were to determine the prevalence of hypertension, diabetes and undiagnosed tuberculosis, and to evaluate the quality of health care provided to hypertensive patients, persons with diabetes, and persons with tuberculosis. Blood pressure, body height, and weight were measured and a miniature chest x-ray was taken. Each participant completed at least two questionnaires. Additionally, non-fasting blood glucose was measured in participants 40 years and older. Persons whose blood test could indicate diabetes were, along with a control group, offered a clinical evaluation. All participants with clinical findings indicating pathology were advised to see their family doctor, who also received clinical results from the health study itself. Venous blood samples were not taken, except in known and newly detected persons with diabetes and in a control group. In total, 74,599 persons aged 20 and older participated (88.1%). The methods applied in HUNT 1 are described in detail elsewhere[1], and so is a comprehensive non-responder study[2,3]. Several studies based on HUNT 1 are published, among these are studies on cardiovascular disease[4-17], diabetes[6,18-23], quality of life[24-29], cancer[30-34] and other topics[35-40].

HUNT 2 (1995-97): OBJECTIVES

The main objectives in HUNT 2 were aimed at the large public health issues like cardiovascular disease, diabetes, obstructive lung disease, osteoporosis and mental health, in concordance with current priorities of the health authorities. Several researchers and research groups presented a wide range of additional scientific questions, some of which were included in the final protocol. The result was a comprehensive health study covering a wide range of topics. The Young-HUNT Study aimed at age group 13-19 was organized separately and is described elsewhere[41,42]. This paper gives

an overview of the population, the contents, the methods applied and the participation in the HUNT 2 study for those aged 20 years and older.

STUDY AREA AND POPULATION

Nord-Trøndelag County is located in the middle of Norway at a latitude of 64 degrees north, and is divided into 24 administrative areas, i.e. municipalities (Figure 1). The county is mostly rural and sparsely populated; the largest of six small towns has a population of 21,000. The average income, the prevalence of higher education, and the prevalence of current smokers are a little lower than the average of Norway. In most respects, however, Nord-Trøndelag County is fairly representative of Norway, for example regarding geography, economy, industry, and sources of income, age distribution, morbidity and mortality.

Due to the Gulf Stream the climate is milder than in other areas of the same latitude. The coastal climate has precipitation as rain in fall, spring, and summer, and snow or rain in winter, but some inland areas have cold, dry winters. During mid-summer there is daylight all night, but days during mid-winter are as short as 5-6 hours, and there may be frost from November through March. Some basic data about Nord-Trøndelag County is given in Table 1.

The population in Nord-Trøndelag County (127,000 residents) is stable, with a net out migration of 0.3% per year (1996-2000), and homogenous (less than 3% non-Caucasian), making it suitable for epidemiological studies.

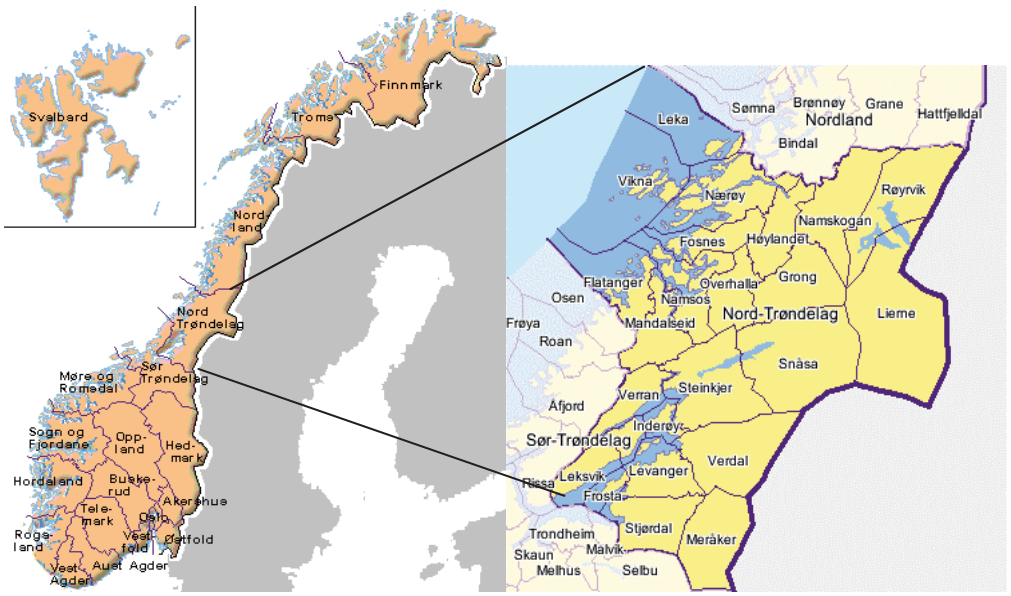
As in HUNT 1 (1984-1986) every individual residing in the county at the age of 20 and older was invited to participate in the HUNT 2 study. Several sub-studies in HUNT 2 were aimed at the elderly, and there was no upper age limit. Some additional sub-studies used other population samples, such as randomized samples, certain age groups, sex-specific samples or samples restricted to certain municipalities.

