UiT The Arctic University of Norway

Faculty of Humanities, Social Sciences and Education
Department of Social Science

Up the downstream

Contributing mechanisms to the persistence of health inequalities in Norway

Sigbjørn Svalestuen

A dissertation for the degree of Philosophiae Doctor – March 2023

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This dissertation is dedicated to Associate Professor
Marcus Buck (1962 - 2023).

Acknowledgements

There are a number of people that have been instrumental in this dissertation reaching its conclusion. A special thank you to my supervisors Professor Tor Midtbø, Associate Professor Marcus Buck, and Professor Anne Elise Eggen for valuable input and guidance throughout these past four years. They have provided valuable and timely responses to any draft I have ever sent them. I further appreciate their willingness to discuss whatever I needed to discuss that day. My co-authors deserve a special thank you for contributing their much needed expertise throughout. My colleagues at the *Department of Social Science*, in the *High North Population Studies* research project, and the *Governance and Democracy* research group deserve special thanks for providing community and a solid research environment. I look forward to our continued collaboration down the line. Principal Investigator Professor Hilde L. Sommerseth deserves particular recognition for running a tight ship throughout.

I wish to extend my gratitude towards the Centre for Global Health Inequalities Research (CHAIN, NTNU) in Trondheim for providing office space and networking opportunities whenever I came to visit in 2019 and 2020. I further extend my gratitude towards the Institute of Health Metrics and Evaluation (IHME) at the University of Washington for accommodating a research stay fall 2022. I look forward to following the work of both CHAIN and the IHME in the future.

To my friends, family, and partner: thank you for all the support and continuous encouragement.

Abstract

This dissertation aims to expand on the current empirical knowledge of and theoretical mechanisms in the social determinants of health. Specifically, it investigates the mechanisms by which social root causes generate health outcomes in order to assess within-state health inequalities in high-income countries such as Norway. It emphasizes the effect of material and immaterial resources that result from positioning in a social hierarchy, typically reflected by income, education, and occupational class.

Article I explores the income inequality–health hypothesis and its relation to income, psychosocial stress, and self-rated health. Using multilevel moderated mediation analysis, I show that while the income–health relationship is indeed mediated by psychosocial stress in all countries under study, this effect is not moderated by income inequality. I conclude that material effects of income at the individual level remain an important explanation of the income–health gradient.

Article II investigates the extent to which socioeconomic determinants of health (education, income, and occupational class) contribute to predicting non-communicable disease (NCD) prevalences. Further, it aims to non-parametrically assess how these health gradients look from the perspective of a random forest algorithm. We conclude that *prediction* is an important tool for scientific inquiry into health inequalities and potentially a useful tool for public health guidance, as it allows unpacking complex functional relationships between NCD outcomes and social determinants of health.

Article III shifts attention towards subnational trends in early life health care input. The article investigates to what extent local patterns of antibacterial dispensing rate trends in young children are associated with the proportion of the local population having achieved high levels of education. Results indicate that the ability to reduce dispensing rates over time at the municipal level is associated with mean population levels of higher education. Optimizing prescribing practices may require area-level strategies, and attention should be given toward sociodemographic challenges at the local level.

Overall, I show that material and immaterial social resources, and their value towards a market that reward said resources, remain important explanans of the persistence of health inequalities even in comparatively high-income and egalitarian countries such as Norway. I argue that the fundamental structures of social inequality must be considered when explaining the persistence of health inequalities, and that policies in pursuit of *population health* are limited in their ability to address this fundamental structure. Empirically, I show that material and immaterial advantage lead to better health outcomes both at the individual and population levels. Methodologically, I show that while methods of association are limited in their ability to estimate causal *effects*, important patterns of health inequalities can still be explored using novel statistical analyses on observational data.

List of Articles

1. Sigbjørn Svalestuen (2022). "Is the mediating effect of psychosocial stress on the income–health relationship moderated by income inequality?" *SSM - Population Health* 20, p. 101302. ISSN: 2352-8273. DOI: https://doi.org/10.1016/j.ssmph.2022.101302. URL: https://www.sciencedirect.com/science/article/pii/S2352827322002816

2. Sigbjørn Svalestuen et al. (2023). "The predictive importance of education, income, and occupation on non-communicable disease outcomes: results from the Tromsø Study." *Under review*

3. Sigbjørn Svalestuen et al. (2022). "Association of area-level education with the regional growth trajectories of rates of antibacterial dispensing to patients under 3 years in Norway: a longitudinal retrospective study." *BMJ Open* 12.9. ISSN: 2044-6055. DOI: 10.1136/bmjopen-2021-058491. eprint: https://bmjopen.bmj.com/content/12/9/e058491.full.pdf. URL: https://bmjopen.bmj.com/content/12/9/e058491

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Part I

Extended introduction

1

Introduction

The aim of this dissertation is to expand on current empirical knowledge of and theoretical mechanisms in the social determinants of health. Specifically, it investigates the mechanisms by which social root causes generate health outcomes in order to assess within-state health inequalities in high-income countries such as Norway. This extended introduction serves to clarify fundamental assumptions applied in the three empirical articles included in the dissertation and discusses mechanisms contributing to the persistence of health inequalities.

The empirical existence of health inequalities is well established. These include differences in a range of health outcomes based on education (Rydland, Solheim, and Eikemo 2020), income (Kinge et al. 2019), work status and labor market participation (García-Gómez 2011; Vinjerui et al. 2020), gender (Bambra et al. 2009), and ethnicity (Krieger 2005). Health inequalities also exist in the aggregate: between subnational units, such as neighborhoods and cities, municipalities, and regions (Bambra 2016), and between states (Mackenbach et al. 2018). Mortality, morbidity, self-rated health, mental health, risk factors, and health care access and input are all examples of the multitude of ways that researchers have studied and identified social differences in health outcomes. The persistence of health inequalities in modern welfare states is often presented as a "paradox" (Mackenbach 2012) or a "puzzle" (Bambra 2011a). First, it is paradoxical that the most egalitarian/generous welfare states do not show the smallest health inequalities. Second, health inequalities have on some measures widened during and after welfare state build-up. This puzzle

suggests significant gaps in our knowledge of the underlying mechanisms that generate health inequalities within countries.

Sen, Anand, and Peter (2004) argue that any discussion of *social* equity and justice must include illness and health and that health equity must be understood within a larger issue of fairness and justice in social arrangements, such as economic allocations and the role of health in human life and freedom. Rawls (1991) argues that basic political institutions should realize the values of liberty and equality and that all persons have an equal right to a fully adequate scheme of liberty and equality (Rawls 1971). While Rawls considers health a natural good and thus rules it out as a basic freedom, his first principle of justice has been used to define health inequality, as the health of individuals depends on both individual health endowments and how health endowments are transformed into health through access to health resources. Within this framework, two people with similar health endowments but from different socioeconomic backgrounds achieving differential health status is therefore considered unjust. In other words, while health itself is not considered a basic freedom, *health access* is (Bommier and Stecklov 2002). A further justification can be made by extending the issue of politics as redistribution, the "who gets what, when, and how" (Mahler and Jesuit 2006; Finseraas 2009). While health cannot be redistributed directly from those well-off to the most disadvantaged members of society, policies may be enacted to ensure that health access and life chances are equally distributed across social arrangements and material circumstances.

The empirical contributions in this dissertation are limited in scope to differences in health outcomes between social groups defined by education, income, and occupation. Collectively, these indicators typically refer to socioeconomic positioning, socioeconomic status, or social class as *social stratification*. Thus, the dissertation primarily engages with social root causes and distal causes of health outcomes rather than proximate causes of health, such as risk factors. In some cases, the articles discuss the ways in which the causal links between health and social groups depend on these factors, but the emphasis remains on the social determinants of health and illness resulting from class-based resource availability and life chances.

The dissertation argues that both material and immaterial resources are likely mechanisms in the persistence of health inequality in modern, high-income welfare states such as Norway (a summary of the articles is available in Table 1.1). First, even in egalitarian welfare states such as Norway, material resources remain unequally distributed. The income–health gradient is appreciable, even if overall material well-being has improved over time. Differences in material resources are a likely root cause of health inequalities generated by income. I show that psychosocial stress is a likely mediator of the income–health gra-

dient; in fact, psychosocial mediation is ubiquitous in all countries under study, regardless of the level of income inequality. This supports the argument that the root generating mechanism of the income–health gradient is less about "inferiority complexes" in a social status hierarchy, and more about the material benefits and stress responses that income generate. Second, I show empirically that both immaterial and material resources are *predictive* of non-communicable disease (NCD) outcomes. However, the empirical social gradients in NCD prevalence depend on the NCD under study and the choice of socioeconomic indicator. I further show that the extent to which socioeconomic indicators contribute to predictive accuracy varies between socioeconomic indicator and measured health outcome. Third, immaterial and flexible resources are unequally distributed geographically within Norway and are correlated with early life health inputs that may affect future health conditions. Geographical inequalities likely result from an interplay between contextual and compositional population effects. Reducing geographical health inequalities requires strong coordinating mechanisms between sectors to fully implement national policy via intersectoral cooperation.

In addition to these mechanisms, the extended introduction describes important trends in modern European public health governance. It argues that while Norway has been considered somewhat unique in Europe as the only country having a national health inequalities reduction program that highlights the effects of income inequality (redistribution) on health inequality, its institutional ability to address the issue in practical terms remains limited with regard to evidence-based policymaking. I argue that acknowledging the social determinants of health and applying some methodological pluralism in research are important for future evidence-based policymaking in health. Extending the findings in Article II suggests that one way of exploring health inequality dynamics in data-rich contexts such as Norway is by leveraging predictive modeling for generating out-of-sample predictive metrics, as doing so expands the methodological toolbox for evaluating empirical findings.

The remainder of this extended introduction is structured as follows: Chapter 2 presents and categorizes major schools of thought in health inequality research and defines the overall framework and theoretical position. Chapter 3 summarizes major trends in public health governance and policy, situates these trends within the Norwegian context, and discusses the meritocratic reward system that defines the value of social resources. Chapter 4 describes the conceptual logic in measurement and discusses some overarching methodological considerations inherent to all work in the dissertation. Chapter 5 presents the individual articles and how they contribute to the literature and the overall thesis statement. Chapter 6 concludes, discusses limitations, and sketches out some potential avenues for future health inequality research.

Table 1.1: Short summary of articles

#	Title	Summary
1	*Is the mediating effect of psychosocial stress on the income–health relationship moderated by income inequality?*	The income inequality–health hypothesis argues that income inequality is causally related to health through psychosocial mediation. Using multilevel moderated mediation analysis, I show that while the income–health relationship is mediated by psychosocial stress in all countries under study, this effect is not significantly moderated by income inequality. I conclude that material effects of income at the individual level remain an important explanation of the income–health gradient.
2	*The predictive importance of education, income, and occupation on non-communicable disease outcomes: results from the Tromsø Study*	We apply an algorithmic approach to a) investigate to what extent social determinants of health such as individual education, income, and work status contribute to predicting non-communicable disease prevalence and b) assess non-parametrically, what these gradients look like from the perspective of a random forest algorithm. We conclude that prediction is an important tool in scientific inquiry and potentially useful for public health guidance as it allows unpacking complex functional relationships between NCD outcomes and social determinants of health.
3	*Association of area-level education with the regional growth trajectories of rates of antibacterial dispensing to patients under 3 years in Norway: a longitudinal retrospective study*	Periodic prevalence and patterns of antibiotic use vary between countries and between socioeconomic and demographic groups within countries, and studies have also shown temporal variations in the dispensing of antibacterials for systemic use. Our analysis shows that the ability to reduce dispensing rates over time at the municipality level is associated with mean population levels of higher education. Local needs and potential root causes of health outcomes should be considered in antimicrobial stewardship to optimize prescription patterns, and attention should be paid to social demographics to further reduce dispensing rates in accordance with political ambitions.

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2

Literature and framework

In this chapter, I detail the scientific background that motivated and inspired the works in this dissertation. First, I trace and label some of the most influential schools of thought incorporated into health inequality research. Second, I apply the previous work and generate a heuristic model that accommodates the stages of the social determinants of health framework. Third, I show how this framework applies to the empirical work in the research articles.

2.1 Major schools of health inequality research

I apply two distinct labels when categorizing schools on health inequalities based on their emphasis: biological and individual models that tend to deal with proximate causal mechanisms that relate individual health outcomes to social positioning through behaviors and risk-factor analysis, and sociopolitical and structural models that emphasize distal causes and political contexts that socially generate and reproduce health inequalities. Central to the structural theoretical approach in the study of health inequalities is that differences in the socioeconomic circumstances of social groups, such as income and wealth inequalities, power relations, environmental context, and access to services, cause differences in health outcomes (McCartney, Collins, and Mackenzie 2013). I further distinguish these foundational assumptions by their level of integration, that is, the extent to which these two schools of thought are integrated into the framework.

A second dimension may be applied to further differentiate between these schools of thought: their assumptions regarding cause and effect. Three such assumptions may be applied: social causation (the assumption that health outcomes are a result of individual and social characteristics), health selection (the assumption that poor health leads to negative socioeconomic outcomes and lower positions in a given social hierarchy), and bidirectionality (the flow of causation shifts back and forth over the life course). The distinction between social causation and health selection, and their synthesis, is further necessary to show that the social determinants of health umbrella perspective is one of several ways of thinking about the causal processes that generate social inequalities in health. These sets of labels serve as ideal types reflecting foundational ideas in thinking about health and population health processes in the many disciplines involved. They thus function to define and situate the works in this dissertation and provide some structure to presenting the works upon which this dissertation is built.

2.1.1 Early thinking and the epidemiological transition

Early thinking developed in the 1800s took a markedly sociopolitical and structural approach to population health and health inequalities. Engels (1845) described the poor living and work conditions of the proletariat as an inevitable result of the capitalist mode of production (Rydland 2020, p. 21). In Norway, Sundt (1855) described how differences in mortality could be explained by differences in living conditions and material standards leading to increased prevalence of transmissible disease. Rudolph Virchow famously espoused that "medicine is a social science, and politics nothing but medicine on a larger scale" and argued that the only way to improve health and reduce disease is by changing society through political action; the former of these two statements is echoed in modern times with the popularity of the Health in All Policies (HiAP) agenda (Mackenbach 2009). Later milestone investigations, particularly in the UK with The Black Report (Department of Health and Social Security 1980), The Acheson Report (Department of Health and Social Care 1998), and research based on the Whitehall studies (Marmot et al. 1978; Marmot, Shipley, and Rose 1984; Marmot et al. 1991) further established social gradients in health as a major public health issue despite the development of the welfare state.

The 20th century also saw what was later termed an epidemiological transition: a shift in the main burden of disease and causes of death from infectious diseases to degenerative cardiovascular diseases and cancers, largely due to increases in living standards and reductions in material deprivation (Wilkinson 1994). This shift is likely to have had some impact on the popularity of biological and individual explanations of health outcomes and health inequalities. Following

this shift, a period of complex models of "host–agent–environment," along with other medical and biological developments, extended epidemiological attempts to link the micromechanisms of disease causation to population health patterns stressing the role of biological evolution and adaptation in the epidemiology of infectious disease. Notably, debates on causation were less about traditional theoretical development and more directed toward the nature and validity of causal inference (Krieger 1994). Developments from these strands resulted in an emphasis on individually based risk factors and behaviors that served as proximate causes of disease, such as diet and exercise regimen (Link and Phelan 1995). Psychosocial theory represents an extension of theoretical frameworks of disease causation to disease distribution. Its starting point is human susceptibility to disease and why some subsets of people seem more susceptible to ubiquitous pathogenic agents; Cassel (1976) argued that in prosperous nations, the moderators of "host resistance" were likely to be found in the social environment, comprised of psychosocial factors generated by human interaction including dominance hierarchies, social disorganization, social change, and marginal status in society. Social support acts as protective of these detrimental factors (Krieger 2001). The foundations set by psychosocial theory resulted in several extensions of the framework, with some variations emphasizing neuroendocrine pathways and the health effects of the allostatic load and others expanding the psychosocial environment to include the health effects of social capital and social cohesion, which strengthen the bonds of civil society and social affiliation (Kawachi, Berkman, et al. 2000; Baum 1999). However, the essence of psychosocial frameworks directs attention toward the biological responses to human interactions and the need for psychosocial resources and less toward asking who and what generates psychosocial stress and how their distribution is shaped by social, political, and economic policies (Krieger 2001).

2.1.2 Social production of disease

The social production of disease, fundamental causes, and the political economy of health perspectives criticize the dominant approach of lifestyle health behavior and psychosocial theories based in this line of reasoning. These theories include the identification of economic and political determinants of health and disease and address issues with individual agency in choosing to live healthy lifestyles and individual ability to cope with stress (Krieger 2001).

Fundamental cause theory (Link and Phelan 1995; Phelan, Link, and Tehranifar 2010) directly addressed the proliferation of individual, lifestyle-based theories by shifting the focus from the risk factors of specific diseases to the social causes of these risk factors (referred to as fundamental causes or "the causes of the causes"). Proponents argued for contextualizing individual-based risk factors

by examining what puts people at risk in the first place. In this perspective, socioeconomic status is important because it reflects the individual's control over flexible resources, such as knowledge, money, power, prestige, and beneficial social connections, that could be used in one way or another to influence health outcomes. Fundamental cause theory shifted the focus from disease-specific etiology, as they conceptualized a fundamental cause as an influence on multiple disease and ill-health outcomes through multiple risk factors and argued that the association between a fundamental cause and health is reproduced over time by intervening mechanisms. Intervening mechanisms are here defined as the tendency for new mechanisms to arise following, for instance, the development of new knowledge or medical technology that, to a greater extent, benefits socioeconomically advantaged groups as they are considered better equipped with the resources to take advantage of this new development (Phelan, Link, and Tehranifar 2010), akin to the diffusion of innovations theory proposed by Rogers (1962).

While fundamental cause theory (Link and Phelan 1995) assumes that individual flexible resources can be spent on health seeking, a limitation of the framework arises from countervailing mechanisms (Lutfey and Freese 2005) in which resources are spent on competing goals such as power or status attainment, which can sometimes function as more powerful motivators than health. Fundamental cause theory requires that the effects of these countervailing mechanisms are cumulatively smaller than the effects of mechanisms producing the fundamental relationship (Phelan, Link, and Tehranifar 2010; Lutfey and Freese 2005). Regardless, fundamental cause theory implies that health inequalities can be reduced by reducing inequalities in flexible resources, at both the individual and the collective level, and by prioritizing interventions that minimize the relevance of said resources (Phelan, Link, and Tehranifar 2010)

The political economy of health tradition (in a broad sense) details the political conditions of the social production of disease. Doyal and Pennell (1979) argued that the orthodoxies present in the medical and scientific model of health and the individual origin of disease obscures the social and economic causes of ill-health. The predominant model at the time viewed ill-health as a result of the individual "way of life," suggesting that ill-health results from individual moral failings. Their criticisms did not reject the physical and chemical laws that govern disease etiology; rather, they argued that these processes must be seen to "operate within a social and economic context which is constantly changing." The political economy of health discusses aspects of the polity, political priorities and policies, and their consequences on health – for instance, how the transition to capitalism may have expanded relative health inequities based on education, the effects of neo-liberal or market-oriented reform on relative and absolute health inequities, the type of welfare state regime and its

relationship to the magnitude of health inequities, and the effects of democratic marginalization over long periods (Beckfield and Krieger 2009).

Beckfield et al. (2015) and Beckfield (2018) developed an institutional framework in order to integrate insights from social stratification and health inequalities research. They have identified four institutional processes linking welfare states to social determinants of health and health inequalities: the extent to which inequality in goods such as income, wealth, or flexible resources (Link and Phelan 1995) are shifted in favor of marginalized groups (redistribution); imposed restrictions (through, for instance, corporatist bargaining) on how much or how little individuals and groups may control of these resources (compression); the extent to which the social determinants of health are mediated by other variables (mediation); and the extent of overlap between institutions that simultaneously distribute resources and directly impact health and health care availability (imbrication). Esping-Andersen (1990) developed a typology of welfare state regimes that details the extent to which reliance upon the market for individual welfare (decommodification), the role of the state in maintaining or breaking down structural inequalities between groups (social stratification), and the relative role of the state, market, and voluntary sectors in welfare provision (private–public mix), which, along with revisions of this typology, has been used in social epidemiology – for instance, in analyses of socioeconomic class polarities created by work status and labor market exclusion (Bambra 2007).

Navarro et al. (2006) show that political parties with egalitarian ideologies are more likely to implement redistributive policies and that welfare and labor market policies aimed at reducing social inequalities have a salutary effect on infant mortality and life expectancy at birth. The strongest relations between politics, policies, and health outcomes appear when power resources such as public social transfers and public expenditure, as well as the participation of men and women in the labor force can be leveraged over long, cumulative years of government. Navarro and Shi (2001) further conclude that the forces of political parties stem from how they represent class and other social interests in redistribution and that labor movements and social democratic parties have been the most committed to redistributive policies. Consequently, countries with weaker labor movements and stronger capitalist classes also have weaker commitments to redistributive policies. Christian democratic parties have made compromises resulting in redistributive policies weaker than those of social democratic parties but stronger than those of their liberal counterparts. Muntaner et al. (2011) find that leftist and egalitarian political traditions show the most consistently salutary effect on population health and that these have large health impacts in advanced and liberal democracies. They further find that absolute health outcomes tend to fare better under social democratic welfare regimes yet are consistent with other welfare regimes regarding health

inequalities and that dependency on trade, foreign investment, and national debt (here understood as globalization) are negatively associated with population health. They conclude that the strongest and most consistent associations with improved population health are advanced levels of democracy and egalitarian political traditions and that the effects of the welfare state as such are inconsistent. Here, the neo-Marxist model of class division and conflict-based theories direct the analysis, where class relations are based upon the control of production and labor power that, in turn, produce managerial relations. Muntaner et al. (2015) argues that neo-Marxist class analysis can break the chain between health inequality research and mysticism in the policymaking process.

The political economy of health literature has raised numerous criticisms of the mainstream focus on policy implications in health inequality research, suggesting either that most calls to political action and calls for the political will to reduce health inequality either show a weak understanding of politics (Greer et al. 2022) or that their policy implications (while appearing politically neutral) merely function to serve incrementalist change or apolitical and technical changes that ultimately justify the current system and reproduce the status quo (Muntaner et al. 2015). Further, policymakers' rationality may be bounded by limited time and information. They may therefore be more likely to build on existing policies and programs rather than attempt system-wide reforms, reinforced by the relative probability of agreement with other political parties when only modest adjustments to the status quo are made (Oliver 2006). In short, incrementalist change increases the probability of policy being successfully enacted, and less political capital is spent in the process.

2.1.3 Health selection

Contrary to all the preceding frameworks, theories of health selection reverse the flow of causation. These theories aim to investigate to what extent ill-health at any stage of the life course may generate a reduction in social class or socioeconomic status. For instance, García-Gómez (2011) argues that there is a significant causal effect from health on the probability of employment; individuals experiencing a health shock are more likely to leave employment and to transition into disability. These effects seem, in turn, at least partially explained by country-level differences in social security arrangements. In terms of income, O'Donnell, Van Doorslaer, and Van Ourti (2015) conclude that ill-health causes income inequality and that income inequality does not damage health directly. They cite that ill-health is indeed a major cause of labor-force withdrawal in middle age, with the effect perhaps moderated by disability insurance compressing income distribution and the balancing of income replacement and work incentives. Further, poor health may make it more difficult to find a part-

ner in the marriage market, directly impacting household income specifically. Young adults experiencing frailty or poor health face a narrower choice of jobs upon entry into the labor market, due to impediments in productivity, carry-over effects from impeded productivity during education, and discriminatory practices in the labor market. In addition, the less educated may be less likely to adapt due to a lack of general human capital, making it more difficult to move from manual to nonmanual labor at the onset of illness. These groups are more likely to exit the labor force when affected by a disability.

Ill-health or exposure in neo- and post-natal life are expected to show economic consequences later on; for instance, low birth weight has been shown to increase welfare dependency (Currie et al. 2010). Ill-health may reduce cognitive functioning, reduce the effect of schooling, and impact opportunities to acquire education, thus impacting human capital accumulation and earnings potential (O'Donnell, Van Doorslaer, and Van Ourti 2015). A prolific area of research, the "fetal origins hypothesis," argues that adverse experiences in utero, such as nutrition deprivation exposure, may impair cognitive functioning and have an effect on metabolic characteristics leading to future disease, suggesting that the most effective target for policy intervention is indeed the mother during gestation (Almond and Currie 2011). The fetal origins hypothesis has since expanded to include investigations into the early childhood environment and intergenerational effects. Research in this area has demonstrated that even relatively mild and brief shocks during early childhood may have lasting measurable impacts on child outcomes, for instance by linking test scores to birth weight, by showing how increases in annual income or transfers impact the incidence of low birth weight, and by investigating (maternal) exposure to influenza and pollution in the early life stages. Though there is considerable heterogeneity in the effects of a given shock, more disadvantaged people often seem to suffer greater harms (Almond, Currie, and Duque 2018).

2.1.4 Integrated approaches

More complicated models of the relationship between health and social positioning reject the somewhat simple causal claims present in other models of health inequality. These models are considered bidirectional. The life course perspective takes an explicit approach to the issue of cause and effect by allowing bidirectional causal effects to occur between different stages over the life course. As such, life course theory explicitly ties these causal effects to age (Wadsworth 1997; Hoffmann, Kröger, and Pakpahan 2018; Rehnberg et al. 2021). Expanding from the assumption that biological programming and the social beginnings of life may carry forward into adult ill-health and socioeconomic positioning, life course approaches assume a constant interplay between social factors and ill-health, creating a dynamic model of health outcomes. For in-

stance, while occupation, education, and income may impact health outcomes, ill-health experiences at an earlier stage of the life course may also impact social mobility and access to certain occupations and incomes at a later stage of the life course. Inherent to life course models is that at least some of the tension between health selection and social causation explanations of health inequality disappears (Dahl, Bergsli, and Van der Wel 2014, p. 43).

Hoffmann, Kröger, and Pakpahan (2018) find that both socioeconomic status and health rely heavily on their prior status and that during transition from childhood to working ages, social causation and health selection processes are equally weak but that the effects of social causation increase in the transition from working ages to old age. Similarly, Rehnberg et al. (2021) find that changes in the causal relationship are more likely to be identified in ages where transitions between age-stratified institutions are common. For instance, in ages where individuals are likely to transition into the labor market, associations from health to income and education are strong. Around retirement age, the association from income to health strengthens. However, in middle age groups, associations are of similar strength in both directions.

A different kind of integrated model can be found in ecosocial frameworks (Krieger 2001) and in complex system dynamics approaches (Galea, Riddle, and Kaplan 2010; Fink, Keyes, and Cerdá 2016). Ecosocial perspectives represent multilevel dynamic perspectives on the determinants of health that situate human beings as one of many co-habiting and evolving on a dynamically evolving planet. Ecosocial frameworks emphasize the evolving interaction between living organisms, matter, and energy, over time and space, from the cell to the ecosystem – its main objective thus being to integrate the social world with its ecological surroundings (Krieger 2001). An example of this kind of thinking is the analysis of health inequalities between races and ethnicities as well as the attempt at investigating whether health inequalities arise from the literal embodiment of unjust race relations (Krieger 2005).

Complex systems approaches problematize the distinction between independent and dependent variables and the modeling of exposures and outcomes in the study of health and population health. Complex systems are systems characterized by feedbacks, interrelations among agents, and discontinuous nonlinear relations. Complex systems methodology emphasizes algorithmic modeling to model interactions between individuals, groups, and their properties within and between levels of influence, simulating results from, for instance, interventions (Galea, Riddle, and Kaplan 2010). In research on the social determinants of health, systems sciences combine the focus on social determinants with a conceptual framework in which genetics, biology, behavior, psychology, society, and environment interact. Macrosocial factors have been given particular attention in systems science approaches to health inequality,

specifically feedback between micro- and macro-level processes and in modeling how health inequalities shape population health overall (Fink, Keyes, and Cerdá 2016).

2.2 Framework and positioning

The preceding sections summarized and categorized the foundations of the literature on social inequalities in health. This section aims to present an overarching framework for the social causation of health and health inequalities. As such, this dissertation, with its empirical constituents, is firmly positioned within the social determinants of health perspective. This is, in large part, to limit the overall scope and define which concepts are to be placed at which side of the equation. Figure 2.1 presents an abstracted causal model of the social determinants of health in individuals. The bottom diamond contains all mechanisms pertaining to the individual level of analysis, and the upper part of the model contains all mechanisms pertaining to the aggregate level of analysis. All variables are contained in the graph's vertices, while all pathways are captured by edges.

Its most basic component is the direct causal relationship between *individual resources* captured by x and *health outcomes* as captured by y. These individual resources are restricted to resources based on education, income, work, and employment. Health outcomes are broadly defined and include disease outcomes such as NCD prevalence, self-rated health, and health care input. z captures all individual-level mediators between individual resources and health outcomes, such as individual behaviors, psychosocial stress, and health-related decision-making. Vertex c captures all individual-level confounders that affect both individual resources and health outcomes. While there are many possible confounders, genetic endowment serves as a useful example. Thus, there are three unique individual-level pathways between resources and health outcomes: direct (λ_{xy}), indirect ($\lambda_{xz} + \lambda_{zy}$), and confounded ($\lambda_{cx} + \lambda_{cy} + \lambda_{xy}$).

Moving to the upper part of the model, w contains all aspects of a polity that may influence individual resources, mediating mechanisms, and health outcomes such as welfare provision, health care provision, public health policies, redistributive policies and representation, and infrastructure. This section is thus best described as "the causes of the causes of the causes," i. e., the political economy of health. The direct pathway (γ_{wy}) between this level and health outcomes include infrastructure such as sanitation and access to clean water. The indirect pathways ($\gamma_{wz}|\gamma_{wz}$) include for example the income inequality hypothesis or the effect of redistributive policies on material well-being. Most important, the w vertex includes the market that *defines the*

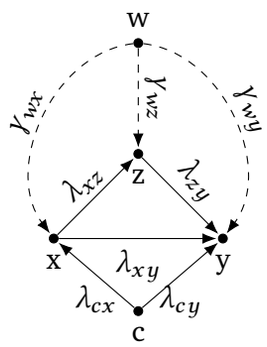

Figure 2.1: A heuristic model of the social determinants of health perspective.

importance of income, education, and work status as individual resources that may be leveraged for health outcomes.

The model thus accommodates individual material and immaterial resource perspectives, proximal causes of disease perspectives, and politico-economic perspectives on health. The polity is conceptualized as the ultimate ancestral cause, as it defines both the market value of material and immaterial individual resources and the consequences of resource deprivation.

2.3 Framework: in the articles

The papers all emphasize the effect of resources in a variety of ways. They engage with proximal (mediating) mechanisms and pathways between resources and health outcomes and with ancestral (redistributive, governance, public policy) causes of individual resource effects.

Of the three papers in the dissertation, Article I arguably comes closest to achieving the preceding framework's ideal, methodologically speaking, as it includes all steps of the causal chain described above in a statistical model on observational data. It empirically investigates all vertices in the framework but limits the empirical investigation to specific pathways.[1]

Article II addresses only vertices x, y, and c (resources, health outcomes, confounders) in the empirical investigation. However, in doing so it addresses how methods of scientific evaluation inform public policy and evidence-based decision-making. It further draws on mechanisms explaining the gradients that include mediators and behaviors (z). As such, it interacts with w and z in its

1. λ_{xy}, λ_{xz}, λ_{zy}, γ_{wz}, γ_{wx}

interpretation of the gradients and the potential use of predictive modeling in evidence-based policymaking.

Article III similarly addresses only resources, health outcomes, and confounders in the empirical investigation. Empirically, it aggregates these concepts from the individual to the subnational level of analysis. In this sense, vertices x, y, and c now contain subpopulation resources while conditions remain defined by the national level (w). The discussion primarily revolves around how compositional differences between local populations may be translated from individual-level mechanisms, but it goes somewhat further and implies that geographical areas are themselves socioeconomically disadvantaged and that differences in community resources may affect the relative success in implementing national health policies and subsequently health outcomes.

/3

Healthy policies?

This chapter aims to describe key aspects of the policy space in which health inequalities persist in high-income countries. First, I detail one of the most prolific trends in public health policymaking in Europe and discuss how public health policies based on social and structural inequalities are vulnerable to reinterpretation and drift toward established methods for disease prevention and treatment. Second, I describe issues of meritocratic governance, how it relates to equality of opportunity and outcome, and how this system may generate social inequalities in life chances. Third, I describe the Norwegian case as a somewhat unique adopter of the social determinants of health paradigm, how policy trends were adopted into legislation, as well as how issues of meritocratic fairness relate to social preferences in egalitarian and libertarian contexts.

3.1 Public health governance in the 21st century

Early 2000s public health governance was largely driven by the assumption that good health would lead to economic prosperity and social progress (Dahl, Bergsli, and Van der Wel 2014, p. 41). However, a central public health policy development in Europe throughout the 21st century is the Health in All Policies (HiAP) agenda. HiAP is described as "an approach to public policies across sectors that systematically takes into account the health implications of decisions, seeks synergies, and avoids harmful health impacts in order to improve

population health and health equity. It improves accountability of policymakers for health impacts at all levels of policymaking. It includes an emphasis on the consequences of public policies on health systems, determinants of health and well-being" (World Health Organization 2013). Though aspects of this policy agenda have a longer history, the umbrella concept itself was introduced for the first time to the European Union in 2006 and has subsequently been established as an instrumental approach to horizontal health policy at the European and at global levels (Ståhl 2018).

HiAP represents "a way of working that embodies a normative vision for a society in which well-being and social justice is a central objective" (Godziewski 2021, p. 232). In practical terms, this *whole of government* way of working includes monitoring population health, increasing public health competence and counseling activities, establishing public health partnerships and intersectoral collaborations, finding funding for public health projects, and establishing public health coordinators (Helgesen, Fosse, and Hagen 2017). These activities reflect the overall strategy and commitment to health promotion and disease prevention (Helgesen 2014). While the HiAP agenda has been successfully adopted in many countries, its ability to effectively address social inequalities in health is unclear. Dahl, Bergsli, and Van der Wel (2014) recommend that social inequalities in health be given equal status to health in the overall HiAP framework, suggesting that the HiAP agenda does not capture a commitment to reducing social inequalities in health. Greer et al. (2022) argues that while HiAP served as a good starting point, it should be integrated and bidirectional, acknowledging that improving health and reducing health inequality enables major co-benefits for other sectors. Lynch (2017) argues that action addressing health inequalities is hampered due to the inherent difficulty of multi-sectoral policymaking, citing that the causal framework for health inequality is sufficiently complex to provide barriers to cross-sectoral action. Such barriers include coordination problems, issues of sustainability, clarity about roles, responsibilities and goals, political power plays, and the need to negotiate roles and resources of public health versus medical actors and health versus other sectors.

A notable criticism of public health policymaking is lifestyle drift: the tendency of health policy to drift downstream toward individual proximal causes of health such as lifestyle, even if it was originally motivated by addressing upstream (distal) political and social causes of inequality and health inequality (Popay, Whitehead, and Hunter 2010). Inherent to this concept is the flawed assumption that lifestyles of different socioeconomic groups are freely chosen, as opposed to being shaped by the social and economic environments that people live in (Dahlgren and Whitehead 2021). In the context of EU health policy, Godziewski (2021) argues that HiAP accelerates lifestyle drift and describes the framework as chameleonic: "the EU interpretation of HiAP as multistake-

holderism diffuses responsibility by shifting it away from government, reflects taken-for-granted norms and world views which are actively hidden behind a language of neutrality and reasonableness." If a government limits its own responsibility in public health and health inequalities, this precludes the possibility of considering HiAP as being about political and social determinants at all.

Lifestyle drift in health policy may occur in other ways. Fosse and Helgesen (2020) argues that social inequalities in health constitute a "wicked" problem, namely a problem that causes disagreement regarding both its causes and its solutions. The main argument is that in the Nordic countries, the solution is to tame the problem by redefining it into a less contested problem such as lifestyle factors. This in turn makes the problem manageable, often via established health services or individual action. The result is that practical policy measures seldom align with the concepts of the social determinants of health that can be found in national policy papers, resulting in a lack of commitment to policies explicitly addressing the social determinants of health. Lifestyle drift makes the wicked problem of health inequalities manageable using existing medical tools and frameworks for on-the-ground disease treatment and prevention, thus bypassing the structural issues of *social* inequality that policies initially recognize.

3.2 Meritocracy, opportunity, and outcome

The Weberian notion of class argues that the market generates classes and that classes are sets of structural positions. Classes are generated by the kinds of assets held by individuals and by what these mean to the market. Class positions are empty until filled and exist independently of individual occupants of these positions. One purpose of Weberian class analysis is to capture how social relationships within these markets shape life chances and their distribution (Breen 2005). These principles may be viewed as a theory about how social relationships within markets are linked to the distribution of life chances. Under the Weberian notion of class, education is one of several such sources of stratification. Education is reflective of social status in a broad manner that is related to both material and non-material resources. Combined with income, these two indicators "provide insights into the material and non-material components of social standing that generate socioeconomic gradients in health" (Beckfield, Olafsdottir, and Bakhtiari 2013)

Muntaner, Eaton, and Diala (2000) deal with stratification and class as two distinct things in the context of mental health, as well as the selection/causation issue. They argue that "social stratification" as understood in the literature on

health inequalities, while inspired by Weberian ideas of social class, has been been generalized and less directly related to Weber through more agreeable, soft terminology like socioeconomic status and socioeconomic position. This conception of class and stratification relies on the ordering of individuals according to dimensions that serve as descriptors of inequalities in social *resources*, in contrast to application of the Marxist concept of class stemming from ownership or control over assets and the means of production. In the context of evidence-based policy, Navarro (2009) criticizes the WHO report on health and health inequality by pointing to the neo-liberal paradigm and to neo-liberalists as the dominating class leading to un-social policy developments and increasing inequality. Further, he argues that since class has been relegated to "status" (less politically contentious) and that the main strategies employed to reduce health inequalities – medical care, disease prevention, and health promotion programs based on behavioral and lifestyle interventions – are insufficient because they do not include political, economic, social, and cultural interventions that actually deal with the social as opposed to the individual determinants of health.

It is important here, then, to emphasize that social status, when viewed in light of Weberian stratification, enables access to fundamental resource in societies organized by merit and systems that reward the acquisition of material and immaterial resources. Modern meritocracy is defined by the value of technical skill, sometimes interchangeably referred to as human capital. Investment in education, for instance, is expected to yield future revenues in income or a preferable bargaining position in the labor market. Equality of opportunity serves as the categorical imperative for the principle of individual merit. Some inequalities within this meritocratic system are thus justified on the basis of achievement, and expectedly, it recreates inequality over generations (Bell 1972). In other words, inequalities are considered *just* if they pass the test of meritocratic fairness, whether the source of inequality comes from individual achievement or from luck.

We arrive at a point of tension between meritocratic organization and health inequalities because the interplay between socioeconomic positioning and health over the life course suggests that individual achievement and luck are likely interwoven in generating health outcomes. Further, the social groups associated with individual achievement (education, income, occupation) are the very same as those often used as social group rankings in health inequality research and often considered unfair.[1]. This is to say that the inequalities considered fair within meritocratic organization and the equality of opportunity paradigm seldom translate to the subject that scholars on health inequality

1. See McCartney et al. (2019) for a selection of features associated with definitions of health inequality

most often measure: health outcomes.[2]

3.3 The case of Norway

Health and health care (in Norway) are both relatively "timeless" issues. They are also considered "valence" issues – issues that tend to be linked to parties with conditions that are positively or negatively valued by the electorate – as opposed to a "position" issue – one that involves advocacy of government actions from a set of alternatives distributing voter preferences (definitions, (Stokes 1963)). However, as described in Chapter 2, social inequalities in health are often presented within a framework of individual health behavior, lifestyle, and other proximal determinants of health. Health and social policies have been relatively important issues for the Norwegian population in most national elections since 1977. In contrast to health and social policies, issues of social *cohesion* have been an issue of comparatively low importance for the majority of the Norwegian electorate (Hesstvedt, Bergh, and Karlsen 2021).

Norway enacted a national insurance scheme (providing benefits such as disability benefits, unemployment benefits, and benefits during sick leave) as early as 1967. However, Norway was for a long time considered somewhat of a laggard in recognizing health inequality as a social problem (Van der Wel, Dahl, and Bergsli 2016). Mackenbach et al. (1997) showed that socioeconomic inequalities in health were no more favorable in the Nordic countries as compared to the rest of Western Europe. Educational inequalities in mortality increased in the latter half of the 20th century (Strand et al. 2010). While most groups fared better over time in terms of overall health, decreases in mortality and increases in longevity in high-income and high-education groups outpaced those in low-income and low-education groups, generating larger health inequalities (Næss, Rognerud, and Strand 2007). These inequalities, along with health inequalities between occupational groups, persisted in Norway in the form of a gradient running from higher to lower social and economic position (Goldblatt et al. 2023). Policy documents throughout the 1990s do refer to and acknowledge health inequalities, but these received comparatively little political concern (Dahl 2002). Later, Norwegian governments would explicitly recognize social inequalities in health as a policy issue for reduction. Several white papers have addressed this issue; a white paper from 2015 addressed issues of mental health and healthy lifestyles and emphasized the gap between the top and bottom of the social gradients, and a white paper from 2019 focused on healthy

2. I have not distinguished between health *inequities* and health *inequalities*. Section 4.1 clarifies the conceptual logic positioning health inequalities as a species within the differences in health outcome *genus*.

behaviors as a way of reducing social inequalities in health in the population (Norwegian Ministry of Health and Care Services 2015; Norwegian Ministry of Finance 2019; Goldblatt et al. 2023); it appeared in a white paper on how research may be used to address global challenges in health, health care, social inequalities in health, and research-based welfare policy (Norwegian Ministry of Education and Research 2013); social inequalities in health both within and between states appeared in a white paper on global health in foreign and development policy (Norwegian Ministry of Foreign Affairs 2012); and one white paper applied the Dahlgren-Whitehead model (Whitehead and Dahlgren 1991; Dahlgren and Whitehead 2021) to explicitly recognize social inequalities in health as an issue for planning, governance, and social organization (Norwegian Ministry of Health and Care Services 2013). The most important white paper, however, was entirely dedicated to developing a national strategy to reduce social inequalities in health and recognized the linkage between social inequalities and health inequalities (Norwegian Ministry of Health and Care Services 2007).

The Norwegian Public Health Act, enacted in 2012, established a reduction of social inequalities in health as a core objective. The Public Health Act mandates national, regional, and local politico-administrative authorities to address the social determinants of health by developing policies and implementing actions (Helgesen, Fosse, and Hagen 2017). It further mandates all sectors of these institutions to work for public health and its fair distribution within the population (Dahl, Bergsli, and Van der Wel 2014, p. 301). Thus, all levels and sectors of government share the responsibility and are accountable for public health and health inequality policy. The Norwegian Public Health act is explicitly based on five basic pillars of public health work: principles of social cohesion in health, HiAP, sustainable development, preparedness, and participation (*Public Health Act 2011* n.d.). Overall, Norwegian public health policy has been described as embedding a strong *whole-of-government* approach to ensure that reducing health inequalities is included in policy development; local municipalities are responsible for delivering many policies in public health and health inequality, supported by the counties, but the policy goals and programs are formulated at the national level (Goldblatt et al. 2023).

While in line with the HiAP agenda, the Norwegian strategy is relatively unique in the broader European context, with Norway being the only country to highlight a reduction in income inequality as a means to reduce health inequalities (Lynch 2017). While this era of policymaking embraces the social determinants of health perspective, concerns have also been raised. Van der Wel, Dahl, and Bergsli (2016) argue that while reducing social inequalities in health remains a high-priority goal, means have been meager and to some extent absent. Challenges, such as a stubborn inequality structure, policy re-orientations, and a lack of focus on the gradient in implementing cross-sectoral reforms, have been

cited in the Norwegian case. Many Norwegian municipalities have tended to pursue individualistic and sectoral approaches to health inequalities, rather than factors outside the domain of the health sector (Grimm, Helgesen, and Fosse 2013). Fosse and Helgesen (2020) argue that more recent policy documents do place a greater emphasis on individual measures, that measures have been directed toward the local level, and that the problem of social inequalities in health has been tamed into problems focusing on individual lifestyle issues solvable by health services or through collaboration between local services, leaving the wider determinants of health somewhat behind.

The persistence of health inequalities through the development of the welfare state and with overall increases in population health has been deemed a great paradox in the literature on health inequalities (Mackenbach 2019). However, social root causes such as material and immaterial resources and their value in the market remain unequally distributed, and the potential consequences of lacking these resources remain even in the most generous and high-income contexts, such as Norway. The fundamental inequality structure in Norway is stubborn (Van der Wel, Dahl, and Bergsli 2016). The dominant trend in European health policymaking in the 21st century has emphasized intersectoral cooperation, multistakeholderism, and biomedical models of health. This approach is sensitive to reinterpretation toward a normatively neutral language (Godziewski 2021) and shifts attention away from underlying social inequalities toward individual-based models of health (Lynch 2017) undermining policies that explicitly call attention to the effect of social inequalities on health. Viewed in this way, the persistence of health inequalities is not a paradox at all, as the fundamental structure of social inequality has remained somewhat unchanged (Dahl and Van der Wel 2015).

Following Eckstein (2015), I here note that while Norway as a *case* certainly has its peculiarities, it shares many political and social traits with other high-income countries. For instance, while the Norwegian population has markedly egalitarian views toward redistribution as compared to the libertarian redistributive values found among Americans, Americans and Norwegians share similar ideas of meritocratic *fairness*, considering inequalities due to individual productivity as fair and those inequalities attributable to luck as unfair (Almås, Cappelen, and Tungodden 2020). And while the health inequality policies in Norway are unique in some ways, they also share the broader HiAP agenda. Given its egalitarian ethos and robust redistributive institutions (Van der Wel, Dahl, and Bergsli 2016) and political intent in reducing health inequalities, Norway serves as a valuable empirical backdrop for studying how and why social inequalities in health continue to persist in modern, high-income welfare states. Further, the substantial responsibility for public health and social inequalities given to local governments allows for analysis of subnational local administrative units. Comparative analysis is, however, ultimately necessary to determine whether

or not the mechanisms generating health inequalities are to be considered contextual or representative of broader generalizations.

This chapter has sketched out important trends in health and public health policymaking in the 21st century and discussed the impact of meritocratic organization and how these relate to differences in health between social groups. I have showed that the Norwegian context is unique with regard to its egalitarian ethos and legislative commitment to reducing health inequalities but that it shows similarities to other countries in its implementation of the HiAP agenda and in its social understanding of meritocratic fairness. In the empirical work provided in this dissertation, Norway should therefore be considered the laboratory in which the generalized framework presented in Chapter 2 is applied.

4

Methodological considerations

This chapter is primarily reserved for discussing overarching methodological issues and assumptions in the research articles, but it also expands on subjects that would not fit in the articles themselves. I restrict the discussion to a handful of major subjects: the usage and operationalization of health outcomes at different stages of the life course, the value system implicit in measurement, the usage of observational data and studies of association and their role in telling causative stories, and how the usage of several levels of analysis affects inference on societal- and individual-level processes. I refer to specific examples from the articles that reflect these issues when necessary. Data sources are described in Section 4.8. Details on the statistical analyses employed in the three papers are described in Section 4.9. I begin the chapter with an attempt to translate the conceptual logic of the ladder of abstraction (Sartori 1970) to the field of health inequalities.

4.1 Health inequality and the ladder of abstraction

In his seminal paper, Sartori (1970) argued that any (comparative) empirical investigation relies on well-defined concepts. Defined concepts and labels serve as data containers into which one can either classify those belonging to a specific kind or quantify those that are similar in kind but differ in degree. The ladder of abstraction details the extent of each data container; a high level of abstraction signifies a concept in which the necessary and sufficient conditions for classification are small in number, thus applicable to a large number of observations (i.e., the concept is maximally extended but minimally intended). A concept is defined by *negation*, by what it is not. At the highest level of abstraction, then, the distinction is categorical, represented by the *genus*. One moves down the rungs of the ladder of abstraction by adding criteria to the concept, such that the concept itself now denotes fewer logical and empirical observations. At the lowest level of abstraction, the concept has maximal intension such that the definition of the concept itself trends toward the contextual and descriptive. Sartori's intension increases with the logical process of adding properties (necessary *and* sufficient) rather than by expanding concepts via adding properties that are necessary *or* sufficient (Mair 2008). The purpose of this ladder of abstraction is to avoid stretching a concept into fitting new cases that sensibly would not fit, leaving the concept and its label unfit as data containers.

There are of course two main concepts in demand of conceptual clarification present in this dissertation: "health" and "class/status." These are contentious concepts indeed. When Callahan (1973) discussed the many criticisms leveled at the WHO definition of "health,"[1] he was expecting to be accused of beating a dead horse. I will leave this beating to the professionals. I will, however, argue that this concept of health seems to evade Sartori's ladder of abstraction entirely; the generalized definition of health seems simultaneously to refer to all humans and yet describe none of them. It is a concept that is simultaneously maximally extended and intended. Further, class theories presuppose that all people and families can be defined as occupants of a class, enjoying some sort of status, or possess a relative amount of social resources and so on. Both concepts seem to occupy all levels of abstraction under the same label; the labels themselves seem (on the surface) inherently continuous, thus escaping definition by *negation*, that is, by what they are not.

1. "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity", later expanded to include the view that health is a resource for everyday life, not the objective of living (McCartney et al. 2019).

Ladders are primarily associated with the *iconography* of health inequalities and has served as a visual conceptual model (Krieger 2008), not a description of its *conceptual logic* per se. However unclear the basic components that enter into the definition of health inequalities are, I find use for the necessary *and* sufficient logic of the ladder of abstraction in its definition.

Table 4.1 translates the ladder of abstraction to the concept of health inequalities. At a high level of abstraction, differences in health outcomes can be understood as those differences in health between people, populations, or subpopulations. It is maximally extended because it does not restrict analysis to specific subgroups, nor does it define the level of analysis or the health outcome to be studied. Defined in this way, normativity is avoided; however, it may be inferred because of the term's connotations; rarely is the term "inequality" discussed without reference to unfairness or an ambition for its reduction. However, this high level of abstraction makes no reference to where health inequalities originate or which inequalities are deemed unjust.

Moving down the ladder, one possible extension of the concept of difference in health outcomes is one in which class or socioeconomic determinants are pursued. At this conceptual level, I explicitly apply ideas of structural determinants and social root causes of health differences, thereby excluding factors such as genetic endowment or natural talent (i.e., chance). It makes an explicit normative statement insofar as it specifies that inequalities that are reasonably preventable through redistributive policy and social organization are deemed unjust and unfair. Indeed, this idea of health inequalities flows naturally from the assumption that differences in health outcomes are systematic and not random (McCartney et al. 2019). This extension of "differences in health outcome" achieves two things: first, it clearly establishes the *genus* as differences in health outcome and thus health inequalities as a species located within the genus. Second, it shows the principle of conceptual distinction by *negation*; social inequalities in health are the differences in health outcomes between social groups. It is not the differences in health outcomes *within* social groups.

At the bottom of the ladder, we could add to the definitions above using intersectionality. Intersectionality constructs a matrix of power, privilege, and disadvantage that place people in mutually constructed categories (Bambra 2022), and these inequalities vary across time and space and between cultures (Gkiouleka et al. 2018). It stands to reason that intersectional health inequalities are bound by culture, time, and space and thus do not travel significantly long distances between contexts. Narrow-gauge theory is required to ultimately pinpoint why such inequalities exist, as they are expected to be embedded in place and history.

Table 4.1: Health inequalities and the ladder of abstraction

Abstraction	Description	Properties
Universal (*Health outcome differences*)	Differences in health outcomes between individuals, populations, and subpopulations.	Maximal extension: does not define subgroups, level of analysis, or health outcome. Non-normative, includes differences related to chance.
General (*Health inequalities*)	Differences in health outcomes between individuals, populations, and subpopulations deemed to be unjust or unfair that plausibly result from social root causes/structural determinants amenable by redistributive policies and social organization.	Balance between denotation and connotation. Restricts analysis to social group differences, defines health by reference to conditions that may reasonably be considered preventable. Normative.
Configurative (*Intersectionality*)	Differences in health outcomes resulting from the intersection of social categories that are mutually constructed and that locate people within a matrix of power, privilege, and disadvantage.	Maximal intension, minimal extension. Contextual information required in order to describe and explain phenomena narrow in scope.

4.2 Implicit values and measurement

The measurement terminology applied in research on health inequalities requires some clarification; namely the distinction between "health inequalities" and "social group differences in health." "Health inequalities" has been used to refer to composite measures of the variation in health status across individuals in a population akin to income inequality measurements such as the Gini coefficient, while "social group differences in health" or "social inequalities in health" refers to health differentials between subgroups of a population (Murray, Gakidou, and Frenk 1999). Like many European authors (Marmot et al. 2010; Mackenbach 2012; Eikemo and Øversveen 2019; Bambra 2022; Dahl 2002), I have used these terms somewhat interchangeably to refer to *social group differences in health* throughout.

This approach defines social position as a fundamental latent variable that determines health and implies alignment with the normative statement that health inequalities are primarily interesting to the extent that they mirror inequalities in socioeconomic status (Wagstaff, Paci, and Van Doorslaer 1991), as opposed to the positive assessment that health inequalities are intrinsically important, independent of their correlation with other aspects of well-being (Murray, Gakidou, and Frenk 1999). Whitehead (1991) argues that if we are to consider inequalities as avoidable and unfair, then studying social inequalities in health is implicitly laden with values. Muntaner (2013) further argues that studies of disease share these implicit values of wanting to eliminate human suffering and explicitly act on them when social interventions and evaluations are conducted. I would add that the implementation of any public health policy is inherently laden with values, regardless of the extent of reasonable and normatively neutral packaging (Godziewski 2021), given the overall aims of these policies.

While Article II specifically calls attention to Popperian logic (Popper 1963) and the argument that theories must be evaluated by their ability to predict outcomes, I find that the articles in this dissertation align to a greater extent with scientific realism as argued by Muntaner (2013). Inherent to this approach is the rejection of *empiricism* and the acknowledgment of theoretical constructs and their implicit value statements in the measurements of health inequalities applied in the articles included in this dissertation. While scientific realism emphasizes "why" health inequalities exist in the first place (Muntaner 2013, p. 852), it does not reject the assumption that theory must in some capacity predict real outcomes. Predictive capability is foundational to the epistemic statement that scientific realism makes (Psillos 2009) and is, as such, consistent with the statement in Article II, albeit with some difference in emphasis. My point here is that measurements of NCD prevalences between different levels of education, income, and work status – while not directly observable as "social mechanisms"

in the realist sense – inherently imply that mechanisms such as social class relations exist as well as the existence of a social causal model. What scientific realism ends up contributing, in this regard, is the acknowledgment of the implicit value statements and the assumed social causation model present in health inequality research. In turn, based on how institutions and the state are assumed to influence stratification, the distribution of resources, or regulation of the labor market and protective policies against unemployment or lack of income, as assumed by welfare state research frameworks (Esping-Andersen 1990; Bambra and Eikemo 2009; Bambra 2011b; Eikemo and Bambra 2008; Beckfield et al. 2015), the political context emerges as the ultimate root cause in the social causation framework. These perspectives are, if not incompatible with a general empiricist approach to the measurement of health inequalities, certainly unreflective of it.