METHODS

Invitation letter and questionnaires

All residents of Nord-Trøndelag County, aged 20 years (reaching 20 years during the year of the screening in their municipality) and older were invited to the health survey in the two-year period from August 1995 to June 1997. The invitation file was created from periodically updated census data from Statistics Norway. The invitation letter was sent by mail attached to a three-page questionnaire (questionnaire 1) and an information folder. The questionnaire was to be completed prior to the screening and returned at attendance to the screening site. A second questionnaire (questionnaire 2) was handed out at the screening site and should be completed and returned by mail free of cost for the participant. A wide range of topics was addressed in questionnaire 1 and 2:

Figure 1. Norway and Nord-Trøndelag County.



- Health: Subjective health, diabetes[18,20,23], lung diseases[43-45], cardiovascular diseases, thyroid diseases[46-49], muscle- and skeletal diseases[50,51], mental diseases (especially anxiety and depression)[48,51-55], quality of life measures, migraine and other headaches[49,55-65], and physical and mental dysfunction, prostate complaints[66,67], quality of life, urine incontinence[68-72], and female reproductive data i.e. on menarche, pregnancies, hormone use, and gynecological diseases.
- Personal environment: Residence, size of household, education, occupation[73-77], in-house environment, neighborhood, friends, and sense of humor.
- Personal habits, like food intake, use of drugs, use of alcohol and tobacco[70,78,79], and physical activities.
- Family medical histories and health services consumption.

Additional questionnaires were used in sub-samples, and are described elsewhere, i.e. questionnaires on lung diseases[42], diabetes[18,42], hypertension[42], hearing disorders[80-84], and vision[85]. Some selected groups were invited to a more detailed examination as part of a phase 2 examination, i.e. participants in studies on diabetes[18], prostate[66,67], headache[57-62,86], lung function[43-45] and bone densitometry[78,79] (Figure 2).

Table 1. Selected statistical data on Nord-Trøndelag and Norway (From the Norwegian Meteorological Institute and Statistics Norway).

	Nord-Trøndelag (Verdal, Reppe)	Oslo (Blindern)	Tromsø
Climate *(Normal)*			
Mean air temperature (centigrades)	4.4	5.7	2.5
Precipitation (mm/year)	910	763	1,031
		Nord-Trøndelag	Whole country
Geography			
Total area (km^2)		22,463	323,877 (*)
Population *(2000)*			
Total population		127,108	4 478,497
Population density/km^2		6	15 (*)
Population (%)			
- in sparsely populated areas		44	23
- in densely populated areas < 2000 inhabitants		20	11
- in densely populated areas ≥ 2000 inhabitants		36	66
Financial situation (1996) Assessed mean income (brto) NOK			
Males		189,100	227,600
Females		116,900	132,300
Infant mortality (1996)			
Deaths under 1 year of age/1000 live births		5.0	4.1
Induced legal abortions (1996)			
(Per 1000 women 15-49 year)		11.5	13.4
Kindergartens (2002)			
Children in kindergartens *(Per cent 1-5 years)*		68.6	65.9
Education (2001) *(Per cent)*			
Below upper secondary level[1]		22.1	21.2
Upper secondary education[2]		60.5	56.6
Tertiary education, short[3]		14.8	17.5
Tertiary education, long[4]		2.7	4.8
Mortality *(Standardized rates. Deaths /100,000 population, average 1996-2000)*			
Deaths, all causes			
Males		1,032	1,086
Females		640	651
Diseases of the circulatory system (I00-I99)			
Males		475	459
Females		277	261
Malignant neoplasm (C00-C97)			
Males		249	282
Females		168	177

(*) Main land, i.e. Spitzbergen excluded.

[1] Not including persons with unknown or no completed education.

[2] Including the level 'Intermediate level' which comprises education based on completed upper secondary level, but which are not accredited as tertiary education.

[3] Tertiary education, short comprises higher education 4 years or shorter.

[4] Tertiary education, long comprises higher education more than 4 years.

Clinical measurements

*S*creening sessions were performed by the two teams (see Appendix) visiting each municipality of the county, with ordinary opening hours between 10 a.m. and 6 p.m. (occasionally from 8 a.m. to 7 p.m.). All clinical examinations were performed indoors at comfortable room temperature. The team surveying the five largest municipalities used more extensive standard office facilities; the other team working in the 19 smaller municipalities used a large, well-equipped trailer with efficient temperature regulation and other modern facilities.

Blood pressure and heart rate were measured by specially trained nurses or technicians using a Dinamap 845XT (Critikon) based on oscillometry. Cuff size was adjusted after measuring the arm circumference. The Dinamap was started after the participant had been seated for two minutes with the cuff on the arm, and the arm resting on a table. Blood pressure and heart rate were measured automatically three times at one-minute intervals. Blood pressures reported in most papers is the mean of the second and third systolic and diastolic blood pressures. Blood pressures measured with the Dinamap device are slightly lower than those measured with a sphygmomanometer, especially for diastolic blood pressure[87].

Height and weight were measured with the participants wearing light clothes without shoes; height to the nearest 1.0 cm and weight to the nearest 0.5 kg.

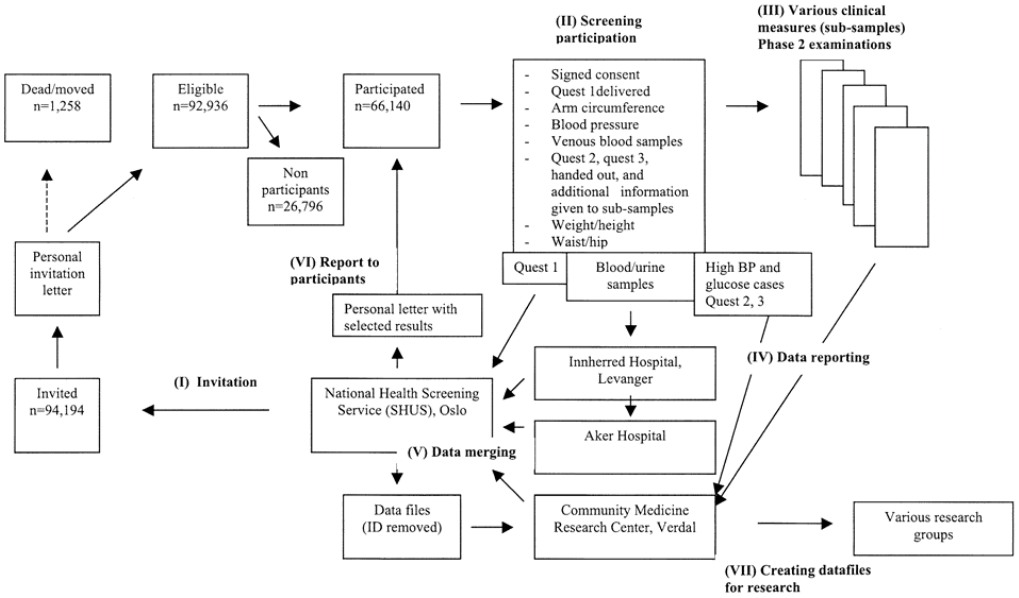


Figure 2. Procedures for invitation, screening, data reporting and creating of research data files. HUNT 2 (1995-97).

Step I: Invitation. National Health Screening Service (SHUS) created invitation letters based on data files from Statistics Norway, including all residents in Nord-Trøndelag County aged 20 years and older. Invitations were mailed a few weeks before the time of screening in the resident's community. The invitation file was regularly up-dated from Statistics Norway.

Step II: Screening participation. All participants went through procedures described in the box.

Step III: Various clinical measures. Phase 2. Participants were selected to various additional clinical measures: bone densitometry, spirometry, hearing, vision. A number of sub-samples were also invited to phase 2 clinical examination: Hemochromatosis, prostate, headache, spirometry, microalbuminuria, hypothyreosis, depression.

Step IV: Data reporting. Questionnaire 1 was sent to SHUS, and blood and urine samples to Innherred Hospital, witch forwarded selected samples to Aker Hospital. Extremely high BP and glucose readings were reported immediately to the Community Medicine Research Center, Verdal, who also received questionnaires 2, 3, and various data sets from additional clinical measurement.

Step V: Data merging. All data were sent to SHUS, where data files were merged.

Step VI: Reports to participants. Based on merged data files from questionnaires, clinical measurements and serum analyses, each participant was mailed a personal report with selected results.

Step VII: Creating data files for research. Data files with removed ID were sent to Community Medicine Research Center (Now: HUNT Research Center), Verdal, where appropriate research files are created and distributed to various research groups based on accepted protocols.

Waist and hip circumferences were measured with a steel band to the nearest 1.0 cm with the participant standing and with the arms hanging relaxed. The waist circumference was measured horizontally at the height of the umbilicus, and the hip circumference was measured likewise at the thickest part of the hip.
Additional clinical measurements were performed in sub-samples, and are described more in detail elsewhere: Lung function[43,44], bone densitometry[78,79], hearing[80-84], vision[85], headache/migraine[49,55-62,64,65], ankle blood pressure (Doppler technique)[88] and sensibility in the foot[18]. In addition some studies and clinical follow-up procedures will be described in papers in progress.