I will, however, briefly return to the ladder of abstraction to note that the normative approach to the measurement of health inequalities is not fundamentally incompatible with the positive approach to measuring differences in health outcomes. They merely exist along different rungs of the ladder of abstraction; differences in health outcomes between individuals and populations is the universal concept to which the concept of health inequalities belongs. I discuss the choice of health and health-related outcomes in more detail below, but I will point out here that the works in this dissertation adhere to this universal concept of differences in health outcomes here sketched out using the ladder of abstraction. Article I discusses the income–health gradient as a matter of differences in health outcomes between income groups. It thus excludes those differences in health outcomes that may exist within income groups. Article II extends the measurements to include educational and occupational groups, while adding discussion on the differences in health gradients between these categories. These articles are committed to the inherent *inequality* connotation. Taken together, they emphasize both immaterial (education, occupation) and material (income) resources in inequality in health outcomes (i.e., life chances). Article III does, however, methodologically occupy the more universal "differences in health outcome" approach. However, it emphasizes issues of inequality by acknowledging the potential consequences of social and geographical differences in early life health input in a theoretical sense.

4.3 Health outcomes

I have pursued a broad approach to measuring health outcomes, reflected in that the three articles presented in this dissertation measure health outcomes in very different ways. The mechanisms discussed and the health outcomes measured are, however, somewhat reflective of a life course approach (Bambra,

Netuveli, and Eikemo 2010) to health inequalities: more specifically, health care input in early life, self-rated health in all adolescent and adult age groups, and non-communicable disease prevalence in mid-life and older cohorts.

In Article I, a self-rated health measurement is applied. Self-rated health has seen prolific use in the study of health inequalities (Olsen, Lindberg, and Lamu 2020; Eisenberg-Guyot and Prins 2020), is correlated with other direct or indirect measures of health and shows good test–retest reliability (Mackenbach et al. 1994), and serves as a predictor of mortality risk (Lorem et al. 2020; Jylhä 2009). However, the precise meaning of self-rated health and its responsiveness to concepts of health and illness have been a major source of discussion in the literature (Martikainen et al. 1999; Altman, Van Hook, and Hillemeier 2016; Jylhä 2009; Smith, Shelley, and Dennerstein 1994; Manderbacka, Lahelma, and Martikainen 1998; Manderbacka 1998; Krause and Jay 1994). Self-rated health may be considered a holistic indicator where the individual is expected to consider culturally and historically varying conceptions of health (what it is), referential considerations such as earlier and others' health experiences and potential future developments (comparison), and conventions in expressing positive and negative opinions on their health (cultural) (Jylhä 2009). The measurement was chosen primarily because it creates an intuitive end point to the hypothesized embodied experience of long-term psychosocial stress, as described by Wilkinson and Pickett (2006) and Pickett and Wilkinson (2015), and its observed income–health gradient in Norway (Olsen, Lindberg, and Lamu 2020). As such, it was expected to be somewhat responsive to psychosocial mediation in all age groups when compared to other morbidity measurements available in ESS7. This paper includes the widest age scope of the three papers; however, the sample emphasizes working ages. Still, health inequalities that may be attributed to material disadvantages in income are likely to affect health at most stages of the life course *a priori*.

In Article II, we specifically target non-communicable diseases as health outcomes due to the great burden of disease this category represents (Vos et al. 2020). Cancers, cardiovascular diseases, and diabetes all show socioeconomic gradients, albeit to a varying extent (Mackenbach 2019). Additionally, non-communicable and degenerative diseases are more likely to occur at middle-to-late stages in the life course, as evidenced by the variable importance score of age in the models. Thus, the differential prevalence of non-communicable disease between social groups is more likely identifiable in the age groups present in the Tromsø Study as compared to studies including all age groups.

In Article III, we specifically target antibacterial dispensing rates as a reflection of health input in pediatric patients. Overuse of antibacterials in the early stages of life has been associated with increased risk of chronic diseases later in life (Korpela and De Vos 2016; Mårild et al. 2013; Risnes et al. 2011; Sander

et al. 2019); the literature relates parental education to overuse in pediatric patients (Thrane et al. 2003; Pichichero 1999; Filippini, Masiero, and Moschetti 2006) and shows an association between area-level deprivation measurements and antibacterial prescription rates (Thomson et al. 2020; Koller et al. 2013; Adekanmbi et al. 2020). We specifically targeted antibacterial dispensing from primary health care services because the majority of prescriptions to children at this early age are prescribed in this sector, and the likelihood of social differentiation for parental health care seeking and parent influence on treatment is greater in primary health care services than in hospital services.

To summarize, health and health-related outcomes were chosen and evaluated for appropriateness as argued by Blane, Smith, and Bartley (1993) at the different stages of the life course. It follows that health outcome measurement used on wide age cohorts opens discussion on the social dynamics that generate health inequalities at different stages, while specificity becomes more important as the width of the age cohort narrows. Failure to address this scope in the measurements would likely result in *underestimating* social group differences in health. This is particularly important as all measurements used in the articles are considered absolute measurements (i.e., "real" numbers).

4.4 Absolute measurements

The distinction between absolute and relative measurements of health inequalities seems a pervasive topic regardless of the health outcome being studied (Regidor 2004a; Regidor 2004b; Mackenbach 2019; Dahl, Bergsli, and Van der Wel 2014; Harper and Lynch 2006). This distinction is strictly mathematical/statistical; relative measurements are those that apply mathematical formulas to generate a measurement that deals in ratios between pre-determined groups, such as relative risk, risk differences, or hazard ratios. Absolute measurements can be defined as the real differences in health outcomes or prevalences between the social groups under study, represented through (for instance) regression coefficients (Dahl, Bergsli, and Van der Wel 2014, p. 65). Some critics of the relative measurement approach have argued that the magnitude (and particularly the estimation of trends) of health inequalities are mathematical artifacts because as populations become healthier, poor health outcomes become rarer, which results in smaller absolute differences tending to inflate relative risk measurements (Scanlan 2006). This idea is somewhat controversial because both absolute and relative measurements of health inequality may increase or decrease concurrently even in high-income welfare states, where *population health* is generally considered good (Mackenbach 2012).

Still, working with the general idea that real numbers are more easily in-

terpreted than ratios and relative measures, I have applied absolute measurements of health inequality throughout when modeling and presenting empirical relationships. Clarity is achieved primarily by leaning the presentation toward predicted absolute values whenever appropriate: in reporting the (non)-moderating effect of income inequality in Article I, in reporting absolute proportions and differences in proportions in prediction accuracy in Article II, and when presenting predicted decrease in municipality dispensing rate trends and its interaction with area-level education in Article III (particularly important due to the centering scheme and square root transformation applied in the statistical model).

4.5 Association and causation

Common to all articles in this dissertation is that they rely on observational data using various methods of association. Emphasis is placed on prediction in reporting results, regardless of the methods used. In this section, I deal primarily with how studies of association may contribute to inference on causal processes and their implications for evidence-based health policy.

At this point, I think everyone is aware that correlation does not equal causation. In light of this, I find the discussion by Hernán (2018) particularly sobering; most studies of association (including the articles in this dissertation) have causal ambitions and goals. Indeed, if the goal was merely a description of the statistical association, we would not need controls, yet most if not all studies (e.g., on health inequalities) include a plethora of controls. Three supporting points arise: first, confounding is a causal concept, not a concept of association; second, skirting around the word "causal" leads to a lack of clarity regarding the goal of the research that is more often than not causal; and third, post-treatment/included variable bias when including controls and a call for parsimony in statistical modeling.

I am equally guilty of this fuzzy phrasing; in Article III, the phrases "association," "relationships," and "link/linked" are present even though most of the discussion of the results features theoretical descriptions of cause and effect. Similarly, Article I is heavily deductive in its approach, directly citing theoretical causal mechanisms and, as such, it comes "dangerously" close to presuming causal effects, though no such statement of causal *effects* per se is claimed. Rather, the language discusses the likelihood of the causal processes explaining the income–health gradient given the results from association. Article II is the least of the offenders, as the explicit goal should be considered associational inference via prediction, although the discussion itself is still informed by causal mechanisms. Similarly to assumptions on measurement, my approach mostly

aligns with the scientific realist (Muntaner 2013) approach by letting theoretical assumptions provide guides and explanations, and thus applying a conception of causation as *robust dependence* (Goldthorpe 2001). In other words, while my statistical approach is associational, my goals are causal.

Estimating causal effects typically remains outside the realm of statistical studies based on association (Pearl, Glymour, and Jewell 2016). Given my causal goals, I find it necessary to contrast causation as robust dependence with the potential outcomes framework associated with Rubin (1974) and Splawa-Neyman, Dabrowska, and Speed (1990). Public health research considers methods of causal inference useful because *if* an association is causal, there exists a possibility for intervention. Described as the gold standard, the randomized control trial is usually considered the best in the evidence-based hierarchy and is implicitly and sometimes explicitly embedded in public health practice, policy formulation, and regulatory processes (Glass et al. 2013). The potential outcomes framework embeds the idea of causation as *counterfactuals*, defined as the difference in state (outcomes) in which a treatment occurred, versus the (hypothetical) state in which it did not occur, all else being equal. Thus, causation and causal effects are based on the question *what if*, which is by definition unanswerable (Cunningham 2021). *What if* questions, however, define the set of potential outcomes in which only one factually occurs.

An extensive set of statistical tools has been developed based on this causal logic, and these could potentially be used in the empirical investigations in the three papers in this dissertation. For instance, matching techniques such as propensity scoring or coarsened exact matching (King and Nielsen 2019; Iacus, King, and Porro 2019) could conceivably be applied in the first article in order to create comparable groups on covariates onto which the treatment and mediator effects were measured, thus approximating the causal *effect* of income on self-rated health. However, while matching is a well-established approach in the context of binary treatments, matching is underdeveloped for continuous exposures (Wu et al. 2022). An alternative would be to reduce the treatment categories into high- and low-income earners. However, this would result in an imbalanced data loss between countries because the in-sample income distributions vary between countries, and would go against the emphasis on the income–health *gradient* in the first place. In the second article, machine learning tools could aid in the estimation of heterogeneous treatment effects (Imai and Ratkovic 2013) under different levels of education, income, or occupation status. This would arguably answer the call for investigating conditional causal relationships in health inequality research (Montez and Friedman 2014) but is ultimately a different investigation to what we propose. Our article could, however, serve to establish baseline accuracies for future endeavors into causal inference on the conditional effects of education, income, and occupation on NCD prevalence and outcomes. As for the third article, future studies based

on constructing counterfactuals and synthetic cases (Abadie, Diamond, and Hainmueller 2015) could feasibly investigate the extent to which area-based policies on antibacterial dispensing rates do indeed have a causal effect. The third article thus shows that sociodemographics should be considered when constructing the synthetic case.

The commitment to causation as robust dependence when viewed in terms of the potential outcomes framework shows some undeniable weaknesses with regard to inference on causal *effects*. However, where the robust dependence approach is found lacking in causal inference, its methods are well equipped to explore the full variation in *patterns* present in the data. Whatever we learn from these observational approaches can, in turn, inform causal designs in future research endeavors. Causal inference is indeed the ultimate goal for the collective research endeavor on health inequalities. Studies of association are a part of the knowledge base that enables researchers to pose reasonable causal questions.

4.6 Methodological pluralism and evidence-based policymaking

There are studies that systematically compare results from observational and experimental studies, and the overall conclusion appears to be that they agree and show comparable results more often than not (Dahl, Bergsli, and Van der Wel 2014, p. 57-8). While Glass et al. (2013) primarily argue that causal inference is necessary to identify the effects of an intervention, they concede that a *sole focus* on causal inference on interventions that are "easy" to evaluate risks ignoring upstream interventions for which the randomized experiment cannot be conducted, and where the potential for the greatest impact may be found. Supporting this point is that randomized control trials are not always feasible in population health or health inequality studies; health inequalities often arise from cumulative exposures over the entirety of the life course, making the counterfactual inference framework somewhat of a challenge to implement (Elstad, Heggebø, and Dahl 2022).

This methodological diversity did, to some extent, motivate our application of a predictive algorithm on estimating the relationship between socioeconomic indicators and non-communicable disease outcomes in Article II. I detailed the scientific rationale for algorithmic modeling above, but another point of emphasis in the article is the potential in using predictive modeling in order to gain more detailed insight into empirical relationships between social root causes and disease prevalence – not only useful for further theoretical modeling,

but also a potential refinement of evidence-based policy development. I will take the opportunity to note that predictions are an inherent part of the data modeling strategy. In the other two papers, prediction is presented as both model fit, and predicted values are used to communicate results. Article II merely makes the predictive framework a major feature.

Two years prior to the white paper on health inequalities (Norwegian Ministry of Health and Care Services 2007), a Norwegian Green Paper (Norwegian Ministry of Education and Research 2005) provided a level-headed take on methodological diversity in health and public health research. It included qualitative and associational studies in the mix of valid and necessary strategies for producing evidence-based policies and evaluating The Health Research Act. What is not mentioned, however, is prediction. A recent white paper discussing a "data-driven economy," primarily as a resource for business and industry (Norwegian Ministry of Local Government and Modernisation 2021), provides extensive references to modern tools of prediction (machine learning, artificial intelligence, and big data). Health data and "real-world data" are mentioned in this context, yet, no particular reference is made to predictive modeling. Rather, The Norwegian Directorate of Health plan to establish a National Health Analysis Platform that will "simplify access to health data for analytical purposes, "make it possible to use health data more actively for developing drugs, medical technology, and services," "create new opportunities for Norway's health industry and attract international enterprises," and "provide knowledge that contributes to improving the quality of the health services and to developing better treatment, prevention, monitoring and research" (Norwegian Ministry of Local Government and Modernisation 2021, p. 53). And while the strategy goes to great lengths to address concerns researchers have about data accessibility and linkages between registries (Eikemo and Øversveen 2019), it severely limits the extent to which real-world health data could in fact be used. The overall phrasing suggests a dominant application of a medical model of health wherein machine learning and data are mere tools for better services, treatment, and, to some extent, preventive action. Indeed, references to social inequality or to health inequalities are nowhere to be found. The extent to which health inequality researchers can harness these data resources for predictive or causal modeling in the future is somewhat uncertain.

Article II cites primarily the ways in which machine learning and predictive modeling can offer tools for exploration and new discoveries for researchers, as well as their potential for extending the toolbox for scientific evaluation. Machine learning tools can help us exhaust possibilities in model specifications and study population heterogeneity. Further, testing a model out of sample minimizes the risk of overfitting and allows for evaluating the overall performance of the model in explaining a given output (Molina and Garip 2019). The issue of overfitting is of particular relevance to evidence-based policymaking,

and it is here that the counterfactual logic used in matching (King and Nielsen 2019; Iacus, King, and Porro 2019) overlaps with the literature on ensemble methods such as the random forest algorithm (Breiman 2001; James et al. 2013) most clearly; they both ultimately aim to *reduce model dependence* in traditional data modeling. Researchers on health inequality should therefore embrace (to some extent) both predictive and causal frameworks and acknowledge their common goal in providing sound, consistent evidence for policymakers to consider.

4.7 Vertical, horizontal, and comparative analysis

The dissertation is made up of articles that include analysis of individual, subnational, and comparative data. This section details some methodological considerations when working with multilevel data.

A notable aspect of the articles is the emphasis on "between-effects," that is, the proportion of the total statistical variance of the outcome that can be attributed to between units or groups, as opposed to "within-variance," the variance within units. Two of the three articles apply a hierarchical approach to data modeling (Luke 2004; Gelman and Hill 2006). The third applies a random forest algorithm (Breiman 2001) in a location-based cross-sectional cohort study. While one of the articles emphasizes and addresses both the within and between variance at the same time, I argue that the focus is still on the variance between units. Article III investigates the extent to which *trends* in antibacterial dispensing rates vary *between* subnational units of administration by using multilevel growth curve modeling. It emphasizes the structural conditions between subnational units of organization by associating this trend variance with municipality population education levels.

Whole-nation bias (Rokkan 1970) is at this point a well-known issue in national and cross-national research. Whole-nation bias describes the tendency to gravitate toward national level data and national units of analysis, leaving researchers blind to subnational political and economic transformation and tending toward research designs that include many variables but small samples (Snyder 2001). Whole-nation bias is further linked to methodological nationalism, where "society" and "nation-state" end up theoretically and empirically equated (Chernilo 2011). For the purposes of this discussion, I extend these issues to studies of single-country and single-region cohort studies.

Greer, Elliott, and Oliver (2015) point out that state-level measures of welfare

effort should decompose into state and regional welfare state effort. If regions are dominant welfare providers and the central state plays little role, then there might be considerable variation. They also point out that, in issues of social risk, there is practically as much difference within a single country (regional) as there is between groups of countries by welfare state type. Municipalities in Norway have a dual role of implementing national policy goals and deciding how to prioritize their funding according to local preferences. This means that they are local decision-makers in their own right and provide the bulk of welfare services, coordinated by local planning in a variety of areas, including public health (Helgesen 2014).

Article III addresses issues of whole-nation bias/methodological nationalism, as it presents a relationship between education and health input measurements, such as antibacterial dispensing rates, that was not previously recognized at the municipal level of analysis. This paper further demonstrates that previous findings on dispensing rate patterns in antibacterials have primarily (though some exceptions exist with analysis on county-level variations) focused on the overall national trend patterns in dispensing rates and on the relative success of national policy guidelines on antibacterial prescription in comparison to other countries. By showing the variation in dispensing rate trends between municipalities over this period, the paper shows what may be missed if we merely consider analysis at the individual or national level. It further shows that the success of national policies for reduction is likely to depend on and vary between subnational contexts. Similarly to Blaser et al. (2021) and Mölter et al. (2018), it argues that antimicrobial stewardship should be considered a subject for multiple levels of government, and how efforts to reduce dispensing rates could avoid penalizing already disadvantaged communities through implementation of area-level strategies.

Article I follows Wilkinson and Pickett (2006) and gains comparativist logic by relating the societal comparison to the national level. However, the empirical investigation is thus unable to discuss variations in the income–health gradient within nation-states. There are two methodological points to consider: first, on what level of analysis the small *N* problem occurs, and second, the usage of comparatively meaningful observations.

The decision to perform this comparative analysis with the nation-state as the second level of analysis boils down to choices regarding the small *N* issue in a multilevel context, that is, the decision between a small number of individuals per group or else a small number of groups. Disaggregating even a large survey, such as the European Social Survey (ESS), to its regional parts means spreading the observations out onto many regional units. While reasonable in many multilevel contexts, mediation analysis specifically suffers from a small number of observations at the level where mediation is performed (MacKinnon,

Fairchild, and Fritz 2007). Where multilevel models suffer, particularly in the cross-level interaction case, is with a small number of group units. The result is that neither analysis would be considered ideal, but the small *N* issue in the comparative literature is thoroughly discussed (Snyder 2001) and gives some confidence to the base analysis performed: the mediation. Further, the regional unit available in the ESS is NUTS.[2] These regions only partially overlap with politico-administrative subnational regions. Regions are primarily classified as units for statistical analysis, based on their population size. The applicability of these regions is thus limited as an observation of "society" as discussed in the literature on methodological nationalism (Greer, Elliott, and Oliver 2015) and, indeed, in the literature on the income inequality–health hypothesis (Pickett and Wilkinson 2015). If used, regional analysis would engage a "fuzzy" definition of to whom people would hypothetically compare themselves without any real definition of the polity. Overall, the national level seemed the more robust choice, despite the trade-off in statistical power in estimating the cross-level interaction term.

In Article II a strictly individual analysis was performed. Data are based on a geographically restricted cohort study; participants were recruited from Tromsø municipality in Northern Norway. Some issues serve as relevant discussions: generalizability, extrapolation, and context. In the article, my co-authors and I at no point relate the findings to factors specifically considered the local context (Tromsø). While such analysis may be reasonable, it would to some extent be at odds with both the other works and represent a shift in the scientific rationale applied in the dissertation. Instead of considering the Tromsø cohort a subject for contextual analysis, we present the analysis in the most generalizable terms by applying a variable-oriented view of NCD prevalence between social groups. From this perspective, analysis of contextual effects would not be possible, as the number of observations is equal to 1, negating the possibility of statistical comparison between places.

The idea of place-effects is not new in research on health inequalities. Often, effects relating to place are distinguished by compositional and contextual effects: the assumption that place-based health inequalities result from the compositional differences of the population (extended individual model) or from features of the area that are not captured by the composition of the population, such as de-industrialization (Bambra 2016; Macintyre and Ellaway 2003). It has been argued that this distinction is somewhat reductionist and that it often treats contextual effects relating to place as a residual category after controlling for individual and place characteristics (Macintyre and Ellaway 2003). Further distinctions can be made where place encapsulates local social

2. Nomenclature of territorial units for statistics, a hierarchical system for dividing up the economic territory of the EU, developed by Eurostat.

relations and shared physical resources (Cummins et al. 2007). More recent efforts have attempted to understand how different aspects of place-based social and environmental exposures tend to get under the skin and directly affect human physiology and health (so-called "embodiment") (Petteway, Mujahid, and Allen 2019), as well as an attempt at merging intersectional thinking to understand how social oppression interacts and interrelates with place (Bambra 2022). Intersectional thinking and frameworks are developing in the study of health inequalities, challenging the idea of a single social hierarchy (Yuval-Davis 2015) and broadening the research agenda to question the situation of specific social groups along with the institutional factors that affect their vulnerability (Gkiouleka et al. 2018). Researchers are starting to include the idea of "place" and other elements of social geography as elements of intersectionality in order to understand how mutually constitutive forms of oppression interact and interrelate with place geography (Bambra 2022). Some researchers argue that in order to do justice to the complexity of health inequalities, one must reject the idea of single-bullet cause and explanations (Eikemo and Øversveen 2019).

The articles in this dissertation remain somewhat ignorant of these later developments. Article III concerns itself with geographical differences in health input and socioeconomic disadvantage, drawing upon both contextual and compositional ideas of what area-level education actually reflects. Article I does address a hypothesis that assumes that income inequality gets under the skin of individuals, but it concludes that it is income, if anything, that gets under the skin given the lack of an intensifying effect of income inequality on the effect of psychosocial stress. Article II with its variable-oriented perspective and local empirical backdrop evades any discussion of contextual and place-based effects. The question then becomes to what extent these results should be considered generalizable to the Norwegian (or global) population, or whether the associations derived from the algorithm significantly differ when compared to other populations. Given the variations in the health outcomes, gradients, and trends shown in the other two papers, extrapolation seems unwarranted. This illustrates some of the limitations of variable-oriented empirical investigations on local communities and may constitute the inverse of the whole-nation bias: a local community bias.

One topic briefly mentioned in Article III is spatial resolution. The modifiable areal unit problem (MAUP) refers to the extent to which inferences change when units of aggregation change. If geographical units are subject to change (i.e., modifiable) and if the units themselves are arbitrary and thus have no intrinsic geographic meaning, then inferences based on these aggregations from non-modifiable entities such as individuals are questionable (King 1997). This is a particular issue of concern in Norway as, over the years, many municipalities have been merged (i.e., *modified*). Mergers likely result in changes

in the proportions and rates employed in the study. While the potential of an MAUP cannot be discarded, this describes the issue in empirical terms. While Haugen et al. (2018) found geographical variation in antibiotic dispensing in Norway to depend on the number of inhabitants at the municipality level, they found no empirical evidence of the modifiable unit problem when aggregating data to different levels of spatial resolution. Addressing the extent to which municipality mergers actually do affect the association between education and trends in antibacterial dispensing rates may be approximated in future research, for instance by constructing synthetic cases (municipalities that *did not exist in the time frame under study*) or by investigating the differences in effect when the new configuration of municipalities are older. This was deemed outside the scope of the paper and thus omitted from its final version. Following this discussion, the idea of the unit of analysis being intrinsically *geographic* or potentially *arbitrary* deserves a short comment. Following King (1997), one may question the idea that a unit must have an intrinsically geographic meaning. From the perspective of political science, it makes more sense to consider aspects of municipalities' geography as *features* of these primarily *politico-administrative* units. There is nothing meaningless about the extent of people and territory governed by a local authority, especially when the authority functions as the provider of primary health care services and administers our dependent variable. That is not to deny the possible existence of an MAUP-type issue. But while the units of analysis are modifiable, they are in no way arbitrary, and neither are their modifications.

4.8 Ethics statement and data sources

Article I collected data from round 7 of the ESS published in 2014. The ESS7 is a cross-country dataset containing 40 185 individual observations from 21 countries. This version includes a rotating module with indicators specifically designed to capture the social inequalities in health within and between countries in Europe. The rotating module allowed construction of a psychosocial stress index for comparative analysis. Article I was not subject to an ethical review nor registered with the Norwegian Centre for Research Data (NSD) as all data sources were secondary, anonymized, and publicly available. Country-level indicators were collected from the Quality of Government standard dataset (Teorell et al. 2021) and the World Inequality Database.

Article II used data from the seventh round of The Tromsø Study (Tromsø7). The Tromsø Study is an ongoing population-based health study in Tromsø, Norway containing data on 21 083 individuals aged 40 years or older. A detailed description of Tromsø7 is available in (Hopstock et al. 2022). Article II was deemed outside the scope of health research and was thus not reviewed

by REK North; however, it was subject to an ethical review by The Tromsø Study Data and Publication Committee (DPU) and registered with the NSD (reference 869500).

Article III used dispensed prescription data from primary care services collected from the Norwegian Prescription Registry (NorPD) and merged these data with official population statistics from Statistics Norway and data on travel time to nearest pharmacy. Details on these sources are available in the article. Article III involves human participants, but the Regional Committees for Medical and Health Research Ethics Norway (2018/1021) exempted this study from informed consent under the Norwegian Health Research Act. Data on prescriptions are retrospective and routinely collected through a national registry (making informed consent difficult), and the project was deemed valuable for the public. Individual prescription information was only used to calculate municipality dispensing rates and volume. The only information used relating to individual patients was their municipality of residence.

4.9 Statistical analyses

Article I uses an extended variation of the classic mediation model formulated by Baron and Kenny (1986) for two reasons. First, the classic mediation model assumes independent observations. In clustered data (in this case; individuals nested within countries), the assumption that observations are independent is violated. Second, the *moderator* (income inequality) can only be observed at an ecological level of analysis (country), while the outcome (self-rated health), exposure (income), and mediator (psychosocial stress) are inherently individual-level variables. The solution was thus to extend the classic 1-1-1 formulation to allow for estimating the moderating effect of country-level income inequality by using the multilevel regression framework (Tofighi, West, and MacKinnon 2013). This was achieved by estimating interaction terms between the moderator, treatment, and mediating variables in the base multilevel models, after which different levels of the moderator at which effects are calculated are set by the researcher (Tingley et al. 2014). In order to keep the presentation somewhat easy to intuit, I report effects at mean, +1 and -1 standard deviations of income inequality. The specific values of income inequality these effects are estimated for can easily be observed using the descriptive statistics also available in the paper. Moderated mediation analyses based on `lmer` objects were fit using the `mediation` package (Tingley et al. 2014). All analyses were conducted in R. To preserve data wherever possible, I used multiple imputation via the expectation-maximization with bootstrapping (EMB) algorithm using the `Amelia` package (Honaker, King, and Blackwell 2011).