Blood sampling

Blood sampling was done whenever subjects attended, i.e. in non-fasting or "random" state. In the period from August 1995 to June 1996 7.5 ml whole blood was drawn, serum was separated by centrifuging at the screening site and immediately placed in a refrigerator. The samples were sent in a cooler to the Central Laboratory at Levanger Hospital, Levanger, the same day or within two to three days (for example in weekends). Serum analyses were performed in fresh blood samples, and the remaining serum and clot stored in the biobank at minus 70 °C. From August 1996 to June 1997 routines were changed: An additional 5 ml of blood was drawn in an EDTA tube. This EDTA whole blood was stored in the biobank instead of the clot. The content of the HUNT biobank is described in Table 2.

Table 2. The HUNT 2 biobank, contributed by participants 20 years and older at HUNT 2, 1995-97. Stored at minus 70°C. DNA extraction is ongoing (Status per June, 2003).

	N	Volume ml (approx)	Extracted DNA N
Serum	65,291	1.5[1]	
Whole blood			
- EDTA	29,875	3-4	7,633[2]
- Clots	32,789	3-5	1,810
DNA samples, total	62,664		9,443[3]

[1] All serum in one tube. Serum is already used for analyses in a few studies, i.e. the volume is smaller in some population samples.
[2] After DNA extraction, left whole blood is stored in separate tubes for CONOR (0.4 ml x 4) and HUNT (0.4 ml x 4)
[3] Extracted DNA is stored in two separate tubes.

Laboratory procedures

Serum samples were analyzed at the Central Laboratory at Levanger Hospital, on an Hitachi 911 Autoanalyzer (Hitachi, Mito, Japan), applying reagents from Boehringer Mannheim (Mannhein, Germany). Glucose was measured by using an enzymatic hexokinase method, total cholesterol and HDL cholesterol applying an enzymatic colorimetric cholesterolesterase method, and HDL cholesterol was measured after precipitation with phosphortungsten and magnesium ions. Triglycerides were also measured with an enzymatic colorimetric method, and serum creatinine by Jaffè method. The day-to-day coefficients of variation were 1.3-2.0% for glucose, 1.3-1.9% for cholesterol, 2.4% for HDL cholesterol, 0.7-1.3% for triglycerides and 3.5% for creatinine[18].

In those who confirmed to have diabetes in questionnaire 1 an extra tube of whole blood was drawn in a 5 ml EDTA Vacutainer tube for analyses of HbA1c at Levanger Hospital. Those confirming diabetes were also re-invited to another blood sampling in the fasting state. Additional information was recorded by the nurses performing the sampling (year of diagnosis, type of treatment, state of insulin treatment, name of their GP). Fasting glucose was measured at the site applying a Hemocue. C-peptide and anti-GAD were measured in the fasting state and analyzed at Aker Hospital, Oslo. C-peptide was measured with a radioimmunoassay method (Diagnostic System Laboratories, Webster, TX), anti-GAD was measured via immunoprecipitation by using [^{3}H] leucine translation-labeled GAD65 as an indicator. Reagents were supplied by Novo Nordisk Pharma AS (Bagsvaerd, Denmark). The results of the C-peptide and anti-GAD analyses were mailed to the HUNT Research Center in Verdal, and forwarded to the GP after comments were attached. Additional analyses performed are described in detail elsewhere: Thyroid function[46,47], ferritin[61,89-94], and urine analyses for microalbuminuria[95-97].

Reporting of test results

Some individuals with extremely high blood pressures (DBP≥125 mmHg) were recommended to visit their GP for re-check the same day or within a few days. The HUNT Research Center was informed, and ascertained that follow-up took place. Likewise, non-fasting blood glucose readings ≥11.1 mmol/l were reported to the HUNT Research Center, who immediately informed the screened person by letter, with a copy to the GP.

About three weeks after the health examination each participant received a letter reporting some selected test results from the screening, like blood pressure, glucose, total cholesterol, height, weight, self reported physical activity, ferritin (part of the hemochromatosis screening). Included were also some advice regarding healthy food, smoking and physical exercise. If the above mentioned tests or the screening for anxiety and/or depression disclosed possible pathology, the participant was advised to see his/her doctor for re-check.

PERSONAL PROTECTION AND ETHICS

Both the core study and each sub-study were approved by the Data Inspectorate of Norway and recommended by the Regional Committee for Medical Research

Ethics, and all information from HUNT is treated according to the guidelines of the Data Inspectorate.

Participation in the HUNT study was voluntary, and each participant signed a written consent regarding the screening, subsequent control and follow-up, and to the use of data and blood samples for research purposes. They also consented to linking their data to other registers (subject to approval of the Data Inspectorate). When the data files have been prepared for research purposes, all names and personal ID numbers have been removed.

In Norway, every individual has a unique 11-digit personal identification number given at birth, and HUNT data are linked to the identification number, allowing cross reference of individuals in both HUNT databases and other regional and national health registries. Such linkage has been performed (for specific sub-studies and after appropriate approvals) to insurance statistics from the National Insurance Administration, to the Cancer Registry, the Medical Birth Registry, Census Data of Norway, the Family Registry and to the Cause-of-Death Registry.

At the time of HUNT 2, no study involving genetic DNA-based research was included. Therefore, an extensive information campaign about functional genomic research was performed in 2002, addressing the entire population of the county. Each surviving adult HUNT 2 participant (n=61,426) received an information folder and a personal letter asking for re-consent to include genetic research. Information was also given by mass media and by a specially designed web-site. In total, 1,185 (1.9%) persons withdrew their consent[98]. The re-consent project was also approved by the Data Inspectorate of Norway and recommended by the Regional Committee for Medical Research Ethics.

PARTICIPATION

In total, 94,194 individuals aged 20 years and older were invited to the HUNT 2 study based on a data file from Statistics Norway. The file was updated at regular intervals before invitations were sent out. Despite these routines, 1,258 were dead or had moved out of the county when the screening team arrived, making a total of 92,936 eligible for participation (Figure 2). Out of these 66,140 participated (71.2%) (Table 3). In all age groups under 70, more women than men participated (75.5% versus 66.7%). The participation was strongly age dependent, with the highest participation in the age group 60-69 for both sexes (84.3% in men and 87.0% in women), and gradually lower participation rate in younger and older age groups. Men aged 20-29 had the lowest participation (42.5%).

As the health survey included several stages, from filling in questionnaire 1 to a number of additional clinical tests and interviews (Figure 2), the term participation could have different meanings. In Table 3 participation is defined as having at least filled in questionnaire 1. In all, 807 individuals who filled in questionnaire 1 did not attend the health examination, and some did not attend all parts of the program. Additionally, some people did not fill inn all questions in the various questionnaires, resulting in different numbers of valid responses in different parts of the database. An overview is shown in Figure 2 (flow chart). For details, see the HUNT website[42] or papers in the reference list. An overview of the program in different population groups is demonstrated in Table 4.

Compared to HUNT 1 the participation in HUNT 2 had decreased by 16.9% (Table 5), most pronounced in men and in both sexes in young age groups (Figure 3).

Table 3. Participants at HUNT 2 by age and gender.

	Men				
	Invited	Dead/ moved	Could have participated	Participated	
Age	n	n	n	n	%
20-29	9,522	94	9,428	4,009	42.5
30-39	8,820	65	8,755	5,417	61.9
40-49	9,096	42	9,054	6,511	71.9
50-59	7,066	34	7,032	5,418	77.0
60-69	5,212	31	5,181	4,366	84.3
70-79	4,803	78	4,725	3,776	79.9
80-89	2,075	253	1,822	1,242	68.2
90+	288	58	230	121	52.6
Total	46,882	656	46,226	30,860	66.7
	Women				
	Invited	Dead/ moved	Could have participated	Participated	
Age	n	n	n	n	%
20-29	8,670	73	8,597	4,819	56.0
30-39	8,176	35	8,141	6,133	75.3
40-49	8,595	13	8,582	7,058	82.2
50-59	6,765	12	6,753	5,787	85.7
60-69	5,443	13	5,430	4,723	87.0
70-79	5,707	32	5,675	4,534	79.9
80-89	3,338	308	3,030	1,960	64.7
90+	618	117	501	266	52.8
Total	47,312	603	46,709	35,280	75.5
	Both genders combined				
	Invited	Dead/ moved	Could have participated	Participated	
Age	n	n	n	n	%
20-29	18,192	167	18,025	8,828	49.0
30-39	16,996	100	16,896	11,550	68.3
40-49	17,691	55	17,636	13,569	77.0
50-59	13,831	46	13,785	11,205	81.2
60-69	10,655	44	10,611	9,089	85.6
70-79	10,510	109	10,401	8,310	79.9
80-89	5,413	562	4,851	3,202	66.0
90+	906	175	731	387	52.9
Total	94,194	1258	92,936	66,140	71.2

Non-participation study

Shortly after completing the field work in 1997, a 2.5% random sample of non-attendants was selected (n=685) for a non-participation study[43]. The aim was to investigate the reasons why they did not attend. Of the 226 individuals we reached by telephone, 173 (76.5%) responded positively to being interviewed. A

Table 4. HUNT 2 (1995-97): Summary of screening programme. Number of valid cases may differ from number of participants due to missing values.