Article II uses the random forest algorithm. Random forest estimation grows a large number of decision trees, allowing them to vote for the most popular class in classification problems (Breiman 2001). Ensemble methods such as random forests alleviate high-variance issues of estimating decision trees. Low-variance estimators such as linear models tend to perform well regardless of their permutation though are more sensitive to bias. By applying random forests, we alleviated the issue of high variance among individual decision trees. A major advantage of the random forest approach is its ability to examine nonlinear functional forms and complex interaction terms among covariates without the analyst having to prespecify a particular functional form or interaction term (Hill and Jones 2014; Jones and Lupu 2018) or the need for variable transformation (Kreatsoulas and Subramanian 2018). Variable importance analyses were estimated by the mean decrease in accuracy (MDA) metric. MDA measures the mean decrease in classification performance after permuting each element of the set of predictors X_j, where j indexes each covariate, over all trees in the forest (Hill and Jones 2014). Partial dependences were estimated for education, household income, and occupation. Partial dependence functions represent the effect of a given variable after accounting for the average effects of the other variables (Friedman and Meulman 2003). They represent the functional forms of the relationship between covariates and outcomes. Responses "Yes" and "Yes, previously" were coded 1, and the response "No" was coded 0. The overall sample size was $N = 21083$ participants. We applied simple mean and median imputation to missing values. Outcomes were imbalanced, with a non-NCD outcome being much more common for all NCD outcomes. If ignored, this would result in a naive predictive algorithm. Class imbalances must be considered in classification models to avoid trivial predictions. We achieved balanced outcomes by randomly selecting a subset of observations (with replacement) from both outcomes in the training set for each decision tree. We elected the base rate as the baseline accuracy because of its atheoretical nature; base rate accuracy is the proportion in the majority class. For balanced binary classification problems, the base rate is equal to 50%. A substantive issue with over- and undersampling is the assumption made on the data-generating process. Every tree assumes that the population distribution of the given NCD outcome is equal to 50%, questioning the external validity of our sampling method. Therefore, before training the models and presenting the out-of-bag (OOB) error estimate, we held out 20% of the data as a test set and compared the OOB error with the test error. Random forests were estimated using the R package `randomForest` (Liaw and Wiener 2002). Partial dependences are calculated using the `pdp` (Greenwell 2017) package. All models were estimated on 1000 decision trees. Outcome undersampling on the negative outcome was applied to adjust for class imbalance. Each tree randomly sampled 200 observations from each NCD outcome class.

Article III uses a multilevel growth curve model. Multilevel growth curve mod-

els are a special case of multilevel models in which the coefficient of time varies between units (Rabe-Hesketh and Skrondal 2008). The variation in dispensing rate between units (municipalities) is modeled as a fixed growth trajectory with a variance component, which means that the parameters of growth can be modeled by background characteristics (Raudenbush and Bryk 2002). The municipalities are repeatedly observed, such that level 1 constitutes the longitudinal part of the model and level 2 captures the variance between the municipalities. We centered all level 1 covariates except time on their cluster-means to achieve orthogonality between the level 1 and level 2 variables (Enders and Tofighi 2007). The covariates at level 1 were annual measurements of poverty, education, and municipality population size, which reflect changes in the municipality by year. The same covariates were aggregated at level 2 as cluster-means (CMs). These covariates reflect differences between municipalities over the period under study. All level 2 covariates were conversely centered on their grand mean (CGM). This centering scheme allows for easier interpretation of main effects in the interaction term, in which the estimated trend coefficient is interpreted as the expected mean dispensing rate trend in municipalities at average levels of population education. Time (L1) was not centered because we were interested in the average trend over the period. A discussion on centering time is available in Biesanz et al. (2004). The multilevel growth curve model assumes that time-variant covariates are not characterized by a systematic growth process, and the inclusion of simultaneous growth processes in a multilevel growth curve model may lead to misspecification and biased effects (Curran, Obeidat, and Losardo 2010). Within-municipality variations in education levels are highly correlated with time ($r = .95$), providing evidence for simultaneous growth and biasing the trend coefficient. We therefore removed the time-variant education predictor, as our goal was to estimate a cross-level interaction effect between the time-invariant education predictor and trends. We performed a square root transformation on the dispense rate metric to improve the model fit, but the coefficients on the square root scale lack the clean interpretability of coefficients on the original scale. We therefore used the square root model for predictions and for the evaluation of statistical significance but present the predicted dispensing rates using the original scale to aid in interpretation. Model fit was assessed using the Akaike Information Criterion (AIC), the Bayesian Information Criterion (BIC) and residual diagnostic plots. All models were estimated using the R package `nlme`, incorporating a compound symmetric error covariance structure to deal with within-group autocorrelation.

5

Presentation of the articles

In this chapter, I present the three articles in the dissertation. I highlight the extent to which the articles address and contribute to the literature on the social determinants of health and the mechanisms that generate differences in health outcomes between social groups. I further highlight how novel use of methods of association have contributed to uncovering the novel empirical contributions shown by the articles.

5.1 Is the mediating effect of psychosocial stress on the income–health relationship moderated by income inequality?

This paper investigates the extent to which the income–health gradient is mediated by psychosocial stress at the individual level and whether this gradient is moderated by income inequality. The article primarily discusses the likely causal pathways that link health outcomes to income and income inequality. The paper relates to the main research question by addressing and explaining the role of income in generating health inequalities as well as by addressing how income inequality can contribute to generating social inequalities in health.

The income inequality hypothesis has been around for over 40 years and has

been a foundational area of research for studies of income–health gradients (Rodgers 1979; Wilkinson 1999; Lynch et al. 2000; Marmot and Wilkinson 2001; Pickett and Wilkinson 2015; Olstad et al. 2022). Largely motivated by the seeming inability of economic development to explain differences in life expectancy between high-income countries specifically, some authors turned to investigations into social hierarchy and psychosocial stress as the potential biological link between social status and health outcomes (Marmot 2001; Pickett and Wilkinson 2015). The main argument can be summarized as follows: large income differences intensify social hierarchies and increase class conflict and feelings of relative deprivation (Elgar 2010). Intensified social hierarchies lead to feelings of long-term inferiority. This feeling of inferiority (termed psychosocial stress, social anxiety, or "status syndrome") increases the burden of low social status and inequality and "gets under the skin," thus harming health through neuroendocrine pathways (Marmot and Wilkinson 2001). This assumes that income inequality itself is an initial cause of social class differences in health by income.

Critics were quick to point out that the psychosocial stress theory conflates the structural sources of inequality and the subjective consequences or experiences of inequality. Neo-materialism argues that an effect of income inequality on health has its sources in the material world through differential exposures, a lack of resources held by individuals, and a systematic underinvestment in human and physical capital and social infrastructure. Income inequality is one result of political-economic and historical processes that shape individuals' private resources and the nature of public infrastructure (Lynch et al. 2000). As detailed in the article, evidence for the income inequality hypothesis is mixed.

This article contributes to the literature on the income–health gradient and the income inequality hypothesis via comparative analysis of psychosocial stress modeled as a *mediator*. While multilevel studies have been frequently applied in the study of the income inequality hypothesis, and one recent study applied mediation analysis by country for a variety of indicators (Olstad et al. 2022), no study to date has addressed the hypothesis via multilevel moderated mediation. I argue that this approach gets to the core of the income inequality hypothesis. First, psychosocial stress is experienced as a function of income inequality. The curvilinear nature of the income–health gradient observed in many studies (Kinge et al. 2019) suggests that income effects on health are stronger the further down the distribution one goes. This suggests that whatever the intervening mechanisms, income affects health greatly when there is less of it. Second, in order for income inequality to affect health, there must be an income–health gradient in the first place. Psychosocial stress as an effect primarily makes sense when considered as a mediator, a pathway from income to poor health, because stress responses must be generated by income

for any added effect of income inequality to take place.

The article's empirical contribution is in showing that income inequality does not intensify the extent to which psychosocial stress mediates the income–health relationship, but that psychosocial stress does mediate the effect of income on self-rated health. I show that psychosocial stress is related to individual income, not income inequality. If income inequality has an effect on health, it is thus more likely a result of how income inequality shapes individual control over and access to private and public resources via material pathways. It is precisely this emphasis of income as a *material resource* that ties the article to the overall framework of the dissertation. The effect that income inequality has is limited to how income is distributed and not the extent to which it intensifies the feelings of social hierarchy.

5.2 The predictive importance of education, income, and occupation on non-communicable disease outcomes: results from the Tromsø Study

This paper investigates the extent to which education, household income, and work status contribute to predicting non-communicable disease outcomes in individuals. The article uses algorithmic modeling via random forest estimation. Random forest estimation allows the estimation and interpretation of statistics such as variable importances and partial dependences, allowing a peek into the black box of predictive algorithms. The article discusses *prediction* as a tool for scientific evaluation and its potential usefulness in public health policymaking, with a specific focus on social root causes. The article thus provides several contributions to the literature. First, it represents a step toward including predictive accuracy as a tool for scientific evaluations of health inequalities. It achieves this aim by discussing why predictive accuracy is inherent to the applicability of theoretical frameworks in "the real world." Second, it adds to the established framework of the evaluation of scientific evidence applied in evidence-based policymaking, suggesting that prediction is a potential future tool for exploiting the power of routine data collection.

Empirically, the article aligns with traditional inquiries on socioeconomic investigations into differences in health outcomes. Akin to Olsen, Lindberg, and Lamu (2020) and Mackenbach et al. (2018), it assumes that comparing social class gradients in health, or socioeconomic differences in health between individual predictors, is inherently useful. However, one may consider that ed-

ucation, household income, and occupational status are the constituent parts of social class. A legitimate criticism is thus that comparing gradients within the generalized concept is somewhat moot. Recent contributions to the field of health inequalities do include composite scores of socioeconomic positioning based on income and education specifically (Lindberg et al. 2022). Arguably, these indicators come closer to capturing the concept holistically. However, the article shows that both gradients and contributions to predictions vary *between* education, household income, and occupation categories. This variance would be obscured by a composite index of socioeconomic positioning or social class. For the purposes of the article – extracting the maximum amount of information from the data themselves by using non-parametric algorithmic modeling – it thus makes sense to separate rather than collapse these indicators.

The article's contribution to the literature is found primarily in its emphasis on predictive/algorithmic modeling, in no small part inspired by similar attempts in political science (Jones and Lupu 2018; Hill and Jones 2014; Broderstad 2023). Additionally, it follows Cranmer and Desmarais (2017) in assuming that relying exclusively on novel hypotheses and empirical tests to follow skips an important exploratory observation of nature. However, we follow Kreatsoulas and Subramanian (2018) and amend this statement somewhat, rejecting that probabilistic prediction alone can substitute substantive understanding of health inequality dynamics. We therefore discuss predictive modeling with reference to the arguments of Montez and Friedman (2014) that research should concentrate on conditional causal effects. It is necessary to stress that algorithmic modeling is a tool like other scientific tools and methods of evaluation; it is not designed to replace traditional theory building or methods of causal inference. Rather, it is a tool that can be useful for theory building because it can uncover patterns that are not obvious or intuitive, and it can suggest sets of features to be included in analysis (Cranmer and Desmarais 2017).

What it does not achieve is integrating causal inference with predictive modeling, a strategy that seems meaningful for scientific evaluation and evidence-based policymaking in the future. In other words, the article is committed to observation and statistical association. Machine learning tools are being explored for purposes of estimating average treatment effects in both clinical settings (Fang et al. 2019) and for purposes of policy evaluation (Kreif and DiazOrdaz 2019). Supervised machine learning algorithms have also been used to identify heterogeneous treatment effects in subpopulations in experimental data (Molina and Garip 2019). Additional future directions include both developing complex deep learning models to model high level nonlinearity and identifying which interventions on risk factors for poor health outcomes yield a higher probability of success (Wiemken and Kelley 2019).

However, the article demonstrates that predictive modeling even in simple

applications such as this one – applied to glean social gradients in health from the data themselves – is inherently useful, especially in theoretical contexts where most theories predict similar outcomes: low-to-high social gradients in health. Thus, its empirical contribution lies in exploring the variation in predictive importance of the different root causes of health outcomes. The article's methodological contribution lies in assessing these using test and training sets to test their out-of-sample applicability and in unpacking *how* the model makes its predictions using partial dependences. Extending the methodological toolbox and methods of evaluation with algorithmic modeling may alleviate some issues with categorical interpretations of the "p-value" under the "null hypothesis paradigm" (McShane et al. 2019; Amrhein, Greenland, and McShane 2019) and issues of false positives in traditional hypothesis testing in big data contexts (Veronesi et al. 2020). It may at least provide some nuance to our conclusions.

The article sketches out potential ways in which out-of-sample predictive modeling may aid in future evidence-based policymaking (particularly in merging this predictive strategy with causal inferences). However, the potential of predictive modeling is constrained by current policy on health research and on how data should or can be used and, perhaps most importantly, by a general understanding of what constitutes *evidence* in the first place. The article is thus an attempt to clarify and deepen the traditional methodological toolbox, both from a scientific perspective and for the potential for future evidence-based policymaking on health inequalities. It is therefore also an argument for methodological pluralism.

5.3 Association of area-level education with the regional growth trajectories of rates of antibacterial dispensing to patients under 3 years in Norway: a longitudinal retrospective study

This paper investigates the extent to which municipal-level population education levels are associated with municipal-level growth trajectories of rates of antibacterial dispensing to pediatric patients in Norway. It achieves this aim by estimating a multilevel growth curve model on dispensing rate data between 2006 and 2016. A coefficient of time is allowed to vary between municipalities. This allows estimating a cross-level interaction term between area-level education over the time period and the coefficient of time. Here, area-level education is primarily considered a representation of place-based socioeconomic disad-

vantage, insofar as population levels of education reflect both the composition of a population in terms of socioeconomic status but also the more contextual conditions of life in a socioeconomically disadvantaged area.

In terms of health inequality, several studies have identified an association between the high use of antibacterials in young children and an increased risk of chronic disease development later in life (Beckstrøm and Småbrekke 2021; Korpela and De Vos 2016; Mårild et al. 2013; Risnes et al. 2011; Sander et al. 2019; Blaser 2016). Antibacterial dispensing rates have further been associated with socioeconomic characteristics at the individual (Thrane et al. 2003) and subnational (Koller et al. 2013) levels of analysis. Optimizing prescribing practices thus seems important for reducing health inequalities in future generations. The article further argues that, if all differences in antibacterial dispensing rates were explained merely by the severity and density of infections, the implication would be that infections requiring antibacterial treatment are geographically unequally distributed, even between pediatric patients.

The article explicitly addresses issues of whole-nation bias (Rokkan 1970) and methodological nationalism (Chernilo 2011; Greer, Elliott, and Oliver 2015) and embraces the subnational comparative method (Snyder 2001) in studies of antibacterial dispensing rates and health care input. This can primarily be seen in arguments that most studies on antibacterial dispensing rates in Norway have either addressed subnational variation in dispensing rates at the county level or discussed overall dispensing rate trends at the national level in a comparative perspective. No study prior to this one has attempted to statistically explain the differences in dispensing rate trends at the local level in Norway.

The paper is characterized by a somewhat broad approach to micro- and macro-level arguments in the literature. While the empirical investigation cannot infer directly on individual-level mechanisms, it discusses likely individual-level causal processes that may contribute to differences in dispensing rate trends in the aggregate: notably, the patient–provider relationship modified by socioeconomic status, health and public health literacy, and the potential biasing effects of disease burden in parental health care seeking. At the macro level, the article discusses lack of parental education at the community level as a source of relative socioeconomic deprivation or advantage between communities as well as the potential usefulness of area-level strategies for antimicrobial stewardship.

I find it here important to note that these micro- and macro-level arguments, while cited from the literature on antibacterial dispensing specifically, are arguments that can be found in the general literature on the determinants of health

and health inequality – for instance, those relating to health literacy (Berkman, Davis, and McCormack 2010), public health literacy (Freedman et al. 2009), and collective action, socioeconomic differentiation in health care usage (Vikum et al. 2013; Vikum, Krokstad, and Westin 2012), and those that relate socioeconomic deprivation to population health outcomes between aggregate units in general. My main point here is that while the paper aligns with the health inequality literature through the potential pathway of early life health input to adult health outcomes, this alignment extends into the general assumptions made within the social determinants of health perspective when populations are compared. Thus, while the paper emphasizes particular mechanisms and dynamics related to (aggregate) socioeconomic advantage and disadvantage that may lead to suboptimal dispensing rate trends, the cover article treats antibacterial dispensing rates as "just another" health outcome.

Relating to the overall literature on health inequality, the paper identifies reduction and optimization in prescribing practices as one potential future pathway for reducing health inequalities in future generations. As a result, it interacts with the life course perspective on health inequalities, suggesting that early life health input and (parental) socioeconomic positioning may impact later health outcomes and that these outcomes are likely stratified across generations and between areas. In order to avoid an ecological fallacy, I find it necessary to stress that the article cannot infer on these individual-level processes directly. Rather, based in what is already established in this field, these processes are likely to translate to and interact with aggregate levels of analysis.

/6

Conclusions

This dissertation set out to explore the persistence of health inequalities in modern, high-income countries such as Norway and to provide insight into contributing mechanisms to health being affected by socioeconomic advantage and disadvantage. It applied data from a variety of contexts, at several levels of analysis, and included both within- and between-country analysis. The individual articles aimed at establishing the extent to which individual resources derived from socioeconomic positioning in meritocratic systems predict health outcomes in a broad manner. The extended introduction generates a holistic framework for the study of the social determinants of health, describes the policy space in which health inequalities are generated, and applies a logic of negation to define the concept of health inequalities.

The health inequality challenge has been studied from a plethora of perspectives, disciplines, and methodological perspectives. Much is already known when it comes to the extent of health inequalities in high-income welfare states such as Norway. The exact mechanisms that generate and perpetuate health inequalities, however, have been difficult to pin down. The issue of health inequalities is complex because these inequalities may be generated at all stages of the life course.

6.1 Contributions

The articles in the dissertation inform the mechanisms of how social resources may influence health outcomes in several ways. Article I details the extent to which psychosocial stress mediates the effect of income on self-rated health as well as investigates the extent to which these effects are moderated by income inequality. Further, Article I shows that psychosocial stress mediates the effect of income on self-rated health even in countries with comparatively low levels of income inequality, such as Norway. The most important contribution in this regard is acknowledging that there is analytical value in conceptualizing psychosocial stress as a *mediator*: a biological mechanism resulting from the extent of social or material resources at the individual level and not itself a *root cause* of said health outcomes. This is an important distinction because the psychosocial environment hypothesis shifts attention away from the consequences of social inequality for individual health outcomes without providing much guidance on what levels of income inequality could potentially flatten the effects of the status syndrome. Conceptualizing psychosocial stress as a mediator emphasizes its biological mechanisms but yields ultimate causal status to material well-being. In line with Kawachi, Subramanian, and Almeida-Filho (2002), I conclude that the apparent tension between (neo)-materialist and psychosocial environment perspectives on the effects of income inequality somewhat disappears when psychosocial stress is considered a mediator.

The application of multilevel moderated mediation is particularly effective at partialling out the correlation from income, psychosocial stress, and income inequality on health outcomes. It thus aims to address the extent to which income inequality intensifies the effect of status hierarchies, which is one of the main sources of divide in the academic literature on the income inequality–health hypothesis. To be sure, comparative strategies have been applied before (Olstad et al. 2022; Layte et al. 2019), and multilevel studies have been a point of discussion in this literature, but merging these ideas with the mediation framework in a formal model is novel. Even with its limitations, I believe it shows that studies of association may still contribute to our empirical investigations, even on topics that span more than 40 years of empirical investigations, thus making a decent argument *for* methodological pluralism in research on the social inequalities in health.

In addition to this methodical novelty, I generated an index of psychosocial stress based on the concept as proposed by Marmot and Wilkinson (2001) – an indicator that proved difficult to locate in cross-national survey data. As such, I believe this to be a contribution to the literature and to the measurement of psychosocial stress employed in a context where comparative analysis can be performed. The indicator is a contribution to the quantitative comparative public health literature that may be applicable for future research endeavors

based on the income inequality–health hypothesis.

The contributions that Article II makes to the literature are primarily empirical and methodological. Machine learning tools are still in their infancy in the social epidemiologic space (Kreatsoulas and Subramanian 2018; Wiemken and Kelley 2019). We expand on previous efforts (Seligman, Tuljapurkar, and Rehkopf 2018) by attempting to peek into the predictive black box (i.e., describing *how* the model makes its predictions). Findings from Article II suggest that material resources are not the only resources at play, as education also shows low-to-high gradients in its predictions of diabetes, stroke, and heart attack prevalence. Education and occupation, however, both predict positive gradients for cancers, while income does not. Education consistently contributes to predictive performance in all NCD outcomes under study. Occupational status, while contributing to the predictive performance of the random forest model for stroke and diabetes, shows comparatively flat gradients in stroke, diabetes, and heart attack, at least compared to household income and education. It shows the unique benefits of discovery that non-parametric algorithms are capable of, and it shows how model performance can be evaluated within the train–test framework and out-of-sample predictions. The extended introduction expands on some of the arguments presented in the article and argues that predictive modeling allows studying population heterogeneity, contributes to reducing issues of model dependence, and ultimately aids in providing consistent evidence for policymakers to consider. The article is therefore ultimately an argument *for* methodological pluralism in health inequality research as well.

Article III contributes to the literature on the social determinants of early life health care input by investigating population trends and patterns in antibacterial dispensing rates. It fills a gap in the literature by investigating the variation in local growth trajectories in dispensing rates and by connecting this variation in trend to sociodemographics via local population education levels. It shows that local variations are substantial, even in a country that (in the European context) shows comparatively low dispensing rates. By showing the local variations in dispensing rate trends, it addresses some of the potential challenges that authorities face in large-scale, national public health initiatives (e.g., express aims at reducing antibiotics consumption), such as issues of coordination between multiple levels of government. It further connects some of these challenges to ideas of socioeconomic disadvantage in a geographical sense and suggests that area-level strategies may be necessary in order to optimize prescribing practices. By exploring these local variations, it contributes to discussions on the meaning of contextual and compositional effects in studies on population health care input.

6.2 Limitations and future research

There are of course several limitations that should be addressed in this dissertation. As discussed in Chapter 4, studies on observational data need pseudo- or natural experiment designs in order to estimate causal *effects*. While I have cited the novel application of methods of association as a contribution to the literature, the inability to estimate causal effects remains a limitation of the methodological approach as a whole. This is a limitation that is inherent when conceptualizing causation in terms of *robust dependence* as opposed to *counterfactuals*. This implies a way forward for the research agenda on health inequalities, one where conditions under which these social resources (either at the individual level or in the aggregate) may have a causal effect (Montez and Friedman 2014), by including machine learning to investigate potential treatment heterogeneity in health inequality contexts.

A further limitation is the somewhat single-point definition of social inequalities in health applied. I have allowed some leniency regarding the understanding of social class/socioeconomic status when discussing empirical investigations in the aggregate (geographical inequalities), yet the important point remains that analyses are restricted to those inequalities that are traditionally associated with Weberian stratification: income, education, and occupational class. As such, I have limited the concept of health inequalities to *specific* groups, but there are several other groups that yield relevant representations of the social inequalities in health concept. For instance, inequality between genders and ethnicities and how these intersect with socioeconomic positioning or class in generating health outcomes are not discussed nor investigated. The empirical analyses in this dissertation are blind to these inequalities. Therefore, future research may consider investigating a variety of systems of social stratification that interact with multiple layers of disadvantage by incorporating intersectional frameworks (Eikemo and Øversveen 2019; Gkiouleka et al. 2018; Bambra 2022). However, one should keep the ladder of abstraction in mind to avoid conflating general categories with distinct social and historical backgrounds.

Additionally, a limitation in this dissertation lies in how it embraces the overarching perspective of social determinants of health. The presentation in Chapter 2 clearly shows that there are different perspectives on the direction of causality; important perspectives and empirical evidence support this claim. Committing to the social determinants of health perspective effectively blinds the researcher (me) from even considering embarking on empirical investigations that recognize the reverse or bidirectional relationship between socioeconomic positions and health outcomes. Clearly, the social determinants of health perspective is *not the only valid* perspective. The framework applied in this dissertation is rigid in the sense that it always places health outcomes on the left side of the equation as something to be explained, rather than explaining the ex-

tent to which ill-health may impair the acquisition of social resources. This approach is not without its benefits, however, as it provided a somewhat consistent guide for formulating hypotheses and conducting empirical investigations throughout.

6.3 Concluding remarks

Taken together, the main argument of this dissertation is that both material and immaterial resources and their distribution resulting from stratification and meritocratic reward systems remain important determinants of health outcomes, even in largely preventable ill-health outcomes and in high-income and egalitarian welfare contexts such as Norway. Early life health care input may have far-reaching consequences (positive and negative) into adult life, and these may themselves be structured in a similar fashion. I have argued that policies aiming to reduce social inequalities in health need to commit to addressing the effect of resources to avoid the lifestyle drift phenomenon. The effects of income on health suggest that material resources remain an important root cause of health; however, this effect is likely mediated by psychosocial stress. Thus, the income–health hypothesis is likely supported by a biological mechanism, but the income inequality–health hypothesis is not. Lastly, I have shown that the extent to which socioeconomic status indicators predict health outcomes varies depending on the chosen measurement for both health outcomes and socioeconomic status. I have argued that predictive modeling in combination with traditional data modeling and causal inference techniques can provide both valuable information for policymakers and new methods for scientific evaluation by reducing model dependence and allowing for novel explorations and discovery.

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Part II

Research articles

Article I

Sigbjørn Svalestuen (2022). "Is the mediating effect of psychosocial stress on the income–health relationship moderated by income inequality?" *SSM - Population Health* 20, p. 101302. ISSN: 2352-8273. DOI: https://doi.org/10.1016/j.ssmph.2022.101302. URL: https://www.sciencedirect.com/science/article/pii/S2352827322002816

SSM - Population Health 20 (2022) 101302

Contents lists available at ScienceDirect

SSM - Population Health

journal homepage: www.elsevier.com/locate/ssmph

Is the mediating effect of psychosocial stress on the income–health relationship moderated by income inequality?

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ARTICLE INFO

Keywords:
Income inequality
Income
Health
Psychosocial stress
Material deprivation
Multilevel moderated mediation

ABSTRACT

Background: There now exists a rich body of literature on the relationship between income, income inequality, and health. The discussion about the impact of income and income inequality on health includes psychosocial mechanisms, such as long-term perceptions of inferiority and social positioning, material advantage from income, and the structural conditions that define what people can do with their material resources.
Aims: This study investigated the extent to which income's effects on health are mediated by psychosocial stress, and to what extent those effects are moderated by country-level income inequality and economic development.
Methods: Data were collected from The European Social Survey, round 7. Multilevel moderated mediation analysis was applied to estimate the extent of psychosocial stress mediation of the effects of income on self-rated health. Moderated parameters were estimated over country-level income inequality and economic development.
Results: Significant full or partial meditation by psychosocial stress was found in all 20 countries studied. Effects moderated by income inequality and GDP per capita showed expected relationships but failed to reach conventional levels of statistical significance.
Conclusions: Individual-level income remains important for explaining the income–health gradient in self-rated health in Europe. The income–health relationship and the extent to which it is mediated by psychosocial stress varies among countries but is not significantly moderated by contextual income or income inequality. Policies should be aimed at allowing a greater proportion of people to live in material comfort and reduced sense of financial precarity, and protecting individuals from harmful consequences of low income.

1. Introduction

Income has long been of interest to health and health inequality researchers. Studies have suggested that health gaps tend to be wider between individuals in the lower to middle parts of income distribution. It has also been shown that the annual life expectancy is increasing for the highest income quartile, while for the lowest income quartile, life expectancy has stagnated (Kinge et al., 2019). Moreover, the income–health gradient for self-rated health is steeper than the education–health and occupation–health gradients (Olsen et al., 2020). The nonlinear relationship between income and self-rated health suggests that whatever mechanisms explain these inequalities, their effects are stronger among those living on a very low income (Mackenbach, 2019; Mackenbach et al., 2005). While empirical evidence for the income–health gradient is well documented, authors disagree on causal mechanisms; that is whether the income–health gradient is socially determined (Gravelle, 1998; Lynch et al., 2000; Wilkinson, 1999), that ill-health generates income inequality through health selection (O'Donnell, Doorslaer, & Van Ourti, 2015; García-Gómez, 2011), or that the flow of causation is bi-directional over the life-course (Hoffmann et al., 2018; Rehnberg et al., 2021).