	Age and sex groups					Other selected groups (*)			
	1 13-19 M/F	2 20-69 F	3 20-69 M	4 70+ F	5 70+ M	6 5% rs	7 Resp. probl.	8 Hyper- tension	9 Diabe- tes
Number of participants	9,139	28,520	25,721	6,760	5,139	2,792	12,955	8,937	2,102
Questionnaire for adolescents	x								
Questionnaire 1 (same for all groups)		x	x	x	x	x	x	x	x
Questionnaire 2 (age and sex specific)		x	x	x	x	x	x	x	x
Questionnaire 3 – lung							x		
Questionnaire 3 – hypertension								x	
Questionnaire 3 – diabetes									x
Questionnaire – hearing		x	x	x	x	x	x	x	x
Heigth/weight	x	x	x	x	x	x	x	x	x
Sitting height	x					x	x		
Hip/waist	x	x	x	x	x	x	x	x	x
Blood pressure/heart rate	x	x	x	x	x	x	x	x	x
Bone mass (radial)						x	x		
Spirometry	x					x	x		
Hearing test		x	x	x	x	x	x	x	x
Vision		(x)	(x)						
NO (Nitrogen Oxide) expiration test	(x)						(x)		
Stored blood sample (biobank)		x	x	x	x	x	x	x	x
Total cholesterol		x	x	x	x	x	x	x	x
HDL cholesterol		x	x	x	x	x	x	x	x
Triglycerides		x	x	x	x	x	x	x	x
Glucose		x	x	x	x	x	x	x	x
Creatinine		x	x	x	x	x	x	x	x
Se-ferritine		x	x	x	x	x	x	x	x
TSH (Thyroidea Stimulating Hormone)		>40 yrs	50%>50 yrs	x	x	x			x
Microalbuminuria						x		x	x

(*) Group 6: 5% random sample (rs) of participants 20 years and older
Group 7: Reporting respiratory problems
Group 8: Taking antihypertensive drugs
Group 9: Reporting to have diabetes (included are also some participants recruited in the sub-study for the elderly)

(x) indicates sub-samples

Table 5. Participation at HUNT 1 (1984-86) by age and gender[1].

	Men			Women			Both sexes		
	Eligible	Participated		Eligible	Participated		Eligible	Participated	
Age	n	n	%	n	n	%	n	n	%
20-29	7,580	5,513	72.7	6,750	5,481	81.2	14,330	10,994	76.7
30-39	9,199	7,956	86.5	8,570	7,987	93.2	17,769	15,943	89.7
40-49	6,762	6,093	90.1	6,482	6,160	95.0	13,244	12,253	92.5
50-59	6,009	5,557	92.5	5,868	5,595	95.3	11,877	11,152	93.9
60-69	6,595	6,164	93.5	6,699	6,302	94.1	13,294	12,466	93.8
70-79	4,474	4,016	89.8	5,363	4,753	88.6	9,837	8,769	89.1
80-89	1,708	1,306	76.5	2,454	1,775	72.3	4,162	3,081	74.0
90+	205	119	58.0	382	200	52.4	587	319	54.3
Total	42,532	36,724	86.3	42,568	38,253	89.9	85,100	74,977	88.1

letter with a short questionnaire was sent to those not reached by phone (n=459). The reasons for not reaching them by telephone were: 335 did not have a telephone, 61 were not at home, 25 had moved out of the county, 33 were dead, and 5 gave no reason. In all, 153 (33%) answered the questionnaire, leaving a total of 326 individuals (47.6%) to be included in the analyses.

In age group 20-44 the main reasons for not attending the health survey were lack of time or having moved out of the county (54%). In age group 45-69

the main reason was busy in job or they had forgotten the invitation or had no reason. In age group 70+ many reported to have regular follow-up by a doctor or hospital and therefore did not need to attend the health survey (Table 6). Some people (9.6%) could not attend because they were immobilized due to disease, and some (4.1%) refused due to long waiting time at the screening site. A few (8.6%) reported that the health survey was unnecessary or that they were unwilling to participate[43].

DISCUSSION

Compared to other population studies HUNT has several special features: It covers a total population within a geographical area, it has a wide age range, it covers an extensive range of topics (nearly 3000 variables), and it has a high participation rate. HUNT 2 was a follow-up of HUNT 1 with identical or similar questions and assessments on hypertension, diabetes and quality of life. HUNT 2 was, however, much more comprehensive, with a wider age range (13 years old and over) and included more data on each participant. Even if the participation rate in HUNT in general was fairly high compared to most other studies in Norway and abroad[99-102], there is always a potential selection problem. In HUNT 2 data from young age groups, especially in men, should be analyzed with some caution. However, a comprehensive non-participation study after HUNT 1 could not find evidence of selection in health measures in young age groups[2,3]. Old non-participants, however, had significantly more health problems than participants of the same age.

The concerted action from research groups, authorities and the population has resulted in a huge database and has initiated extensive research activities. HUNT is part of CONOR (Cohort Norway)[101], which is a network of Norwegian health studies and biobanks with identical core variables, enabling linkage of databases to achieve a larger and an even more representative population with increased statistical strength. Through several years HUNT has initiated collaboration with various research groups in other European countries and in the USA. Procedures for data access are established, and more than 100 researchers in Norway and abroad are currently working on HUNT data covering studies within a wide range of medical topics. Up to now, HUNT has been the basis for about 150

Figure 3. Participation at HUNT 1 and Hunt 2 by age in men (M) and women (F).

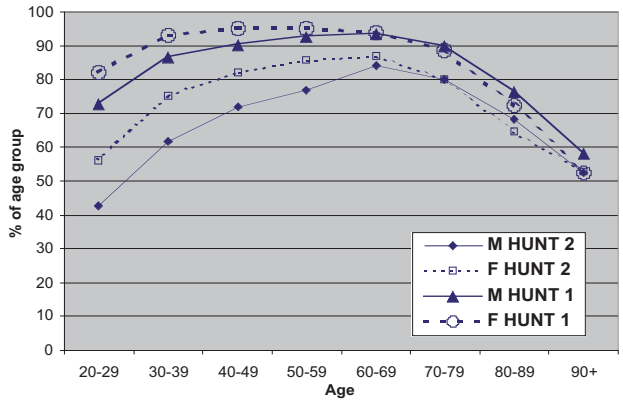


Table 6. Reasons for non-participation in HUNT 2 [43]

	20-44 years		45-69 years		70+ years		Total	
Reasons for non-participation	n	%	n	%	n	%	n	%
Follow-up by physician/hospital	11	5.8	10	13.7	8	28.6	29	10.0
Long waiting hours at screening site	8	4.2	4	5.5	0	0	12	4.1
Busy in job	42	22.1	18	24.7	2	7.1	62	21.3
Immobilised by disease	16	8.4	6	8.2	6	21.4	28	9.6
Moved, or long time absent	59	31.1	10	13.7	6	21.4	75	25.8
Forgot/no reason/other	36	18.9	21	28.8	3	10.7	60	20.6
Unnecessary/unwilling	18	9.5	4	5.5	3	10.7	25	8.6
Total	190	100.0	73	100.0	28	99.9	291	100.0

scientific papers, 15 Ph.D. theses and a large number of reports created for regional and state authorities for use in preventive health care and health planning.

A fundamental premise for population studies is high confidence and legitimacy in the study population. The strategy to achieve and withhold this confidence in the population of Nord-Trøndelag has been successful, and resulted in high participation rates and even enthusiastic public and political support for HUNT and the HUNT Research Center, which is located in the middle of the study population. The decline in participation from HUNT 1 to HUNT 2 has raised some concern, as this probably is part of an international trend. In HUNT 2 the decline was most prominent under the age of 60 (Figure 3). Today, more people are seeing their physician and are having regular medical check-ups[18], and many young people are busy with job and children. The logistics in the screening itself might also play an important role. Due to a larger screening program the time spent at the screening site was longer at HUNT 2 compared to HUNT 1, making it difficult for people who needed to leave their job. In addition, distances to the screening site increased, as the team had to operate from only one location within each municipality (Se Appendix). Due to the larger screening program people also had an appointment hour instead of an appointment day at HUNT 1, making participation less convenient. In addition, there seemed to be less interest in public health and preventive medicine than some decades earlier. Even primary physicians seemed less engaged in public health issues than they used to be in the 1980s. All these factors might have contributed in making health surveys less attractive. Future surveys must take into account that modern people are busy and expect a smooth and efficient screening system with no waste of time. However, the non-participation study performed after HUNT 2 gave no evidence that people had negative attitudes to the study itself (Table 6).

A main challenge for the HUNT study has been to initiate research using the biobank, especially functional genomics. Initiatives are taken in collaboration with the FUGE project[103]. As the genomic era develops the re-consent project including the information campaign about functional genomics was an important experience. This result also confirmed our impression that the HUNT study has a high confidence and legitimacy, and the population is in general strongly supportive[98]. Another main challenge is the planning and realization of a third HUNT study (HUNT 3). The confidence and supportive attitude from the population should be a good platform for conducting future population studies with high participation rates in the Nord-Trøndelag County.

APPENDIX: ORGANIZATION AND FUNDING OF HUNT 2

The HUNT database is a result of tight collaboration and joint actions between local, regional, national and international partners through the last 20 years. The study has involved a large number of individuals and organizations.