Further disagreements on the causal processes linking income to health can be made within the social determinants of health perspective. Materialists argue that the aggregate relationship between income and population health is an artifact of the individual level income–health gradient (Gravelle, 1998). Neo-materialists (Lynch et al. 2000, 2004) argue that income at both the individual and society levels fundamentally reflect the detrimental effects of living in poor material conditions combined with politico-economic processes that govern private resources and public welfare systems. Psychosocial stress theorists (Marmot, 2001; Pickett & Wilkinson, 2015; Wilkinson, 1999) argue that income inequality itself is the issue; relative positioning in the income hierarchy can generate long-term biological stress responses with detrimental health effects.

This study applied multilevel mediation modeling to investigate the effects of individual income on self-rated health. It examined the extent

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https://doi.org/10.1016/j.ssmph.2022.101302
Received 29 August 2022; Received in revised form 21 November 2022; Accepted 23 November 2022
Available online 29 November 2022

to which psychosocial stress mediates those effects. Also examined was the extent to which the direct effect of income and the mediated effect of psychosocial stress are moderated by country-level income and income inequality.

2. Income, income inequality, and health

Studies on the effect of income inequality on health can be traced back to 1979. Rodgers (1979) conducted a cross-sectional international analysis on the association between the Gini coefficient and national mortality statistics. At the individual level, it is widely accepted that higher incomes and other socioeconomic characteristics are associated with many indicators of health (Lynch et al., 2004; O'Donnell et al., 2015). This association presents the shape of a gradient in even the wealthiest of countries (Olsen et al., 2020).

However, the empirical relationship between income inequality and population health is contested. Two important reviews (Lynch et al., 2004; Wilkinson & Pickett, 2006) published in the early 2000s serve as useful illustrations. Lynch et al. (2004) concluded that income inequality in affluent countries is not associated with population health differences as a general phenomenon. It was cited that most of the negative or mixed findings were conducted post-1995, presumably using better quality data. Some studies were characterized as showing mixed results due to findings that were inconsistent between population age groups and a priori predictions of the income inequality hypothesis (IIH). The researchers also noted that multilevel studies found no significant associations after controlling for within-country individual factors and sensitivity to country inclusion in the sample. They found stronger evidence for the IIH in studies using aggregate regional and state-level data from the United States. Again, multilevel studies presented less support. Furthermore, both aggregate and multilevel evidence suggested little or no effect of income inequality in a number of other rich countries.

Wilkinson and Pickett (2006) concluded that 70% of the analyzed papers were wholly supportive of the IIH. The researchers argued that null findings can primarily be explained by the size and type of the analyzed area; analyses of parishes, counties, and census tracts tended to yield unsupportive findings more frequently than country and regional data. Further, results were sensitive to control variable selection. While their perspective generally related to the psychosocial links between income inequality and health outcomes, they did not ignore material factors in their analysis. They argued that the social structure is built substantially on material foundations. The core of the argument is thus that materialism matters, but the link between income inequality and health is not completely explained by material factors. The psychosocial perspective they proposed provides a new path to health outcomes through the negative effects of social comparison.

Wagstaff and Van Doorslaer (2000) explained the divergent findings to some extent. They argued that data from aggregate-level studies are insufficient for discriminating between competing hypotheses. They reviewed evidence for the absolute-income hypothesis, the relative-income hypothesis, the IIH, and the deprivation hypothesis. Out of the four, they found strong support only for the absolute-income hypothesis. They concluded that income inequality only affects population health due to its effects on the poor. They found no convincing support of the relative-income hypothesis whatsoever. They further noted that eight out of nine hypotheses will predict an association between average health and income inequality. Observing this effect empirically will not distinguish between the proposed explanations for the prediction. The same is true for average income with the same eight hypotheses. They concluded that research on these hypotheses up to the 2000s had been incapable of shedding any light on relative income and income inequality affecting individual health. Moreover, the individual studies that were feasibly able to do so showed less than compelling results for the relative-income hypothesis and the IIH.

Beckfield (2004) found that the relationship between health and inequality disappeared in fixed-effects models that addressed unobserved heterogeneity. Mellor and Milyo (2002) argued that previous findings of an association between income inequality and health are partly the product of an ecological fallacy and the failure to control for individual covariates, year effects, and geographic characteristics. Kragten and Rözer (2017) found that while OLS and multilevel models yielded a positive association between income inequality and health, fixed-effects models and analyses of sub-groups associated income inequality with poor health. Torre and Myrskylä (2014) found increases in age- and gender-specific mortality rates where there were increases in income inequality even when controlling for shared period factors and country fixed effects. The strongest effects were observed for children and young-to-middle-aged men. Curran and Mahutga (2018) applied fixed-effects modeling to compare differential effects of income inequality between countries with varying levels of economic development. The results showed a larger effect of income inequality in poorer countries. Similarly, Oorschot (2013) found that while the IIH was supported in low- and middle-income countries, there was no significant relationship between life expectancy and income inequality in high-income countries. They argued that, to some extent, a high level of economic development tempers the potential negative effects of income inequality due to the population's command over essential public goods and services (and more of them). However, they also found that the relationship between levels of income inequality and life expectancy was not robust over time. They also found that the level of economic development moderated the effects of the level of wealth on life expectancy.

Doorslaer and Koolman (2004) found that income contributed to health inequality. However, there were significant variations between European countries in how much health inequality could reasonably be attributed to income differentials. While they found that health inequality was positively correlated with income inequality per se, it was a weaker link than in previous research. Gugushvili et al. (2020) found that perceived changes in income inequality affected self-reported health, as opposed to a direct effect of income inequality. Their work expanded on the psychosocial mechanism because they concerned themselves with how people see and feel inequality in their everyday lives. McFarland, Hill and Montez (2022) found that the association between income inequality and life expectancy in the United States was moderated by state-level policy liberalism. Layte et al. (2019), using data from five cohort studies from four European countries, found higher levels of inflammation and greater differentials in inflammation by socioeconomic positioning in countries with comparatively high levels of income inequality.

In a meta-analysis, Ngamaba et al. (2018) found that subjective well-being and income inequality were only significantly associated in developing countries. Maynou et al. (2015) investigated spatiotemporal processes of regional health convergence and found that convergence rates varied significantly. A recent panel data analysis of 26 European countries for the period 1995 to 2004 found no evidence of a relationship between life expectancy at birth and income inequality (Blázquez-Fernández et al., 2018). Olstad et al. (2022) compared the extent to which psychosocial stress mediates the effect of subjective social status, perceived income adequacy, and educational attainment on self-rated overall health between four countries at varying levels of income inequality. They found no evidence for psychosocial stress being a more important mediator of the association between subjective social status and self-rated overall health in more unequal societies.

One systematic review concluded that area-level income inequality was associated with poorer mental health Tibber et al., 2022 in spite of several methodological limitations in the studies. Sommet et al. (2018) found that income inequality and psychological health are linked, but only for people experiencing financial scarcity. Further, in a systematic review of income inequality and depression, Patel et al. (2018) found that around two-thirds of the 26 reviewed studies supported a link between income inequality and risk of depression.

Pickett and Wilkinson (2015) re-reviewed the literature with explicit consideration given to the potential causal relationship between income inequality and health. They found that the body of evidence to date indicated a strong causal connection due to satisfying the major epidemiological criteria for causality: temporality, plausibility, consistency, and a lack of alternative explanations. Further, they argued that null findings can be explained by inappropriate scales of measurement, mediating variables being used as controls/confounders, use of subjective measurements of health, and short follow-up periods. While their review did not explicitly address the causal mechanisms (focusing instead on methodological criteria for evaluating cause-and-effect), they persisted in the most parsimonious explanation for these effects being social class accentuation and status differentiation. They noted that future studies should make explicit attempts to clarify the causal nature of the empirical relationship.

Another review evaluated the research by distinguishing research efforts that were based on longitudinal, panel, and cross-sectional data (Truesdale & Jencks, 2016). Overall, the only relatively strong relationship identified was between income inequality and social inequalities in life expectancy in single country time series. This suggests that the relationship is weak in cross-sectional and panel data analyses. The evidence for a relationship between average life expectancy and income inequality were considered weak in time series and panel data evidence and is merely moderate in the cross-sectional context.

Findings on the empirical relationship between income inequality and health are mixed. Diverging conclusions can be explained in part by the methodology used (e.g., criteria for support/no-support) and differences in framing (e.g., "evidence for a causal claim" and "averages and disparities"). However, these reviews show that the effects and theoretical pathways of income inequality on health are still under discussion more than 40 years after Rodgers (1979).

2.1. Psychosocial stress and environment: mechanisms

Wilkinson (1994) argued that as societies progress through epidemiological transitions—shifting from infectious diseases as the main causes of death to degenerative cardiovascular diseases and cancers—the mechanisms explaining income gradients in health transition as well. Within-country income gradients in mortality remained, but gross domestic/national product (GDP/GNP) per capita as a predictor of between-country mortality underperformed as explanans in states with long life expectancies. Rather, country-level income inequality showed a more robust association with life expectancy in wealthier countries. Although the impact of psychosocial factors on health had previously been discussed, Wilkinson expanded and suggested that health outcomes are "less a matter of the immediate physical effects of inferior material conditions than of the social meanings attached to those conditions and how people feel about their circumstances and about themselves." Proponents of the relative deprivation argument cite the fact that there is an income gradient in health outcomes rather than a difference explained by poverty alone. They also note that mortality disadvantages remain even with rising real incomes and that living standards among the poorest are much higher than before.

Early formulations of psychosocial theory argued that the social environment could alter host susceptibility to pathogenic agents by affecting neuroendocrine function (Cassel, 1976). Future studies carried these ideas forward, as psychosocial frameworks typically direct attention to endogenous biological responses to human interactions (Krieger, 2001). Long-term feelings of subordination or inferiority are expected to stimulate chronic stress responses that have consequences for physical and mental health (Bambra, 2011). Psychosocial variables like feelings of control, anxiety, insecurity, depression, and social affiliation have been cited as successfully explaining the health gradient. These stimulations may have an effect on health either directly or indirectly. Directly could be through the influence of social relations on neuroendocrine pathways to disease (such as chronic stress leading to wear and tear on the body and mind; allostatic load), and indirectly through stress-related behaviors, such as smoking (Marmot, 2001; Wilkinson, 1994, 1999).

The theoretical perspectives of psychosocial stress emphasize social integration. Inequality produces disintegration and individualism, which undermine the potential beneficial health effects of social support. This links the psychosocial stress hypothesis to the concept of social capital (Putnam, 2000). Also linked is the notion that generalized trust and social cohesion are conditions for a number of factors associated with well-functioning societies (Uslaner & Mitchell, 2005). Social capital, cohesion, and trust generate social support through friendships and social networks. This effect has been argued to be as protective for health as smoking is deleterious (Pickett & Wilkinson, 2015). However, where there is great inequality, there also tends to be underinvestment in the various forms of soft capital, such as education and medical services. Overlapping with Wilkinson empirically and theoretically, these factors have typically been used in materialist arguments (Beckfield, 2004).

2.2. Neo-materialism: mechanisms

The psychosocial environment as the missing link for explaining the non-relationship between GDP per capita and mortality in high-income countries was criticized by Lynch et al. (2000). They argued that the selection of high-income countries was too restrictive and found a stronger relationship when the sample size was extended to include countries outside of the OECD. More importantly, they disagreed about the underlying mechanisms linking income inequality to mortality statistics. They argued that income inequality does not reflect feelings of inferiority and the perception of place in a social hierarchy based on relative position according to income. Instead, they stated that income inequality is one of many manifestations of historical, cultural, and political-economic processes that influence the private resources available to individuals and shapes the nature of public infrastructure. While the psychosocial environment hypothesis assumes universal associations (due to persistent perceptions of relative position regardless of actual living conditions), the neo-materialist view assumes contextual processes. The criticism is partially based on the practical implications of dealing with health inequality under psychosocial theories and goes so far as to argue that the psychosocial environment hypothesis implies mass psychotherapy to alter perceptions of relative disadvantage. Neo-materialist explanations argue that the income–health gradient exists because of a combination of the material possibilities of individual income and the conditions that govern what income enables. Despite the redistributional and decommodifying efforts of the welfare state through cash transfers, taxation, and benefits, there still exist substantial inequalities in material advantage across the globe (Mackenbach, 2012). Income gives access to goods and services and limits exposures to physical and psychosocial risk factors. Neo-materialism gives primacy to structure when explaining health outcomes and health inequality. Individual agency is limited, and public policy and services create the pattern of social inequality (Bambra, 2011).

2.3. Expectations

Psychosocial stress is understood as one possible pathway at the individual level by which income may impact health (Kawachi et al., 2002; Wilkinson, 1999). Income may affect health more directly if material conditions are strained (Gravelle, 1998; Lynch et al., 2000). Psychosocial stress may fully or partially mediate the effect of income on health, leading to the following expectations:

- **H1:** *Psychosocial stress significantly mediates the relationship between individual income and health outcomes.*
- **H2:** *Income has a significant direct effect on health outcomes at the individual level.*

The IIH assumes that large income differences intensify social hierarchies and class conflict, as well as increase feelings of relative deprivation (Elgar, 2010), thus intensifying the effect of the "status syndrome". Further, material conditions are expected to worsen overall in the countries with low economic development:

- **H3:** *The mediating effect of psychosocial stress and the direct effect of income are significantly moderated by income inequality.*
- **H4:** *The mediating effect of psychosocial stress and the direct effect of income are significantly moderated by economic development.*

3. Statistical analysis

As the classic mediation model (Baron & Kenny, 1986) assumes independent observations, multilevel mediation analysis should be applied in contexts of clustered data to account for bias in standard errors due to a lack of independence in observations (Tofighi & Thoemmes, 2014). This is the case for the European Social Survey (ESS). Two hypotheses assume that the mediated and direct effects from the multilevel mediation model are moderated by country-level income inequality (**H3**) or economic development (**H4**). The 1-1-1 multilevel mediation framework is therefore extended by including country-level moderators to predict random (income) slopes (Tofighi et al., 2013). This is achieved by including interaction terms between the moderator, treatment, and mediating variables. Once the base models are fitted, different levels of the moderator at which effects will be calculated are set by the researcher (Tingley et al., 2014). Coefficients and 95% bootstrap confidence intervals are calculated for mean and one standard deviation in levels of income inequality and economic development, respectively.

Missing values were addressed by multiple imputation using the expectation-maximization with bootstrapping (EMB) algorithm using the Amelia package (Honaker et al., 2011). Final results were combined over separate estimations from $m = 5$ imputed datasets. Household income data were unavailable from Estonia. Estonia was therefore omitted from the final sample. Results from models using listwise deletion are available in figure B1a and B1b in the appendix. Base multilevel models were fit using the lme4 package (Bates, 2010). Moderated mediation analysis based on lmer objects were fit using the mediation package (Tingley et al., 2014). All analyses were conducted in R.

4. Data

Individual level variables were collected from the seventh round of the ESS (ESS, 2014). This round was selected because it is the only round to date containing a module on social inequalities in health in Europe.

Self-rated health was measured using the single item "How is your health in general? Would you say it is …" completed on a five-point scale with answers ranging from "very bad" to "very good.". Self-rated health has been applied in health and health inequality research both as a single item measurement (Beckfield et al., 2013; DeSalvo et al., 2006; Lorem et al., 2020) and a multi-item composite indicator (Olsen et al., 2020). Self-rated health has been shown to predict other health outcomes such as mortality risk (Lorem et al., 2020). Self-rated health was selected because it reflects interlinked social, psychological, and biological processes (Balaj, 2020) and should be an responsive indicator to perceptions of ones position in the income gradient and the potential effect of income inequality.

The ESS measures income by giving respondents a showcard with ten income brackets in the local currency and ask respondents to place their households total net income in one of the brackets. While the categories on the scorecard are intended to represent household income deciles, deviations from the expected uniform distribution in many countries warrants some caution in interpreting the income measure as such. Rather, the income measurement should be interpreted as an individuals position on their countries socioeconomic ladder (Donnelly & Pop-Eleches, 2018).

Marmot and Wilkinson (2001) define feelings of control, anxiety, insecurity, depression, and social affiliation as psychosocial indicators. The ESS7 contains a selection of items related to these dimensions, of which 14 items were selected for constructing the index. An overview of the components is available in Table 1. Insecurity and feelings of control were captured by indicators of autonomy at work and feelings about the household income. Depression and stress-related symptoms were captured by indicators of happiness and sadness, self-reported depression, sleep quality, and feelings of lethargy. Social affiliation was captured using indicators of how often a respondent meets friends and participates in social activities, self-reported number of intimate relationships, and feelings of loneliness.

The majority of items were measured using a four-point scale ranging from "None or almost none of the time" to "All or almost all of the time". Autonomy at work and influence over work policy were measured on an eleven-point scale. Respondents were provided seven-point scales to determine how often they meet friends and their number of intimate relationships. A five-point scale distinguished their frequency in social activities as compared to others. These items were collapsed to comply with the four-point scale applied in all other items. Items were inverted where necessary to conform to low-to-high directionality in the psychosocial stress measurement prior to final calculation. Finally, the psychosocial stress index was created using the arithmetic mean, giving all items equal weight. A complete schematic of component transformation is available in table C1 in the appendix.

Education is often used as a measurement of socioeconomic status alongside income (Olsen et al., 2020). However, education is also an important determinant of income (Lahelma, 2001) and research has suggested some reporting heterogeneity in self-rated health between educational groups (Balaj, 2020). There are theorized mechanisms linking education to health through alternate pathways; such as individual cognition or early-life socioeconomic circumstances (Lindberg et al., 2022). Controlling for education serves to parse this variance from the income indicator.

Co-habitation with a partner was included as the income indicator measures household as opposed to individual income. Controlling for co-habitation with a partner thus serves to partial out the income differentials reported from combined incomes.

Gender was included as a control, as gender differences in the proportion of people reporting poor or very poor health in the ESS7 have been observed (Balaj et al., 2017). Age was included as a control as the income–health gradient and its mechanisms may vary over different stages of the life course (Hoffmann et al., 2018; O'Donnell et al., 2015; Rehnberg et al., 2021).

Country-level indicators were collected from the Quality of Government standard dataset (Teorell et al., 2021) and the World Inequality Database. Country level income is measured as GDP per capita. Income inequality is captured by the Gini coefficient in the main model. Following De Maio and Fernando (2007) and Pickett and Wilkinson (2015), top-and-bottom sensitive income inequality indicators were included for sensitivity purposes. Summary statistics are provided in Table 1.

5. Results

Results from multilevel mediation modeling are presented by country in Table 2 and Fig. 1. Results from moderated mediation models are presented in Figs. 2 and 3. Results from models using the top 10%, top 1%, and bottom 50% income share as indicators for income inequality are available in appendix A.

The average causal mediation effect (ACME) of psychosocial stress varied from 0.017 (Norway) to 0.061 (Hungary). The mediating effect of psychosocial stress on the income–health relationship is clearly significant in all countries. The specific mediation process only becomes clear in relation to the size and significance of the direct effect of income.

Table 1
Summary statistics prior to EMB imputation. Calculated scale reliability psychosocial stress index: $\alpha = 0.785$. See appendix for complete component transformation scheme. Estonia ($N_j = 2045$) was dropped prior to EMB imputation as household income data were unavailable, yielding a final sample of $N = 38140$ in $j = 20$ countries post imputation.

Variables	Mean	Std. Dev.	Min.	Max	N	NA
Individual data						
Self-rated health	2.82	0.92	0	4	40136	49
Income	5.32	2.78	1	10	31889	8296
Psychosocial stress	0.95	0.41	0	3	34372	5813
Age	49.28	18.74	14	114	40086	99
Gender	0.53	0.50	0	1	40163	22
Education	12.90	3.94	0	50	39828	357
Partner	0.59	0.49	0	1	40035	150
Country data						
Gini index	0.30	0.04	0.25	0.38	20	0
Top 10% income share	0.35	0.05	0.29	0.50	20	0
Top 1% income share	0.11	0.03	0.07	0.17	20	0
Bottom 50% income share	0.21	.03	.13	.25	20	0
GDP per capita	41007.75	11696.99	25297.95	66018.42	20	0
Index components						
Feelings about income	0.95	0.84	0	3	39809	376
Autonomy at work	1.18	0.93	0	3	36595	3590
Influence work policy	1.64	1.01	0	3	36401	3784
Depression, how often	0.44	0.67	0	3	39975	210
Effort, how often	0.65	0.78	0	3	39964	221
Happy, how often	1.04	0.81	0	3	39812	373
Enjoying life, how often	1.06	0.85	0	3	39851	334
Feel sad, how often	0.53	0.67	0	3	39933	252
Can't get going, how often	0.55	0.71	0	3	39882	303
Sleep was restless	0.77	0.84	0	3	40007	178
Meet friends often	1.11	1.05	0	3	39595	590
Intimate relationships	1.65	0.64	0	3	39835	350
Social activities, how often	1.48	0.74	0	3	39603	582
Lonely, how often	0.39	0.69	0	3	39940	245

Table 2
Overview of effects by country. Effects were controlled for age, gender, education, and living with a partner. Total sample size post EMB imputation $N = 38134$. Final results combined over separate results from $m = 5$ imputed datasets.

Country	ACME	Direct	Total	P. Med.	N
Austria	0.025	0.008	0.032	0.768	1795
Belgium	0.032	−0.008	0.025	1.297	1769
Switzerland	0.025	0.002	0.027	0.932	1532
Czech Republic	0.039	0.034	0.073	0.534	2148
Germany	0.033	0.011	0.044	0.749	3045
Denmark	0.019	0.012	0.031	0.619	1502
Spain	0.035	−0.007	0.028	1.275	1925
Finland	0.020	0.029	0.049	0.412	2087
France	0.031	0.019	0.050	0.621	1917
Great Britain	0.031	0.022	0.052	0.586	2264
Hungary	0.061	0.002	0.063	0.966	1698
Ireland	0.036	0.016	0.052	0.698	2390
Israel	0.045	0.011	0.056	0.811	2562
Lithuania	0.045	0.013	0.058	0.777	2250
Netherlands	0.039	0.016	0.056	0.704	1919
Norway	0.017	0.021	0.038	0.447	1436
Poland	0.038	0.011	0.049	0.771	1615
Portugal	0.034	0.015	0.049	0.689	1265
Sweden	0.041	0.021	0.062	0.662	1791
Slovenia	0.022	0.017	0.039	0.558	1224

Income's direct effects ranged from the smallest of −.008 (Belgium) to the largest of 0.034 (Czech Republic). There was evidence for two different mediation processes. In countries where the direct effect of income is significant (Czech Republic, Finland, France, Great Britain, Ireland, Netherlands, Norway, Portugal, Sweden, and Slovenia), the total effect of income was partially mediated by psychosocial stress. The direct effect of income is not significant in Austria, Belgium, Switzerland, Germany, Denmark, Spain, Hungary, Israel, Lithuania, and Poland. In these countries, the evidence suggested full psychosocial mediation. Spain and Belgium showed marginally different patterns to the other countries. Point estimates suggested competitive mediation, which is a negative direct effect of income competing with a positively mediated effect of psychosocial stress. However, as the direct effect of income in these countries are non-significant, full mediation is concluded.

Total effects varied in line with each component effect. In countries with an insignificant direct effect of income, the total effect was slightly greater than the mediated effect of psychosocial stress (excepting Spain and Belgium). In countries with a significant direct effect of income, the component effects tended to have similar proportions.

Consulting the "P. Med" column in Table 2, the proportion of the total effect mediated by psychosocial stress varied from 41.2% to 96.6%, excluding proportions above 1. This table shows the problematic nature of evaluating the proportion of the mediated effect in isolation; competitive mediation means that the proportion of the total effect being mediated is uninterpretable as a traditional proportion.

Psychosocial stress accounted for a substantial proportion of the total effect of income in all countries studied, showing support for hypothesis **H1**. Hypothesis **H2** found support in countries with partial mediation, amounting to 10 out of 20 countries included in the sample.

5.1. Relationship with income inequality and GDP per capita

Fig. 2a plots the mediated, direct, and total effects over three levels of income inequality. Point estimates of the mediated effect of psychosocial stress on the income-health gradient are similar across the income inequality scale. The 95% bootstrapped confidence intervals suggest that the differences in the mediated effect of psychosocial stress between income inequality levels is not significant. The same can be said of the total and direct effect of income on self-rated health. Both the direct and mediated effects are significantly different from zero, supporting hypotheses **H1** and **H2**.

Fig. 2b plots the mediated, direct, and total effects over three levels of income measured in GDP per capita. Contrary to income inequality, the point estimates in the direct effect are the main drivers of changes in

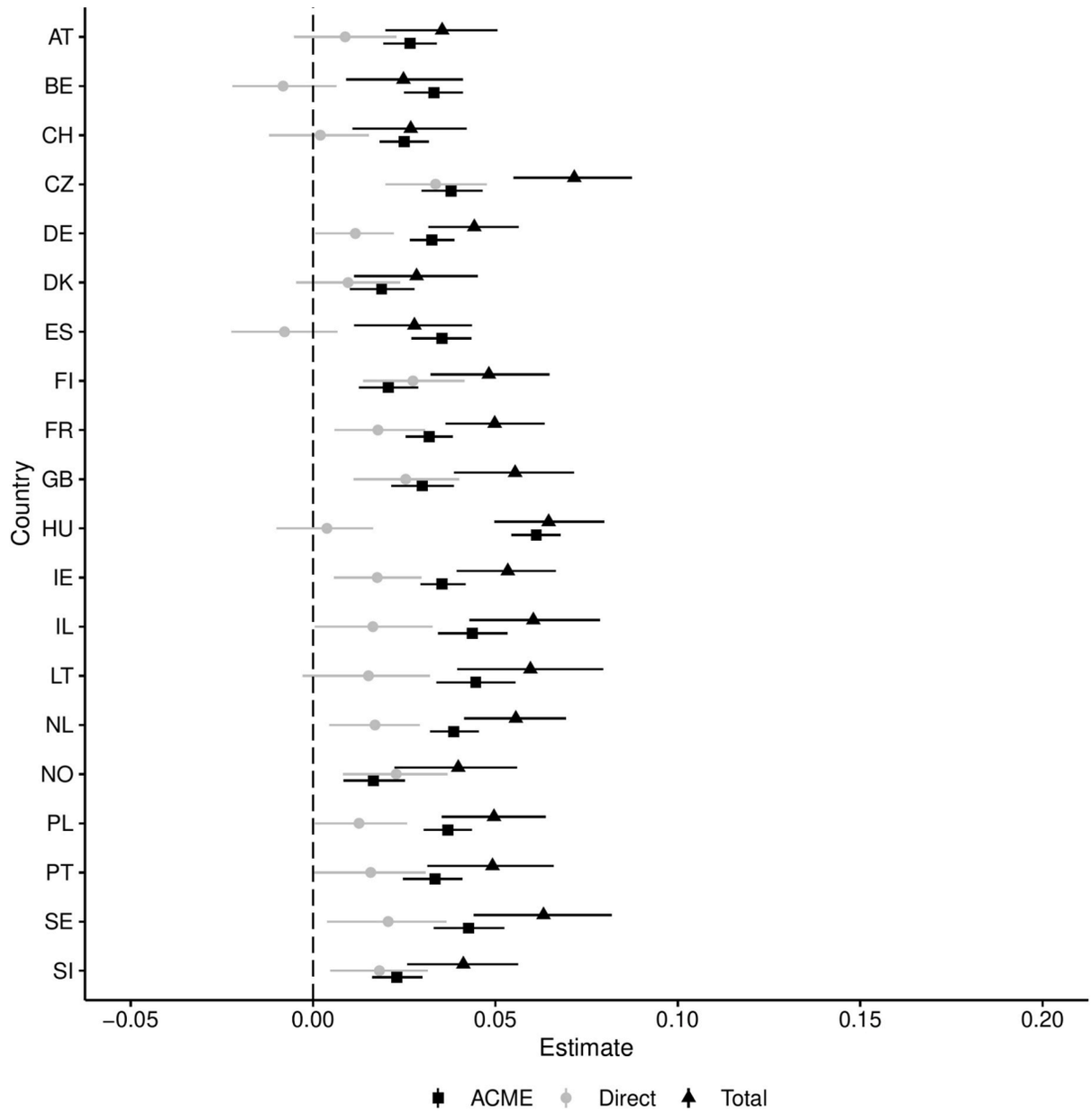


Fig. 1. Overview of mediated, direct, and total effect sizes including 95% bootstrap confidence intervals, by country. Effects were controlled for age, gender, education, and living with a partner. Final results combined over separate results from $m = 5$ imputed datasets.

the total effect of income. At a higher GDP, the direct effect of income tends to be smaller, while the mediated effect stays relatively stagnant over different levels of economic development. However, neither the direct effect of income, the mediated effect of psychosocial stress, or total effect are significantly affected by the level of economic development.

Fig. 3a and b plot the proportion of the total effect being mediated by psychosocial stress at three different levels of income inequality and GDP per capita, respectively. These figures are extremely similar but for different reasons. Increases in the point estimate of the mediated effect account for most of the variation in the total effect over income inequality levels. Conversely, decreases in the direct effect account for most of the variation in the total effect over different levels of GDP per capita. In both cases, this results in a minor increase in the predicted proportion of mediated effect. Similar to previous estimates, however, the proportion mediated effect does not vary significantly at different levels of either income inequality or economic development. Any differences between the observed effects failed to reach any conventional measure of statistical significance. **H3** and **H4** are not supported.

6. Discussion

Individual income matters for self-rated health, regardless of country-level income and income inequality. This does not mean that material poverty is the only factor in play. The psychosocial stress mediator accounted for 69.3% (median) of the total effect of income on self-rated health across countries, suggesting that psychosocial stress is correlated with income and self-rated health and accounts for a substantial amount of the covariance between income and self-rated health.

The IIH argues that long-term feelings of inferiority act as primary mechanisms of why income matters for health (Pickett & Wilkinson, 2015; Wilkinson, 1994) and assumes that the situation worsens in societies with higher levels of income inequality. That is, the potential for feeling worse is a result of relatively lower positioning in the hierarchy, exacerbated by the gulfs in income generated by income inequality. This prediction largely failed in the context of ESS data.

The more probable reason for a strong mediating effect is rooted in the lived experience of stress and how this covaries with individual-level incomes. Rather than considering the psychosocial environment a

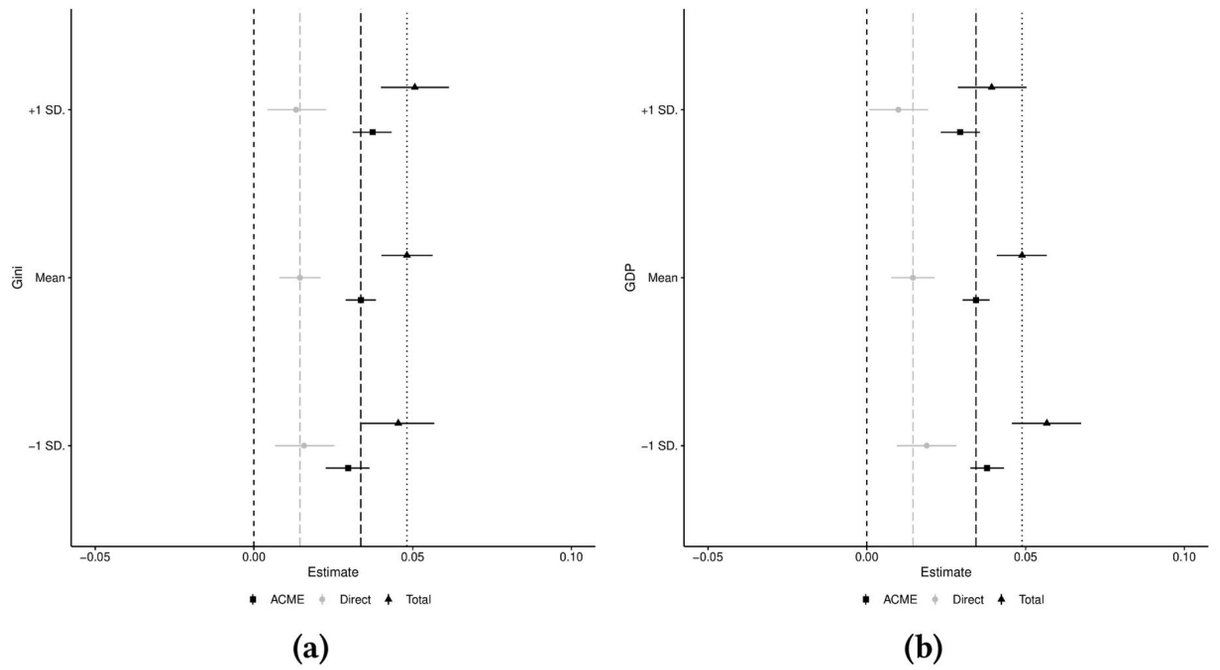


Fig. 2. *Left: ACME, direct, and total effects from multilevel moderated mediation model at three different levels of income inequality. Right: ACME, direct, and total effects from multilevel moderated mediation model at three different levels of GDP. Both figures include 95% bootstrap confidence intervals. Vertical lines are centered on the mean estimate and zero. Final results combined over separate results from* $m = 5$ *imputed datasets.*

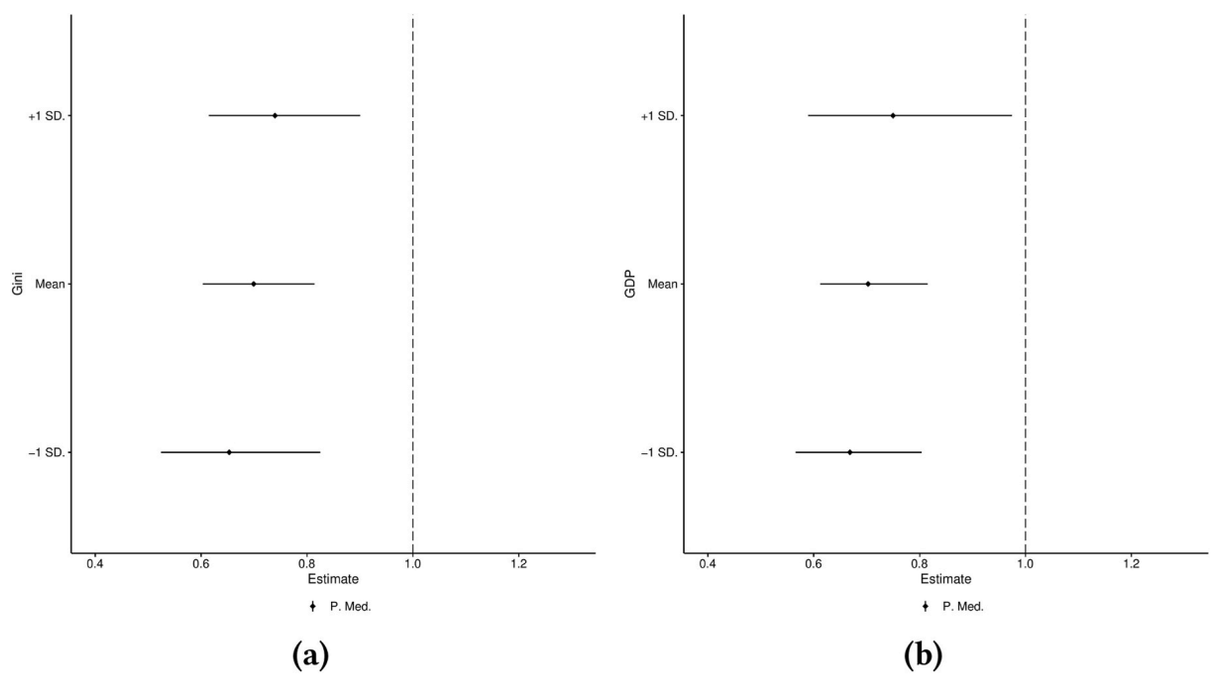


Fig. 3. *Left: Proportion of mediated effect of income from multilevel moderated mediation model at three different levels of income inequality. Right: Proportion of mediated effect of income from multilevel mediation model at three different levels of GDP* per capita. *Both figures include 95% bootstrap confidence intervals.*

standalone effect resulting in stress, psychosocial stress may result from low income itself. This would be due to those in low-income groups having a greater prevalence of less comfort, more worries about finances, depression, fatalistic tendencies, lack of control, and lacking social affiliation.

Lynch et al. (2000) argued that "health inequalities result from differential accumulation of exposures and experiences that have their sources in the material world." They also posited that the income distribution is a result of historical, cultural, and politico-economic processes that shape the nature of public infrastructure. The psychosocial interpretation argues that while the negative emotive experience is rooted in material income conditions, the negative effects occur due to a low position on the social hierarchy, specifically (Wilkinson, 1994, 1999). Kawachi et al. (2002) argued that, in reality, these explanations are not mutually exclusive or possible to disentangle. One key factor when discussing psychosocial and material causation is distinguishing between underlying pathways to health, and initial causes of health. Psychosocial factors like low social status and lack of control are often labeled psychosocial determinants, although they may be triggered by material factors. It is here that the theories intersect most notably, when

considering how material hardship in lower socioeconomic groups is a likely source of psychosocial stress (Mackenbach, 2012). Empirical overlap between material factors and the hypothesized emotional experiences of inequality is likely. Following Kawachi et al. (2002), if we consider the psychosocial stress hypothesis to be a causal pathway, there is no apparent conflict between the two. All material resources have some psychosocial meaning attached to it, but they also provide a sense of material security. This sense of material security (or scarcity) combined with both material and neo-material perspectives would help explain why there is a gradient in the first place. The end result is less focus on a position of inferiority and the subjective experience of income inequality, and instead, more focus on the psychological benefits of financial stability and security.

This implies that psychosocial pathways are not an initial cause. The lack of an appreciable effect of income inequality alone on effect sizes suggests as much. Pickett and Wilkinson (2015) argued that because income inequality has been linked to lower levels of social cohesion and generalized trust, it means that inequality must act as a social stressor. The psychosocial explanation of the income effect is argued to be biologically plausible when linked with the detrimental health effects of chronic stress. What is missing empirically is the expected exacerbation of mediated and total effects over income inequality. The psychosocial stress effect of income is therefore to a greater extent about general feelings or behaviors associated with low income. The effect of income being fully mediated by the psychosocial stress index in many countries suggests that psychosocial stress matters for self-rated health, linking material goods to psychosocial pathways.

The IIH, regardless of mechanisms or empirical support, cannot exist without an income–health gradient. One can apply most theoretical frameworks and reach similar conclusions that there are statistically appreciable differences in health based on income groups. Theoretical divergence on this effect occurs because of the shape of that gradient. That is, income–health differences are not merely the differences in health between the rich and the poor. An income gradient in health is the necessary backbone upon which a hypothetical income inequality effect rests. The IIH is motivated by the inability of the income–health hypothesis to explain relative homogeneity in population health between the wealthiest of countries (Marmot & Wilkinson, 2001). In other words, the IIH exists only in relation to the income–health hypothesis. Regardless of the effect or lack thereof from income inequality on health, the literature mostly agrees on policy recommendations: reducing income inequality will lead to better population health. Reducing income inequality strategically means raising disadvantaged people out of material hardship, falling back on the established mechanisms of the income–health hypothesis.

6.1. Strengths and weaknesses

A major strength of this study is its novelty. Several studies have embraced the comparative nature of the IIH (Layte et al., 2019; Olstad et al., 2022), but no study to date has tested the IIH in a multilevel moderated mediation framework. Further, the study establishes a novel psychosocial stress measurement based on the conceptual framework presented by Marmot (2001) that may be used or amended for future comparative studies on the income–health gradient and tests of the IIH.

The psychosocial stress index includes items measuring depressive symptoms, lethargy, and restless sleep. Single-item stress measurements have been shown previously to converge on similar psychological symptoms, sleep disturbance items, and well-being (Elo, Leppänen, & Jahkola, 2003). While depression is also a component of health in self-ratings, self-rated health as a concept is comprehensive, inclusive, and non-specific. It applies contextual frameworks of evaluation to ones own health status such as culturally varying conceptions of health, makes reference to previous experiences and the health status of others, and reflects cultural conventions in expressing health and health related issues (Jylhä, 2009). As long as psychosocial stress is partially defined by depression, anxiety, and the like, some conceptual overlap between health and psychosocial stress is inevitable. However, correlations in the ESS7 show that items in the index reflecting depression and well-being are more strongly correlated internally than with self-rated health. The psychosocial stress index only accounts for $R^2 = 0.21\%$ of the variance in self-rated health. These points suggest that psychosocial stress and self-rated health are related, but distinct concepts.

As noted by Beckfield (2004), sample (country) variations may impact the estimated country-level correlations. While there are ample sample sizes at the individual level, a small number of countries means comparatively large standard errors and increases the probability of sub-sample variability. Further, the sample does not fully reflect the global variation in income inequality or economic development. While this region is theoretically relevant for the IIH, future studies should aim to include a larger sample of countries that represent the global variation in income inequality and economic development.

Zhao, Lynch Jr., and Chen (2010) argued that partial mediation suggests an incomplete theoretical framework, but notes that there are instances where the direct effect is an a priori expectation. While it can be argued that material effects themselves should be mediated, by, for instance, measuring house ownership or similar sources of capital, the direct effect is simply assumed to represent material effects of income. There are at least two behavioral mechanisms that may bias this interpretation of a direct effect as materialistic: scarcity theory, (Mullainathan & Shafir, 2013) where an additional cognitive load due to poverty means individuals prioritize short-term needs at the expense of long-term planning and decision-making; and diffusions of innovations (Rogers, 1962), which is the tendency for the rich or highly educated to adopt innovative health behaviors early. Effectively estimating potential biasing effects of scarcity theory necessitates a measurement of an individual's cognitive capacity and their relative cognitive load specifically attributable to scarcity. That is not exactly a standard indicator in international comparative survey data. Additionally, the diffusion of innovations mechanisms are interrelated with other theoretical assumptions and difficult to parse from existing frameworks. The adoption of healthy behaviors and health-related technology could proxy this effect, but would be restrictive to specific conditions (such as preventive breast cancer screening) that are likely to be insensitive approximations.

Fairchild and McDaniel (2017) pointed out that mediation is mostly appropriate in data contexts where temporality can be established. They argued that examining mediation analyses with cross-sectional data requires the researcher to provide a compelling rationale that temporal ordering of the examined variables is correct. Income must precede a biological stress response. Ideally, income would be measured at time $T-1$. However, stress and income levels are expected to exist concurrently. As the psychosocial stress hypothesis de-emphasizes material well-being for the lived experience of relative income, it should result in temporal overlap. Given that ESS data are repeated cross-sections and not repeated individual observations, no before-and-after treatment may be observed at the individual level. In this study, direct, total, and mediated effects should be understood as correlational in nature. Mediation being identified in data is not the same as concluding a process of mediation. However, mediation as a process linking income to health is theoretically plausible. This study primarily infers on the likelihood of these causal pathways.

Still, the possibility that the income-health relationship is reversed or bi-directional is a fundamental issue in cross-sectional studies. Ill-health may impact the probability of employment, and experiencing a health shock increases the likelihood of leaving employment and transition into disability (García-Gómez, 2011). Early life health conditions may constrain economic success in adulthood, as ill-health in childhood may affect opportunities to acquire education or reduce the efficiency of schooling (O'Donnell et al., 2015). Psychosocial theory attempt to create a link between socioeconomic positioning and health outcomes by directing attention to endogenous biological responses to human interaction (Krieger, 2001). Extending the health selection argument, it is

possible that ill-health causes psychosocial stress for instance through difficulties with coping or onset depression. However, it is difficult to conceive of reverse *psychosocial mediation* from health to income in this case; the direct mechanisms from ill-health to reduced income seem more likely.

This study considers age as a confounder of the income–health relationship and is agnostic to age-differentiated causal mechanisms between income and health. It also includes respondents ranging from adolescence to old age. Earlier research has suggested that the relationship between income and health varies over the life-course. This is particularly apparent in age groups where transitioning between age-stratified institutions are common; labor market entry and retirement ages (Rehnberg et al., 2021). This age-differentiated relationship extends to age-specific causal mechanisms. Hoffmann et al. (2018) argue that social causation is more important than health selection in the second part of the life course, in the transition from adulthood to old age. While this study does not address age-specific mechanisms, including all age-groups available in the statistical model aligns with the universal assumption in psychosocial theory; that perceptions of relative positioning in the social hierarchy are always present and that all citizens are to some extent subject to the hypothesized effects of income inequality (Lynch et al., 2000).

A natural extension for future research includes comparative analyses of repeat observations from individuals in order to investigate to what extent changes in individual income or psychosocial stress affect health outcomes and changes in health outcomes differently, depending on economic context. Future studies may also attempt to parse the mediative effect of psychosocial stress on the income–health relationship by age-groups, in order to specify the exact mechanisms at play at different stages of the life-course.

7. Concluding remarks

Individual-level mechanisms remain important for explaining the income–health gradient in Europe. Evidence of the IIH is mixed, and the psychosocial stress mechanism should be pursued and researched further insofar as it may represent a biological response to individual income levels. While effects of individual income remain relevant, the effects of income are not merely material; a higher level of material comfort tends to correlate with a lower level of psychosocial stress.

Lacking evidence of an income inequality effect specifically does not entitle policymakers to avoid redistributive income policies. Policies should be aimed at allowing a greater number of people to live with a certain degree of material comfort and a reduced sense of financial precarity. Reducing income inequality by targeting those at a comparatively low income, reducing the potential consequences facing low income earners through generous welfare benefits, and ensuring an equitable distribution of public and private resources remain potential pathways to achieve health gains through both material and psychosocial mechanisms, despite the lack of convincing evidence for the IIH specifically.

Declaration of competing interest

None.

Data availability

Data will be made available on request.

Acknowledgements:

This work was supported by the High North Population Studies, UiT The Arctic University of Norway. I would like to express my appreciation and gratitude to the participants of the The Political Determinants of Health in the EU 2022 workshop for valuable comments and considered criticism on an early draft of this manuscript.

A Alternate inequality measurement models

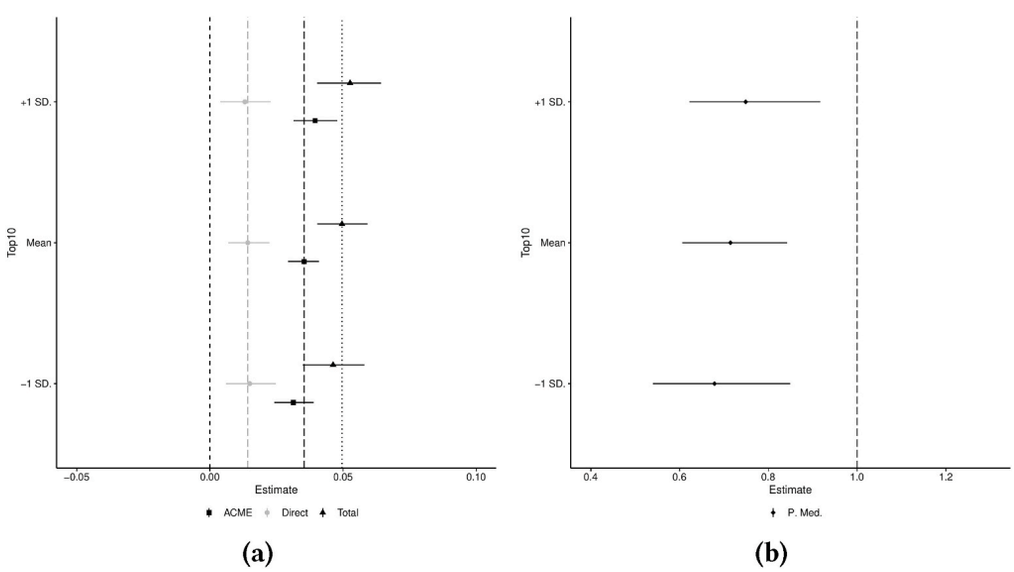
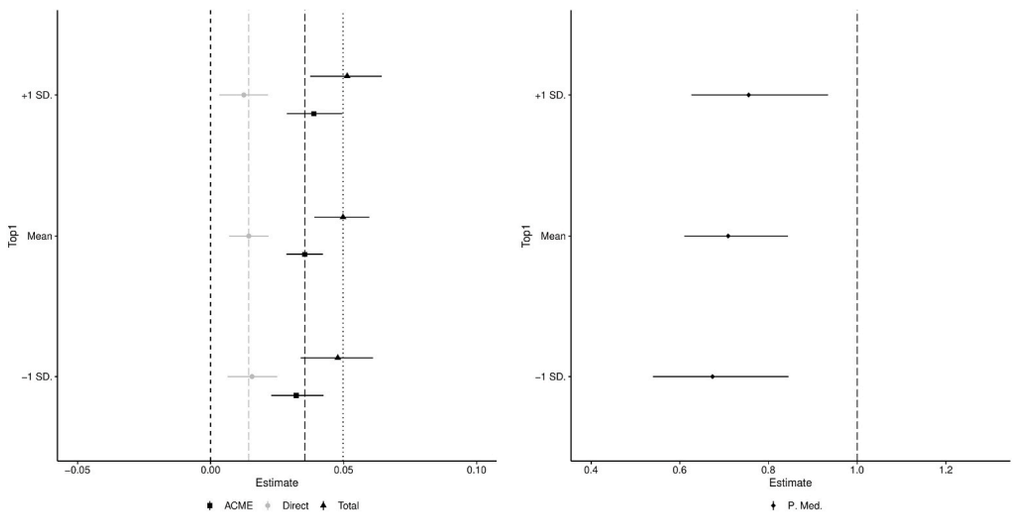
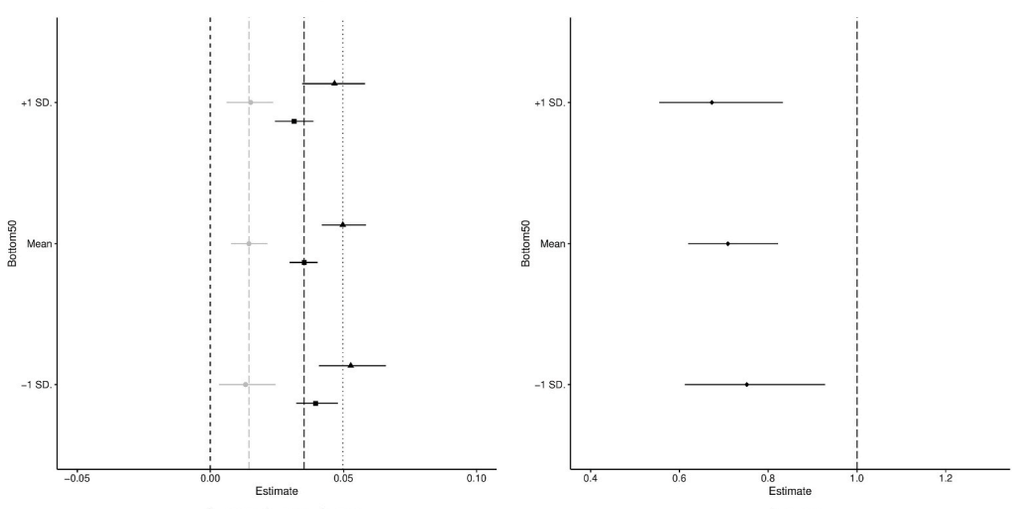


Figure A1. Left: ACME, direct, and total effects of multilevel moderated mediation model at three different levels of income inequality (top 10% income share). Right: Proportion of mediated effect at three levels of income inequality.



(a) (b)

Figure A2. Left: ACME, direct, and total effects of multilevel moderated mediation model at three different levels of income inequality (top 1% income share). Right: Proportion of mediated effect at three levels of income inequality.



(a) (b)

Figure A3. Left: ACME, direct, and total effects of multilevel moderated mediation model at three different levels of income inequality (bottom 50% income share). Right: Proportion of mediated effect at three levels of income inequality.

B Main models using listwise deletion

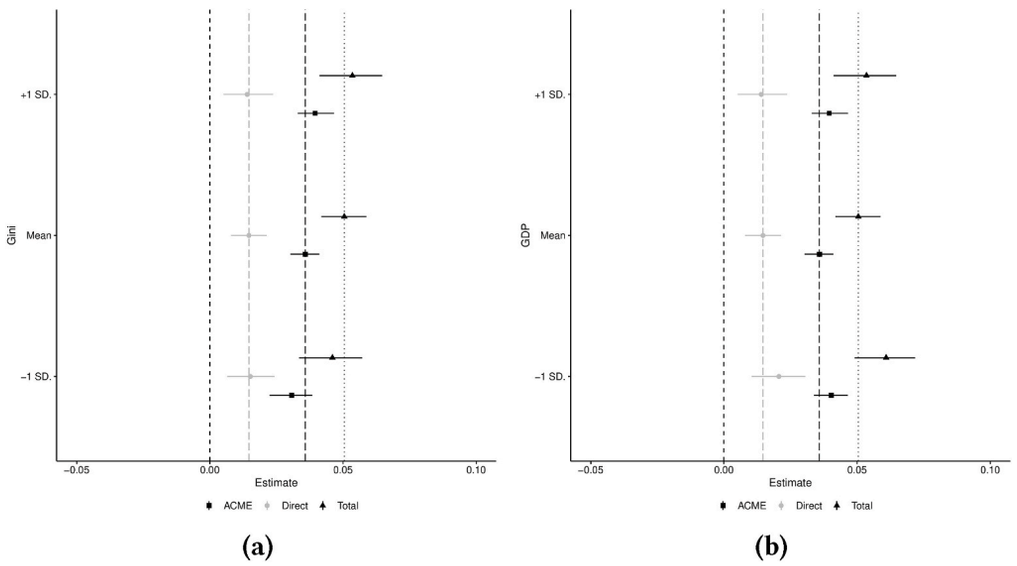


Figure B1. Left: ACME, direct, and total effects from multilevel moderated mediation model at three different levels of income inequality. Right: ACME, direct, and total effects from multilevel moderated mediation model at three different levels of GDP. Both figures include 95% bootstrap confidence intervals. Vertical lines are centered on the mean estimate and zero. Both models based on $N = 28814$ observations by listwise deletion.

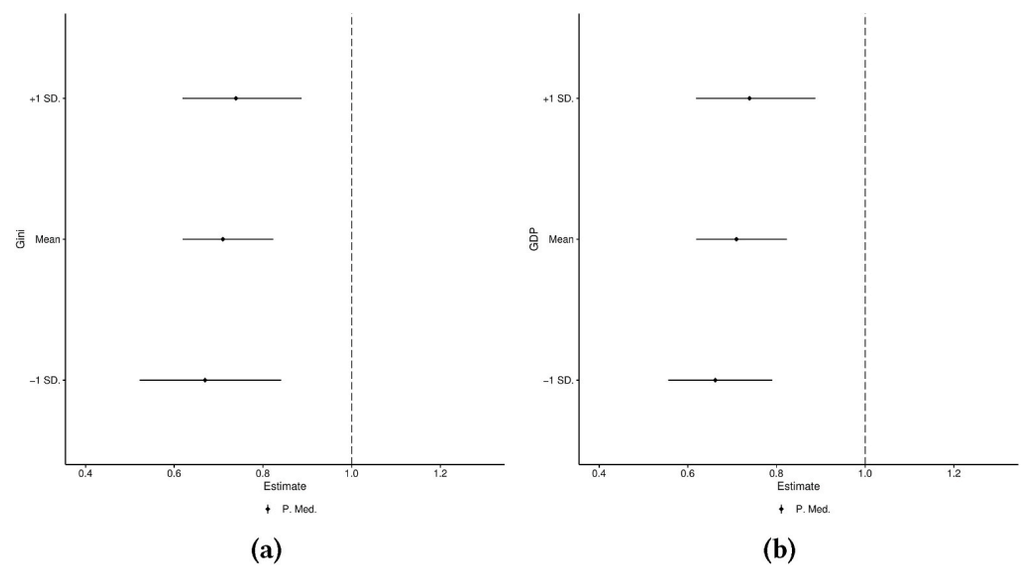


Figure B2. Left: Proportion of mediated effect of income from multilevel moderated mediation model at three different levels of income inequality. Right: Proportion of mediated effect of income from multilevel mediation model at three different levels of GDP per capita. Both figures include 95% bootstrap confidence intervals. Both models based on N = 28814 observations by listwise deletion.