The National Health Screening Service (SHUS)(*) was responsible for the technical performance of the basic screening among all persons 20 years and older, and for setting up one field team of five persons to participate in the collection of data in Nord-Trøndelag during the two year period. This “mobile team” had a fully equipped trailer with office facilities where the health screening took place. The mobile team was responsible for the screening in the 19 smaller municipalities. They needed, however, adjacent localities for some of the examinations, for instance the audiometry. In the “stationary team” SHUS provided the leading nurse and the operator for the various technical devices during the first five "run in" months, and also provided technical equipment. SHUS was responsible for the invitations and provided the various sub-studies with necessary addresses, name tags etc. that were used for invitations, reminders etc. Questionnaire 1 and the results of the blood tests were sent to SHUS where the primary statistical analyses were done and appropriate response letters were sent back to the participants. SHUS had the license for storing and linkage of the main files including the national identification number, and prepared research files for statistical analyses.

The National Institute of Public Health (*), Oslo, and Community Medicine Research Center (*), Verdal. The Community Medicine Research Center in Verdal administered the other field team (“the stationary team”), consisting of 12 persons responsible for the screening in the five larger municipalities. Various sub-studies were also organized by the Community Medicine Research Center. The sub-studies included diabetes mellitus, high blood pressure and coronary heart disease, in addition to the Young-HUNT study[41,104-106], and the Bronchial Obstruction in Nord-Trøndelag (BONT) study[43,44]. The Young-HUNT, i.e. the screening of all persons between 13 and 19 years old, was fully organized by the Research Center in Verdal through an established group consisting of the primary investigator, secretary and field workers; seven persons in total. The BONT study, which also included bone mass measurements, was run from the Research Center. The executive group consisted of a primary investigator and field workers, totally six positions covered by 22 different persons. Altogether, the Research Center in Verdal administered approximately 50 collaborators, and was responsible for administrative and practical work ranging from contract negotiations concerning locations and recruiting personnel, to sending out reminders to participants. The National Institute of Public Health, Oslo, Section for Epidemiology, was responsible for the

hearing study in HUNT[80-84]. Their team consisted of a coordinating audiometrist and four field workers (audiometrists and assistants).

Innherred Hospital, Levanger(*). The Central Laboratory at Innherred Hospital, Levanger, was responsible for all routine blood and urine analyses that were part of HUNT. Some specific samples were sent from the laboratory to Aker hospital in Oslo for thyroid status analyses, and to the National Institute of Public Health for immune status. Results from these analyses were transferred to the HUNT database at SHUS, Oslo, to form the basis for response letters to the participants. Innherred Hospital also provided facilities for the biobank, i.e. localities for freezers containing whole blood and serum samples at minus 70°C.

The Faculty of Medicine, NTNU. Scientists at the Faculty of Medicine had a central position in projects on osteoporosis, urinary problems in men, baldness in men, vision impairment, sense of humor and migraine/headache.

NOVA (Norwegian Social Research). A separate sub-study was aimed at residents in nursery homes and other institutions for elderly, organized by NOVA. The field team consisted of two nurses who visited institutions for interviews and clinical measurements.

In addition to the institutions mentioned above, general practitioners and district nurses in the whole county, the 24 municipalities and county authorities, a number of private regional and national organizations, and a number of other national institutions and universities, The Norwegian Research Council, and The Ministry of Health, actively supported the study. HUNT is also part of the nation-wide CONOR (Cohort Norway) collaboration, constituting a network of national health databases and biobanks, in which HUNT is the largest single unit[101].

FUNDING

HUNT 2 was funded by joint efforts of a large number of partners. Main contributions came from The Ministry of Health, through The National Institute of Public Health and The National Health Screening Service (SHUS). The Nord-Trøndelag County Council, The Norwegian University of Science and Technology and The Norwegian Research Council also provided essential funding. Sub-studies were supported by The Norwegian Research Council or a number of private organizations, like The Diabetes Association, The Norwegian Association of Asthma and Allergy and The Norwegian Women's Public Health Association (Norske Kvinners Sanitetsforening). The Hearing Study was funded by the National Institutes of Health (NIH), USA, and some sub-studies were supported by pharmaceutical industry: AstraZeneca (the BONT Study), GlaxoSmithKline (the diabetes study) and MSD (the prostate study). In total, the core study had a cost of about NOK 21 mill., and the sub-studies additionally NOK 10 mill.; infrastructure resources made available by the institutions not included.

(*) Reorganizations have taken place in several collaborating institutions after the field work was finished: The Community Medicine Research Center (now HUNT Research Center, Verdal) was previously part of The National Institute of Public Health. From 2001 the center is part of the Department of Community Medicine and General Practice, Faculty of Medicine, Norwegian University of Science and Technology (NTNU). Innherred Hopsital, Levanger, and Namdal Hospitals, Namsos, were previously owned by The Nord-Trøndelag County Council. From 2001 they are owned by the Ministry of Health and administered by The Regional Hospital Administration (Helse Midt-Norge) and are named Levanger Hospital and Namsos Hospital. From 2002 The National Institute of Public Health (Folkehelsa) and The National Health Screening Service (SHUS) are merged into the Norwegian Institute of Public Health, Oslo.

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