C Transformation scheme

Table C1
List of indicators, anchor labels, and transformation output for all categories in all items used in the psychosocial stress index. Note that ampersands and hyphenation indicate separate categories and ranges on the original scale. Items 2–9 share anchor labels and were separated in table to show the inverted collapsed scale.

#	Item	Original	Collapsed
1	Feelings about income	Living comfortably	0
		Coping	1
		Difficult	2
		Very difficult	3
2–7	Felt depressed	None or almost none of the time	0
	Effort, how often	Some of the time	1
	Feel sad, how often	Most of the time	2
	Can't get going, how often	All or almost all of the time	3
	Sleep was restless		
	Lonely, how often		
8–9	Happy, how often	None or almost none of the time	3
	Enjoying life, how often	Some of the time	2
		Most of the time	1
		All or almost all of the time	0
10–11	Influence work policy	Had no influence	3
	Autonomy at work	1–4	2
		5–9	1
		Had complete control	0
12	Meet friends, how often	Never & Less than once a month	3
		Once a month & Several times a month	2
		Once a week	1
		Several times a week & Every day	0
13	Intimate relationships	None	3
		1–3 & 4-6	2
		7–9	1
		10 or more	0
14	Social activities, how often	Much less than most	3
		Less than most	2
		About the same & More than most	1
		Much more than most	0

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Article II

Sigbjørn Svalestuen et al. (2023). “The predictive importance of education, income, and occupation on non-communicable disease outcomes: results from the Tromsø Study.” *Under review*

Article III

Sigbjørn Svalestuen et al. (2022). "Association of area-level education with the regional growth trajectories of rates of antibacterial dispensing to patients under 3 years in Norway: a longitudinal retrospective study." *BMJ Open* 12.9. ISSN: 2044-6055. DOI: 10.1136/bmjopen-2021-058491. eprint: https://bmjopen.bmj.com/content/12/9/e058491.full.pdf. URL: https://bmjopen.bmj.com/content/12/9/e058491

BMJ Open

Association of area-level education with the regional growth trajectories of rates of antibacterial dispensing to patients under 3 years in Norway: a longitudinal retrospective study

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To cite: Svalestuen S, Svendsen K, Eggen AE, *et al*. Association of area-level education with the regional growth trajectories of rates of antibacterial dispensing to patients under 3 years in Norway: a longitudinal retrospective study. *BMJ Open* 2022;**12**:e058491. doi:10.1136/bmjopen-2021-058491

► Prepublication history and additional supplemental material for this paper are available online. To view these files, please visit the journal online (http://dx.doi.org/10.1136/bmjopen-2021-058491).

Received 19 October 2021
Accepted 19 August 2022

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ABSTRACT

Objective To examine the association between area-level education and the local growth trajectories in antibacterial dispensing rates in Norwegian municipalities among children under 3 years old.

Design Retrospective, longitudinal study using individual primary care prescription data from the Norwegian Prescription Database for the period 2006–2016. Data were collected on the date of dispensing, the type and amount of antibiotic, the patient's age, sex and municipality of residence and linked to municipality-level statistics on education available from Statistics Norway. We used multilevel growth curve modelling, with a linear trend variable modelled as a random effect and a cross-level interaction between linear trends and the proportion of the population in the municipality having received a university or college education.

Setting The local government level in Norway. The sample includes all municipalities over the study period.

Outcome measure Number of dispensed antibacterial prescriptions per 100 children in individual primary care by municipality and year.

Results We identified a significant negative linear trend in the square root of the dispensing rate for children under 3 years old during the period. This trend varied between municipalities. A negative cross-level interaction term between population education levels and random trends showed that municipalities with an average level of population education saw a reduction in their square root dispensing rates of −0.053 (95% CI −0.066 to −0.039) prescriptions per 100 children. Each additional percentage point in population education contributed a further −0.0034 (95% CI −0.006 to −0.001) reduction to the square root dispensing rate.

Conclusions Municipalities in which a larger proportion of the local population have high educational achievements have been more successful in reducing antibacterial dispensing rates in children under 3 years old. Adopting area-level strategies and addressing local community disadvantages may help to optimise practices and prescribing patterns across local communities.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Complete antibacterial dispensing data allow estimations of local community dispensing rate trends and their associations with education at a high level of spatial resolution.
⇒ By including all Norwegian municipalities, we explored the total extent of local variations in dispensing rates under national reduction policy guidelines.
⇒ Aggregate data cannot directly infer individual-level decision-making and needs.
⇒ We were unable to control for the geographical burden of infectious disease in the age groups under examination.

INTRODUCTION

The periodic prevalence and patterns of antibiotic use vary between countries[1] and between socioeconomic and demographic groups within countries,[2–6] and studies have also shown temporal variations in the dispensing of antibacterials for systemic use.[7 8] One study from Norway found an overall reduction in the number of dispensed prescriptions among children aged 0–2 between 2005 and 2016, with the prevalence varying between counties.[9] Another study found that, among Norwegian children aged 0–2, 1-year olds consistently had the highest antibacterial dispensing rates between 2008 and 2016.

Several studies have attributed variations in antibacterial use to socioeconomic characteristics,[3–5 10–12] often including an indexed area-level deprivation measurement to capture several dimensions of deprivation (eg, education, income, barriers to housing, crime, employment). Crowding, hygiene, lower host resistance due to poor nutrition, stress and smoking prevalence create a greater risk of infectious illness among people of lower socioeconomic status, but general practitioners'

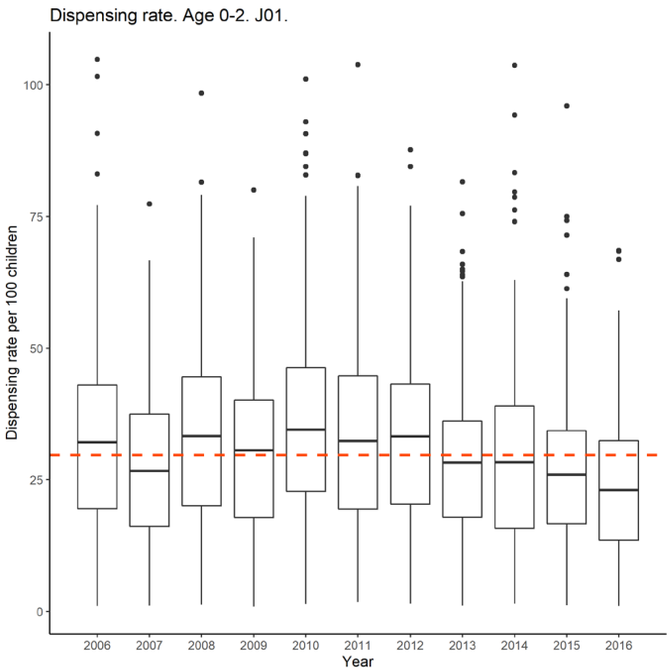

Figure 1 *Box-and-Whisker plot of dispensing rates by year. The dashed line is the grand mean dispensing rate throughout the period. The main takeaway from this figure is the notable variation between municipalities within a specific year. The intraclass correlation coefficient of the null model indicates that 62.8% of the total variance is between municipalities.*

treatment practices and their interactions with family attitudes towards demanding certain treatments may influence prescription dispensing,[2 13] resulting in geographic and temporal variations in aggregate statistics. Education is associated with the awareness and proper use of antibacterials[14–16] and with the individual capacity to obtain, process and understand health information,[17 18] and cultural factors, such as individual versus collective value systems, and future-oriented behaviour have also been associated with prescription patterns at multiple levels.[19]

Studies on variations in dispensed antibiotics in Norway have not explicitly modelled local variations in dispensing rate growth trajectories in terms of socioeconomic composition. The aim of this study was to investigate the association between population education levels and growth trajectories in antibacterial dispensing rates at the municipality level using longitudinal data and a multilevel growth curve model.

MATERIALS AND METHODS

The Norwegian Prescription Registry (NorPD) contains all prescriptions with a valid unique personal identifier redeemed at Norwegian pharmacies; details of the NorPD are published elsewhere.[20] We considered the period from 2006 to 2016 and included 734 359 prescriptions. We aggregated prescriptions if the same individual received two or more prescriptions for the same antibacterial drug on the same date, and we excluded records for individuals aged more than 1095 days (3 years) and those who died during the observation period. We used the following data from the NorPD: sex; year and month of birth; unique personal identifier; municipality of residence; date on which the prescription was dispensed at the pharmacy and the Anatomical Therapeutical Chemical Classification System (ATC) code at the fifth level. As we only had information on the birth month in our data, we assigned a fictious birth date of the 15th of the birth month and calculated age as the date of dispensing minus this date.

Data in NorPD are pseudonymised, allowing longitudinal observation of an individual who is anonymous to the researcher. Individual data were aggregated at the municipality level, and dispensing rates were calculated as the yearly number of prescriptions within a municipality per 100 children. We linked the aggregated prescription data to publicly available data on all Norwegian municipalities using the unique municipality identification number system. Analyses were restricted to ATC J01: antibacterials for systemic use.[21] The data cover the entirety of Norway at the local administrative level. Figure 1 presents a box-and-whiskers plot of the calculated local dispensing rate by year. Online supplemental appendix figure A1 presents a sample of trends and intercepts fitted to the dispensing rate metric.

Exposure and covariates

Our exposure was the proportion of the population in a municipality who had received tertiary education (university level for 3 or more years).[22] We chose tertiary education as our education indicator for two reasons. First, the literature states that knowledge of the proper use of antibiotics is more common among people who have received a higher education,[14–16] and second, the Norwegian education system ensures all young people the legal right to education up to and including upper secondary education, but no such right exists for higher education. Thus, continued education past the secondary level is an active choice, in contrast to structured schooling, so we would expect local population diversity.

We included a covariate for the proportion of the population in a municipality living in a household with less than 60% of the national median income,[23] which is the standard definition of low income in the European Union. The association between deprivation and dispensing rates[3–5] suggests that poverty may confound the relationship between dispensing rates and population education, and including this covariate served to partial out effects that could be attributed to education rather than to material deprivation.

The municipality population size may be related to levels of regional deprivation in education and to regional development and may, therefore, impact access to healthcare services. A previous study identified an association between municipality population size and dispensing rates in Norway,[6] and municipality size is, therefore, likely to confound the link between education and dispensing rates. Populations of Norwegian municipalities vary from

fewer than 400 to more than 600 000 residents, and to best capture this variance, we calculated the natural logarithm of population size collected from official statistics[24] as an indicator of municipality size.

Finally, we included an indicator for the median travel time to the nearest pharmacy, calculated using Google Maps to determine travel time between all addresses in Norway and their three nearest straight-line pharmacies, selecting the shortest travel time by car for each address before aggregating to the municipality level. A previous Norwegian study[25] found a link between dispensing rates and travel times to pharmacies in Norway. If education levels are geographically determined, they are also likely to correlate with pharmacy access, and it is, thus, important to partial out the effects of ease-of-pharmacy access from the educational coefficients.

Statistical analysis

Multilevel growth curve models are a special case of multilevel models in which a coefficient of time varies between units.[26] The variation in each unit of the dispensing rate is modelled as a fixed growth trajectory plus a random error term, which means that the parameters of growth can be modelled by background characteristics.[27] Applying this to our data, the municipalities are repeatedly observed, such that level 1 constitutes the longitudinal part of the model and level 2 captures the variance between the municipalities.

We centred all level 1 covariates, except time, on their cluster means—that is, centring within cluster—to achieve orthogonality between the level 1 and level 2 variables.[28] The covariates at level 1 were annual measurements of poverty, education and municipality population size, which reflect changes in the municipality by year. The same covariates were aggregated at level 2 as cluster means. These covariates reflect differences between municipalities over the period under study. All level 2 covariates were conversely centred on their grand mean. This centring scheme allows for easier interpretation of main effects in the interaction term, in which the estimated trend coefficient is interpreted as the expected mean dispensing rate trend in municipalities at average levels of population education. Time (L1) was not centred because we were interested in the average trend over the period (see Biesanz *et al*[29] for a discussion on centring time in growth curve models).

The multilevel growth curve model assumes that time-variant covariates are not characterised by a systematic growth process, and the inclusion of simultaneous growth processes in a multilevel growth curve model may lead to misspecification and biased effects.[30] Within-municipality variations in education levels are highly correlated with time ($r = .95$), providing evidence for simultaneous growth and biasing the trend coefficient. We, therefore, removed the time-variant education predictor, as our goal was to estimate a cross-level interaction effect between the time-invariant education predictor and trends. We detail this choice further in the online supplemental appendix and demonstrate the consequences of simultaneous growth on trend estimation in online supplemental table A1.

We performed a square root transformation on the dispense rate metric to improve the model fit, but the coefficients on the square root scale lack the clean interpretability of coefficients on the original scale. We, therefore, used the square root model for predictions and for the evaluation of statistical significance but present the predicted dispensing rates using the original scale to aid in interpretation. Untransformed and square root transformed dispensing rate distributions are available in online supplemental appendix figure A2 and A3, respectively.

The model fit was assessed using the Akaike information criterion, the Bayesian information criterion and residual diagnostic plots. Residual diagnostic plots are available in online supplemental appendix figure A4–A7. All models were estimated using the R package *nlme*, incorporating a compound symmetric error covariance structure to deal with within-group autocorrelation. A model equation and a parameter description are available in the online supplemental appendix.

Patient and public involvement

No patients were involved.

RESULTS

The model results are shown in table 1, and figures 2 and 3 are based on estimates from the model. An untransformed version of the model is available in online supplemental table A2. Table 2 shows summary statistics for the types of antibacterial in the database, together with the total number of defined daily doses dispensed, summarised by year and subgroup. Table 3 presents summary statistics. Online supplemental table A3 includes detailed summary statistics on within and between components specifically.

From model 1 in table 1, it can be seen that the estimated mean trend of the square root dispensing rate at mean levels of population education is equal to −0.053 (SD=0.0927, p<0.001). A one percentage point increase in cluster mean education reduces the trend coefficient of the square root dispensing rate by −0.0034 (p=0.0051), *ceteris paribus*. There is, thus, a greater reduction in the dispensing rate in municipalities in which a larger proportion of the population has received tertiary education.

Figure 2 presents the predicted trajectories in the dispensing rates based on cluster mean education levels. An important observation is that the trends are, on average, negative within the boundaries of the data. Even the municipalities with the lowest levels of population education (11%) show predicted reductions in dispensing rates. The predictions fan out from similar intercepts due to the small and insignificant 'main' effect of education (the effect when $T = 0$, p=0.892) in the model. The figure shows that the municipalities with low levels of population education have predicted reductions

Table 1 Multilevel linear growth curve model

Coefficient	√Dispensed Rx per 100 children	P values
Level 1		
Trend	−0.053 (−0.066 to −0.039)	<0.001
Poverty	−0.098 (−0.125 to −0.070)	<0.001
Population (ln)	1.265 (−0.061 to 2.592)	0.062
Level 2		
Education	−0.002 (−0.027 to 0.023)	0.892
Population (ln)	0.408 (0.290 to 0.525)	< 0.001
Poverty	−0.085 (−0.130 to −0.041)	< 0.001
Travel	−0.0003 (−0.0004 to −0.0003)	< 0.001
Trend×Education (L2)	−0.0034 (−0.006 to −0.001)	0.005
Intercept	5.459 (5.340 to 5.578)	< 0.001
Variance components		
Standard deviation. μ_1	.0927	
Standard deviation μ_0	.8647	
Misc.		
ρ Compound symmetry	.000	
Groups	426	
Observations	4503	
Log Likelihood	−6442.764	
Akaike information criterion	12913.53	
Bayesian information criterion	13003.3	

95% CI in parentheses.
The model uses the square root of the transformed dispensing rates as outcomes. This model is used for the prediction (figures 2 and 3) and evaluation of statistical significance and rates of change. Complete information is missing only for two municipalities due to municipality mergers during the period.

of approximately two prescriptions per 100 children, while municipalities with comparatively high levels of population education have predicted reductions approximately equal to 10 prescriptions per 100 children over the period. In figure 3, several municipalities can be seen to have a positive-predicted trend after adjusting for the interaction with education. Most municipalities, however, show a predicted negative trend in the cross-level interaction model, and the size of the negative trend varies with population education in the municipality.

DISCUSSION

While there has been a national decrease in antibacterial dispensing rates in Norway,[31] the current study shows that trends vary between Norwegian municipalities for patients below 3 years of age, with municipalities in which more of the population has received tertiary education showing larger decreases in dispensing rates. Several efforts have been made to reduce antibacterial dispensing rates, notably by updating national guidelines for the use of antibacterials[32] and through intervention campaigns.[33] If one views high education levels as a form of socioeconomic advantage, the results suggest that municipalities with socioeconomically advantaged populations have been more successful in reducing dispensing rates.

Our findings support the existing literature on the relationship between relative socioeconomic deprivation and antibacterial dispensing rates. Low parental education has been linked to higher prescribing rates in paediatric patients,[2 5 13 34] and we would expect the same individual mechanisms to translate to aggregate statistics. If a lack of higher education in a community is considered a form of regional deprivation, then these results are consistent with other data on the association between area-level deprivation indexes (which include education in the index) and dispensing rates.[3 4 11]

We chose tertiary education as our education indicator because proper use of antibiotics is more common in people who have received higher education,[14–16] and our findings are consistent with these expectations. In addition, the Norwegian education system ensures all young people the legal right to education up to and including the upper secondary level, but no such right exists for higher education. Thus, continued education past secondary level is an active choice in which we would

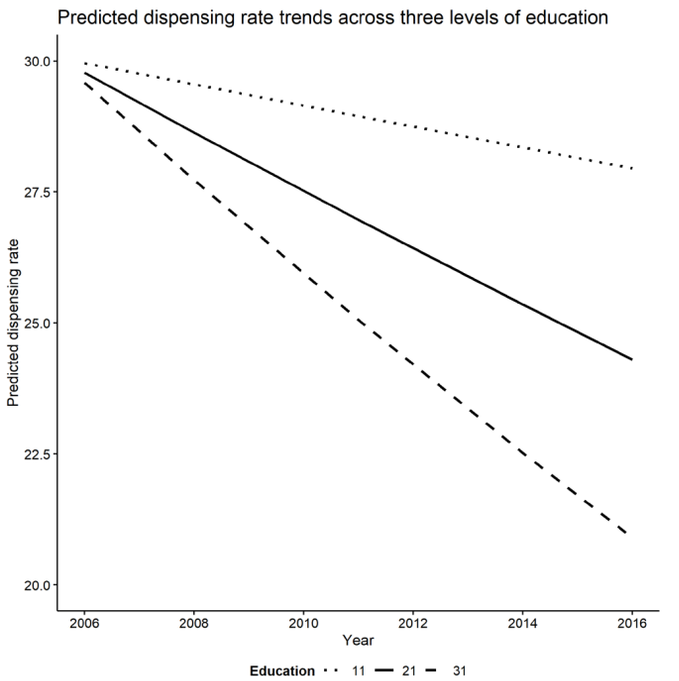

Figure 2 *Predicted cross-level interaction effect between trends and education. The Y-axis displays the dispensing rate on the original scale. The middle line represents the average cluster level of education, while the outer lines are predicted trends for±2 SD from the mean education levels. Predictions fan out from similar intercepts due to the insignificant main effect of education (effect when T=0).*

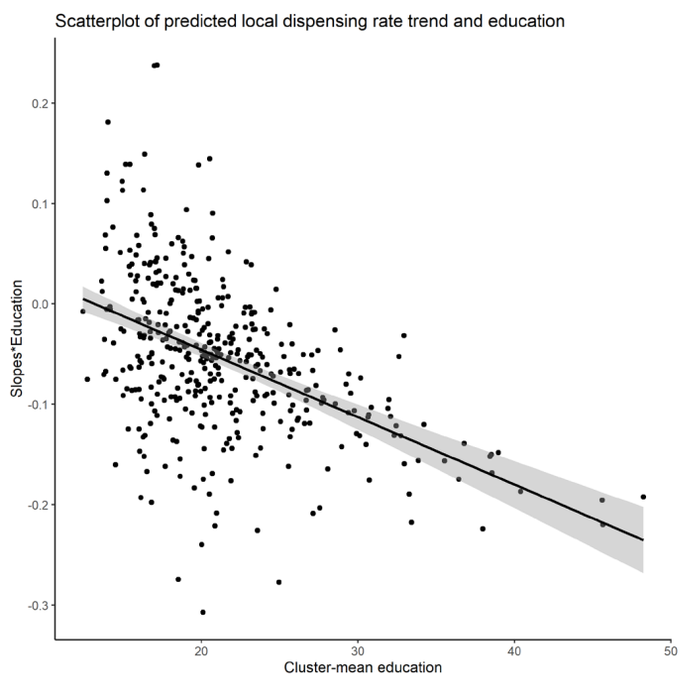

Figure 3 *Predicted slopes by population education. The points are the predicted square roots of the dispensing rate trends for each municipality. All 426 estimated trends are presented and plotted against education on the X-axis. The figure shows that the leaders in dispensing rate reductions also tend to have higher proportions of people with tertiary education and, conversely, that low performers tend to have lower levels of tertiary education. Please note the Y-axis scaling when interpreting the figures.*

expect local population diversity, in contrast to structured schooling.

Health literacy is also associated with higher education,[17 18] but education is an inaccurate proxy for individual health literacy.[35] However, the overuse of antibacterials and policies implemented to reduce consumption are not only an issue of individual health but also of public health. Successful enactment of public health policies directed at reducing antibacterial dispensing rates may rely in part on the ability of individuals and groups to obtain, process, understand, evaluate and act on information needed to make decisions that benefit the individual and the community,[36] allowing collectivist and long-term values to outweigh individualist short-term decision-making. It is possible that education enables an understanding of the individual and family as being embedded in society, such that individual decisions on antibacterial treatment are more likely to be made within the framework of a greater public health concern.

The Norwegian healthcare system provides universal healthcare access, and health inequalities in care utilisation have diminished over time.[37] Need-adjusted socioeconomic differentiation in healthcare usage has empirically been observed mostly in the use of private medical specialists and hospital outpatient care.[38] However, these observations do not necessarily include all differentiation in healthcare usage in Norway, such as potential geographic variations, and, importantly, these studies do not include parental healthcare seeking. If parental healthcare seeking translates to paediatric healthcare seeking, healthcare usage may, hypothetically, not be socially determined in volume, but rather in kind. People from advantaged socioeconomic backgrounds may interact and use healthcare inputs more efficiently, thus achieving the same amount of health investment with less healthcare services. They may also consider the potential consequences of antibacterial use more frequently, driving the dispensing rate downward.[5]

Importantly, children are themselves not actors in this framework. Decisions on treatment are made by physicians and parents, which suggests that the healthcare provided to children is dependent on parental socioeconomic status and how they seek healthcare for their children as well as the physician's prescribing habits and responses to different individuals and social groups. Several studies have identified an association between the high use of antibacterials in young children and an increased risk of chronic disease development later in life,[31 39–43] so optimising prescribing practices would seem important for reducing health inequalities in future generations.

Area-level strategies, as opposed to national-level strategies, for antimicrobial stewardship have been suggested in other countries[10]; given the local and regional variations in dispensing rates and reduction trends in Norway, we agree with previous authors[19] that effective antimicrobial stewardship requires that the issue be addressed from a multilevel systems perspective and that social, structural and cultural determinants also be considered

Table 2 Total dispensed DDD per 1000 children by ATC J01 subgroups

Year	J01A	J01C	J01D	J01E	J01F	J01G	J01M	J01X
2006	0.4	1009.1	19.9	77.9	526.2	7.6	1.0	17.4
2007	0.3	923.1	16.3	58.2	453.9	2.9	1.0	11.9
2008	0.2	1158.4	19.8	73.6	504.3	9.2	0.9	13.0
2009	0.2	1057.2	18.4	69.5	418.3	6.9	0.5	10.1
2010	0.2	1296.7	22.5	74.6	502.5	0.7	0.8	9.8
2011	0.1	1170.5	21.7	70.1	566.4	2.7	1.3	8.0
2012	0.4	1195.9	17.0	68.1	484.1	1.1	1.3	7.3
2013	0.4	1001.6	20.9	66.7	355.6	0.9	2.0	5.6
2014		1104.1	24.2	71.2	367.3	1.3	1.6	7.4
2015	0.1	965.6	21.8	67.1	299.9	0.9	1.3	8.7
2016	0.0	911.2	20.1	58.3	260.8	2.0	1.8	5.2

DDD, defined daily dose; J01A, tetracyclines; J01C, beta-lactam antibacterials, penicillins; J01D, other beta-lactam antibacterials; J01E, sulfonamides and trimethoprim; J01F, macrolides, lincosamides and streptogramins; J01G, aminoglycoside antibacterials; J01M, quinolone antibacterials; J01X, other antibacterials.

when implementing policy at the local administrative level. The overall responsibility for health policies in Norway lies with the National Ministry of Health, and stewardship of antimicrobial resistance in Norway relies on existing administrative structures of disease prevention and control, with sectoral operative responsibility and weak coordination mechanisms.[44] National political strategies do target primary healthcare services at the municipal level, but the need for and potential drivers of antibacterial treatment may vary between municipalities. We expect the efficacy of national policies for reducing antibacterial dispensing rates to partially depend on the local population's socioeconomic composition.

Strengths, limitations and methodological considerations

Unlike several authors who have applied indexed deprivation measures containing a variety of deprivation indicators, we focused on education specifically because it is a common component of deprivation indexes, which present a trade-off between interpretation and capturing a holistic concept of deprivation. It is, thus, unclear which features of such deprivation indexes drive empirical variations in dispensing rates, and translating theoretical mechanisms from the individual level to aggregate statistics then becomes even more challenging due to the number of dimensions in such indexes. The effects of income and occupation deprivation have been studied separately,[4] but no such analysis has been performed using an education indicator. Education is a key socioeconomic characteristic for health determinants, and by investigating education specifically, our results are more readily interpreted and more clearly relatable to the specific mechanisms discussed in the literature.

A strength of this study is the completeness of the dispensing rate metric. The NorPD contains all prescriptions dispensed in the period under examination, excluding usage in hospitals. We argue that this has two advantages. First, we expect education to matter more in the context of primary healthcare, because parents are active participants in healthcare decision-making, and second, the primary healthcare service is administered at the municipal level in Norway. Observed trends are, therefore, likely to be a result of local community needs and behaviours and local decision-making processes.

A limitation of this study is the lack of information on the geographical burden of disease, although regional differences in dispensing rates are unlikely to be explained by differences in the severity and density of infections and more likely to be related to differences in medical practices.[9] A Welsh study similarly found no support for regional differences in prescriptions being explainable by chronic conditions in the adult population.[3] Indeed, if the entire variance could be explained by the burden of infections, the implication would be that infections requiring antibacterial treatment are geographically unequally distributed, even between paediatric patients.

Another limitation is the limited inferences that can be made regarding individual outcomes based on aggregate

Table 3 Pooled statistics, including summary statistics for yearly observations for all municipalities, before centring

Statistic	N	Mean	Standard deviation	Minimum	Maximum
Dispensed Rx/100 children	4519	29.7	16.3	0.9	104.9
Education	4515	21.2	5.9	9.1	51.9
Population	4519	11885	35479	200	658390
Poverty	4518	10.0	2.4	3.7	21.8
Trend	4519	5.01	3.16	0	10
Travel time (sec)	426	1674	1882	182	13129

The variable Dispensed Rx/100 child is the dependent variable used in the model. Travel time is presented in decimal minutes and is time-invariant due to only being observed once. An extended table of summary statistics, including both centred and non-centred values, is available in the online supplemental appendix.

statistics. Further research is necessary to conclude an association between parental education, individual interactions with healthcare services and paediatric antibacterial dispensing rates in Norway.

CONCLUSION

Our analysis shows that the ability to reduce dispensing rates over time at the municipality level is associated with mean population levels of higher education. Local needs and potential root causes of health outcomes should be considered in antimicrobial stewardship to optimise prescription patterns, and attention should be paid to social demographics, like education, that may affect health behaviour, preferences and usage, which may help to further reduce dispensing rates in accordance with political goals.

Contributors SS conceptualised, designed and drafted the manuscript; prepared data; and performed the statistical analysis. KS contributed data. LS provided ethics approval and data from the prescription registry. SS, KS, AEE, and LS critically revised the manuscript. SS acts as the guarantor. All authors read and approved the final manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants but Regional Committees for Medical and Health Research Ethics Norway (2018/1021) exempted this study. Exempt from informed consent under the Norwegian Health Research Act. Data on prescriptions are retrospective and routinely collected through a national registry (making informed consent difficult), and the project was deemed valuable for the public. Individual prescription information was only used to calculate municipality dispensing rates and volume. The only information used relating to individual patients were their municipality of residence.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Data may be obtained from a third party and are not publicly available. Data on antibacterial dispensing can be obtained by application to a third party (The Norwegian Prescription Registry) and are not publicly available. Travel time data are available from the corresponding author upon request. Data collected from Statistics Norway are licensed under the Creative Commons Attribution 4.0 International (https://www.ssb.no/en/diverse/lisens) and are available from the corresponding author upon request.

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APPENDIX

Model description

The two-level linear growth curve model with a cross-level interaction effect with cluster-mean education is represented by the following equation:

$$\begin{aligned} L1: \sqrt{Y_{tj}} &= \beta_{0j} + \beta_{1j}T_{tj} + \beta_2 EDU_{tj}^{CWC} + \beta_3 lnPOP_{tj}^{CWC} + \beta_4 POV_{tj}^{CWC} + \epsilon_{tj} \\ L2: \beta_{0j} &= \gamma_{00} + \gamma_{01}EDU_j^{CM} + \gamma_{02}lnPOP_j^{CM} + \gamma_{03}POV_j^{CM} + \gamma_{04}TR_j + \mu_{0j} \\ \beta_{1j} &= \gamma_{10} + \gamma_{11}EDU_j^{CM} + \mu_{1j} \end{aligned}$$

Error terms are all assumed normally distributed:

$$\begin{aligned} \epsilon_{tj} &\sim \mathrm{N}(0, \sigma_\epsilon^2) \\ \mu_{0j} &\sim \mathrm{N}(0, \sigma_{\mu_0}^2) \\ \mu_{1j} &\sim \mathrm{N}(0, \sigma_{\mu_1}^2) \end{aligned}$$

Consulting the $L1$ part of the equation: β_{0j} are random intercepts, $\beta_k X_{tj}^{CWC}$ are the fixed time-variant coefficients where variables are centered-within-cluster, $\beta_{1j}T_{tj}$ is a time-variant trend variable where the first year is set to 0, and ϵ_{tj} is the level-1 error term. In the $L2$ part of the equation, γ_{00} is the mean municipal level intercept, $\gamma_{0k}X_j^{CM}$ are coefficients for level 1 covariate cluster-means (CM), $\gamma_{04}TR_j$ is a coefficient for median travel time to nearest pharmacy, while μ_{0j} is the intercept variance component. The linear trend variable is modeled as a random effect with μ_{1j} variance component $\gamma_{11}EDU_j^{CM}$. $\beta_2 EDU_{tj}^{CWC}$ is a cross-level interaction between the cluster-mean education level across the time-period and the random linear trend. The term $\beta_2 EDU_{tj}^{CWC}$ was removed in the final model to address the issue of simultaneous growth.

Table A1: Model 1 includes the time-variant education predictor, model 2 is the same as the in-text model. This table aims to show the consequences of simultaneous growth on the estimated trend coefficient and confidence intervals.

	$\sqrt{Dispensed\ prescriptions\ per\ 100\ children}$	
	Model 1	Model 2
Level 1		
Trend	−0.015 (−0.050, 0.019) [.385]	−0.053 (−0.066, −0.039) [<.001]
Poverty	−0.098 (−0.125, −0.071) [<.001]	−0.098 (−0.125, −0.070) [<.001]
Population (ln)	1.562 (0.210, 2.914) [.024]	1.265 (−0.061, 2.592) [.062]
Education	−0.069 (−0.127, −0.010) [.021]	
Level 2		
Education	-0.004 (−0.029, 0.021) [.751]	−0.002 (−0.027, 0.023) [.892]
Population (ln)	0.409 (0.292, 0.527) [<.001]	0.408 (0.290, 0.525) [<.001]
Poverty	−0.085 (−0.130, −0.040) [<.001]	−0.085 (−0.130, −0.041) [<.001]
Travel	−0.0003 (−0.0004, −0.0003) [<.001]	−0.0003 (−0.0004, −0.0003) [<.001]
Trend×Education (L2)	−0.003 (−0.005, −0.0005) [.019]	−0.0034 (−0.006, −0.001) [.005]
Intercept	5.271 (5.072, 5.471) [<.001]	5.459 (5.340, 5.578) [<.001]
Var. Comp.		
Std. Dev. μ_1	.0929	.0927
Std. Dev. μ_0	1.0912	.8647
Misc.		
ρ Comp. Symm.	.000	.000
Groups	426	426
Observations	4,499	4,503
Log Likelihood	−6,431.018	−6,442.764
Akaike Inf. Crit.	12,892.04	12,913.53
Bayesian Inf. Crit.	12,988.21	13,003.3
Note:	95% CI in parentheses. P-values in square brackets.	

Simultaneous growth and MLM interpretation under centering scheme

Model 1 includes all level 1 covariates. Model 2 excludes the group-mean centered education (L1) covariate due to simultaneous growth issues resulting in collinearity between L1 education and trend.

This contrast table shows the effect of simultaneous growth on estimated parameters. The only difference between the models is the removal of the L1 group-mean centered education indicator. Confidence intervals are shown in parentheses.

Group-mean centering level 1 covariates leads to orthogonal relationships between levels; the correlations between level 1 and level 2 covariates are equal to 0. In a model without the uncentered trend variable, excluding level 1 coefficients would not affect level 2 estimates under group-mean centering. In fact, the estimates would be the same regardless of whether level 1 covariates were even in the model [30]. However, since the trend variable is *not* centered, some correlation will exist between levels through correlation with the trend variable, explaining the minor changes in level 2 coefficients. These changes are unsubstantial and only result in minor changes in L2 estimates.

Simultaneous growth leads to a very simple issue of near perfect collinearity between L1 education and the trend variable. This is the reason for the dramatic change in the trend coefficient size and confidence interval. Simply put, the trend effect in model 1 is biased due to collinearity with the L1 education covariate. While there are ways to deal with this problem through *multivariate* growth curve modeling [32], we are primarily interested in the cross-level interaction effect between education traits and the random trend. As such, we prefer the more parsimonious modeling option removing the cluster-mean centered education variable from the level 1 part of the equation.

Interpreting coefficients under centering scheme

Centering and cross-level interactions changes the interpretation of certain coefficients. We base the interpretation on model 2 and focus on three main coefficient interpretations a) the main trend effect and its variance, b) the main trait education effect and c) the cross level interaction term.

Due to grand-mean centering L2 covariates and the inclusion of an interaction term, the main trend effect ($-.015$) is interpreted as the expected square root dispense rate trend for municipalities with a mean level of trait education (21.15%), *ceteris paribus*. This is a random coefficient, and its random parameter μ_1 suggests that the standard deviation from the fixed term is equal to $.919$. The main education effect ($-.002$) is the expected effect of education at $T = 0$ (2006, trend is not centered). This is clearly shown by the very similar intercepts in figure 2 and 3. Lastly, the interaction term (-0034.) is the expected decrease in trend for every pp increase in education traits. This model is the basis for figures 2 and 3.

For other L1 coefficients (sans the trend coefficient), a one-unit increase entails a one unit change from a covariates given group mean. The coefficient is thus the average effect of a one unit increase from a given group mean, *ceteris paribus*.

Centering and growth

Notably, we choose not to center the level 1 trend variable for two reasons; firstly, the panels are only slightly imbalanced. Centering the trend variable on the group means practically results in a grand mean centered trend variable (correlation with uncentered trend indicator: $r = .97$), with

no real consequences to the coefficient estimates. The only consequence is on the intercepts and the intercept variance due to the zero point being established in 2011 for all but a few groups. Secondly, the model is a linear random growth curve model. Centering the trend covariate is more of an issue in situations where a polynomial growth curve might be fitted.

Intercept and slope correlation

Intercepts and slopes are negatively correlated at $r = -.597$. This is a natural consequence of bounded data; dispensing rate cannot be less than 0. Municipalities with low starting dispensing rates will naturally not be able to reduce dispensing rates as much as those with higher starting dispensing rates. This is of no particular concern for estimating the interaction term; indeed, the non-significant main education coefficient implies that the intercept variance is not explained by mean population education levels. This is also clear when investigating figure 2 in the main text.

SUPPLEMENTARY FIGURES AND TABLES

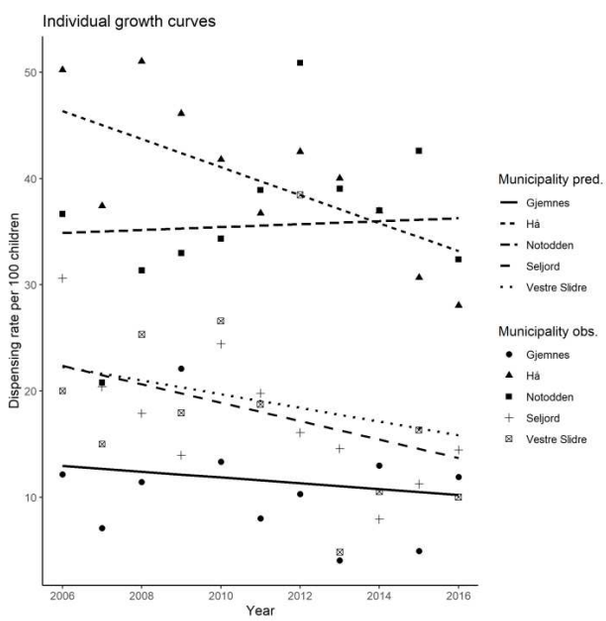

Figure A1: *Linear growth curve predictions and observations from a simple random trend null-model for five random municipalities. Municipalities were randomly sampled from a strata of slope quantiles to ensure that slope variance was represented in the figure. Note that the Y-axis is scaled by min-max observations in the subsample, not the entire distribution.*

Table with transformed and untransformed dispense rates

Table A2: Multilevel growth curve models. Both models include all covariates. Model 1 uses the square-root transformed dispense rates as outcomes. This model is used for prediction (figures 2 and 3) and evaluation of statistical significance. Model 2 uses the dispense rate as the outcome.

	$\sqrt{\text{Dispensed Rx per 100 children}}$	Dispensed Rx per 100 children
	(1)	(2)
Level 1		
Trend	−0.053 (−0.066, −0.039) [<. 001]	−0.608 (-.750, -.466) [< . 001]
Poverty	−0.098 (−0.125, −0.070) [<. 001]	−1.061 (-1.352, -.769) [< . 001]
Population (ln)	1.265 (−0.061, 2.592) [.062]	13.980 (.278, 27.683) [.046]
Level 2		
Education	−0.002 (−0.027, 0.023) [.892]	0.026 (-.239, .291) [. 848]
Population (ln)	0.408 (0.290, 0.525) [< .001]	3.983 (2.767, 5.199) [< .001]
Poverty	−0.085 (−0.130, −0.041) [< .001]	−0.845 (-1.311, -.379) [. 001]
Travel	−0.0003 (−0.0004, −0.0003) [< .001]	−0.003 (-.003, -.002) [< . 001]
Trend × Education (L2)	−0.0034 (−0.006, −0.001) [.005]	−0.041 (-.066, -.017) [. 001]
Intercept	5.459 (5.340, 5.578) [< .001]	32.689 (31.425, 33.952) [< .001]
Var. Comp.		
Std. Dev. μ_1	.0927	.918
Std. Dev. μ_0	.8647	11.54
Misc.		
ρ Comp. Symm.	.000	.000
Groups	426	426
Observations	4,503	4,503
Log Likelihood	−6,442.764	−17,097.230
Akaike Inf. Crit.	12,913.53	34,222.460
Bayesian Inf. Crit.	13,003.3	34,312.240
Note:	*p<0.05; **p<0.01; ***p<0.001	
	95% CI in parentheses. P-values in square brackets.	

Dependent variable distribution before and after square root transformation

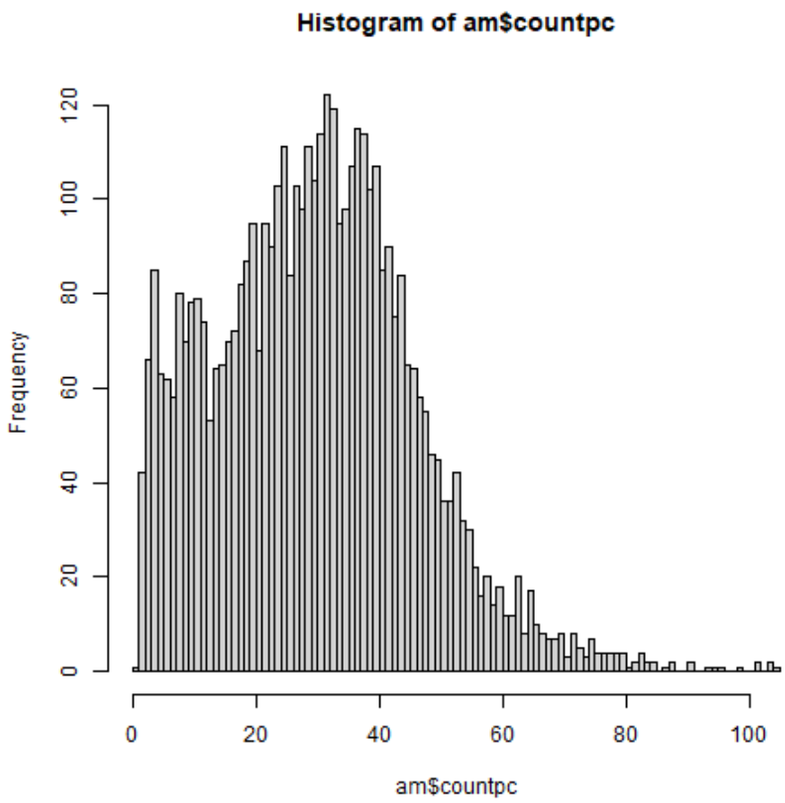

Figure A2: Dispense rate distribution before square root transformation. The distribution is closer to a Poisson distribution, due to the natural bounds of the data.

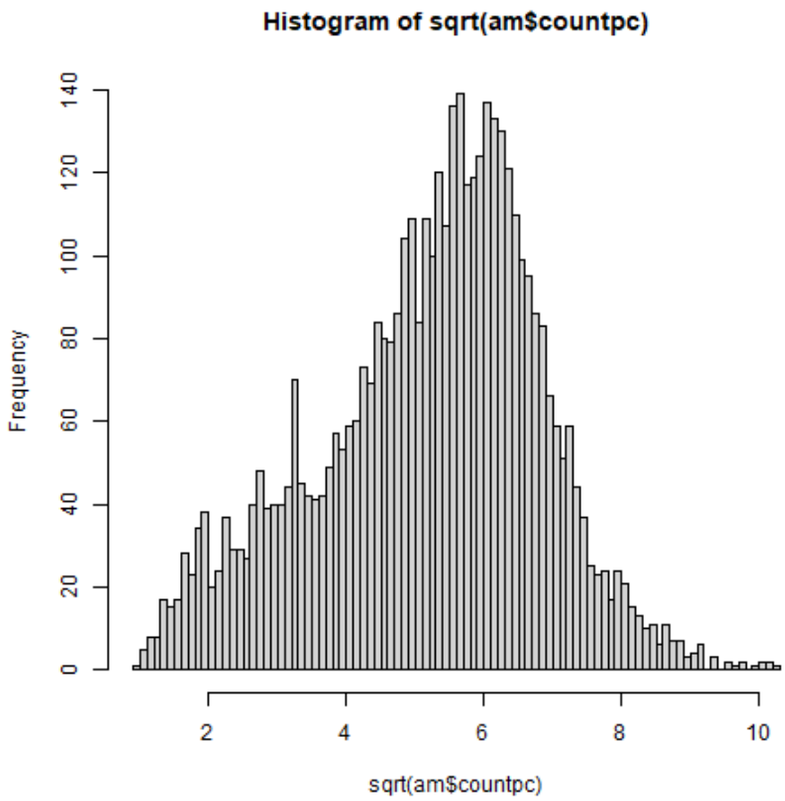

Figure A3: Dispense rate after square root transformation. Where the log-transformation (not shown) aggressively overcorrects the issue, leading to a worse fit than the untransformed version of the model, the square root transformation only moderately corrects the distribution, making residuals more well-behaved than the untransformed model. We emphasize that we performed this transformation to solve a statistical issue particularly present when investigating the residuals vs. the fitted values, and as such were guided by the data rather than theory. However, as the prediction plots, significance tests, and coefficients show, these modeling changes do not affect results in a significant way.

Residual plots main model

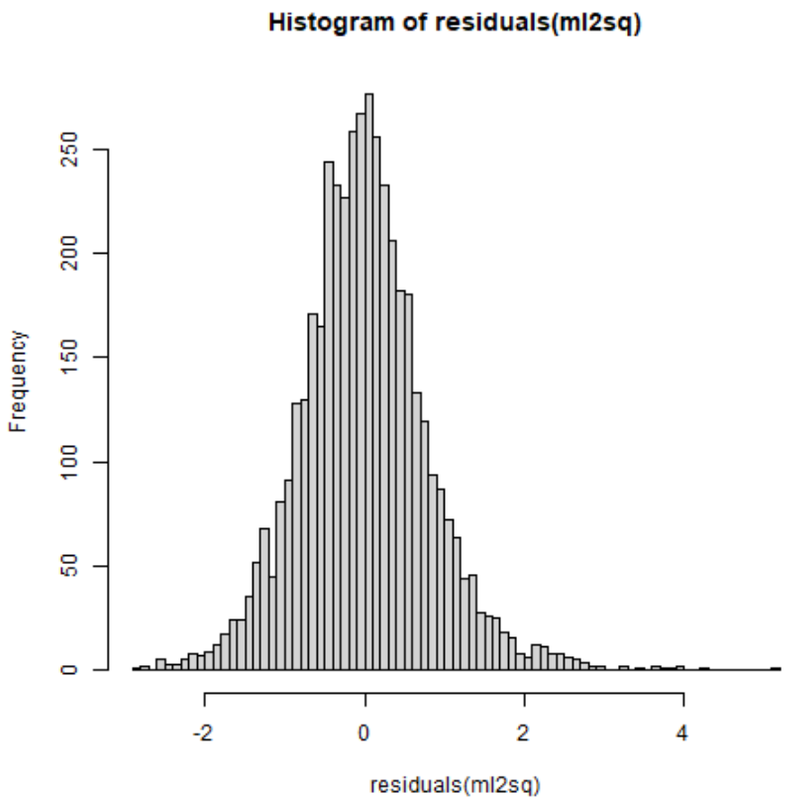

Figure A4: Level 1 Residual distribution after square root transformation of the dependent variable. While a marginally longer tail on positive residuals, we find no particular issues with this distribution.

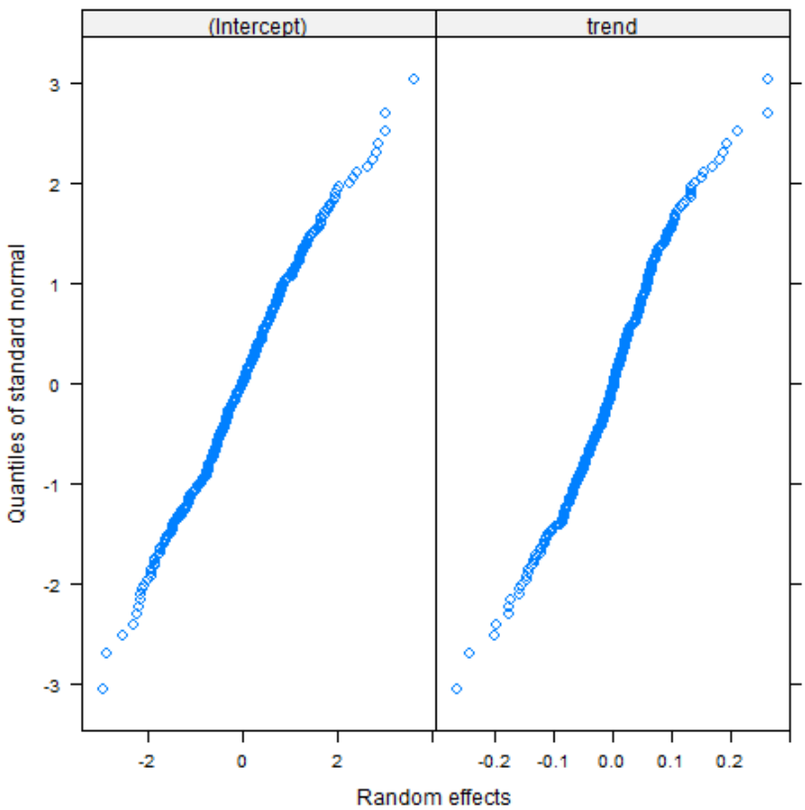

Figure A5: QQ-plot of the random terms in the model. We find that these are approximately normally distributed.

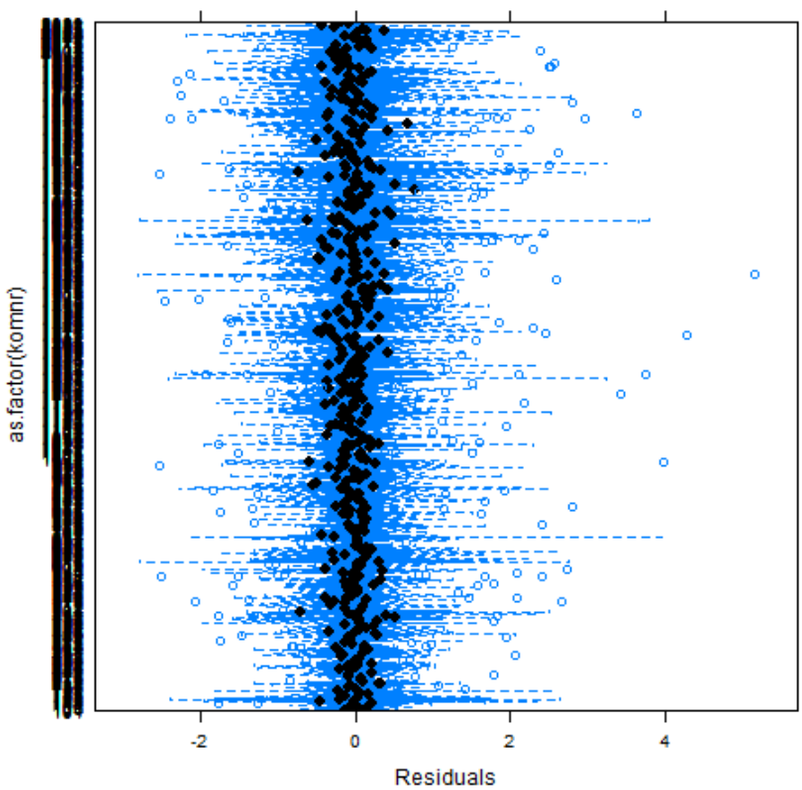

Figure A6: Level-1 residuals by municipality. Residuals seem overall to be centered at 0 with random deviation from this mean. Some differences in variance between municipalities is expected, as the number of repeat observations is relatively small (11).

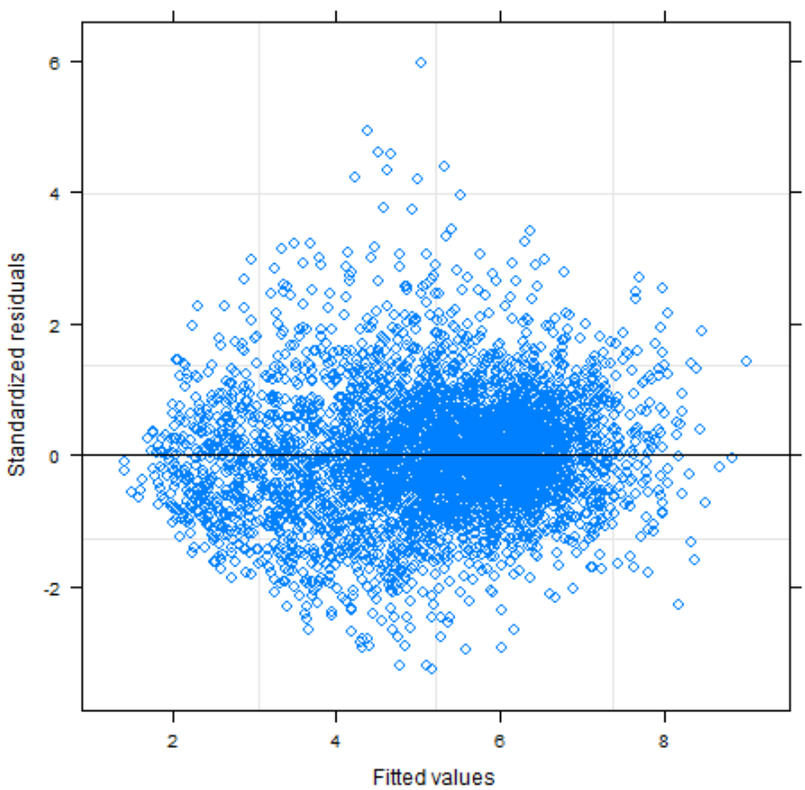

Figure A7: Standardized residuals vs. fitted values plot. We saw some problems with heteroskedasticity in the unadjusted model. While logarithmic transformation aggressively overcorrected the issue, the square root transformation adjusts for the moderate skewness and provides confidence to estimated standard errors.

Full version of summary statistics table

Statistics	N	Mean	St. Dev.	Min	Max
Pooled					
Dispensed Rx/100 chld.	4,519	29.7	16.3	0.9	104.9
Education	4,515	21.2	5.9	9.1	51.9
Population	4,519	11,885	35,479	200	658,390
Poverty	4,518	10.0	2.4	3.7	21.8
Within					
Dispensed Rx/100 child	4,519	0.00	9.58	−40.38	74.42
Education	4,515	0.00	1.87	−5.25	5.97
Population	4,519	0.00	2,180	−60,394	59,5842
Poverty	4,518	0.00	1.07	−3.46	5.76
Between					
Dispensed Rx/100 chld.	428	29.0	13.5	2.8	70.3
Education	428	21.0	5.6	11.2	48.2
Population	428	11,505	34,795	212	598,805
Poverty	428	10.0	2.2	5.1	18.6
Travel (sec.)	426	1,674	1,882	182.0	13,129

Table A3: Summary statistics grouped by levels. Pooled statistics include summary statistics for yearly observations for all municipalities before centering. The dependent variable. The within section shows descriptive statistics for all cluster-mean centered covariates, that is the level 1 parameters in the model. Note the mean 0 ensuring no correlation between level 1 and level 2 covariates. The between section represents the level 2 variables used in the model. These are 428 cluster-means for all covariates excluding travel times, due to municipality mergers before data collection.