UiT The Arctic University of Norway

Faculty of Biosciences, Fisheries and Economics
School of Business and Economics

Grandparents Matter –

Multigenerational transmission of health and health behaviors

Emre SARI

A dissertation for the degree of Philosophiae Doctor (PhD) May 2023

Note: My grandmother, Hafize Sarı.
Photo by Fikriye Görgün.

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by

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To my late cousin Cem Sultan Sarı...

Dünyada herşey için, medeniyet için, hayat için, başarı için, en hakiki mürşit ilimdir, fendir.

Science is the most real guide for civilization, for life, for success in the world.

Gazi Mustafa Kemal Atatürk

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Emre Sarı
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Abstract

Health is an essential component of human capital, and health inequalities are a major issue. While substantial research on the intergenerational transmission of health and health behaviors has been conducted, the questions whether and how grandparents' health affects grandchildren remain underinvestigated. This thesis aims to better understand health inequality mechanisms by analyzing multigenerational health and risky health behavior transmission. I employ methods to further elucidate multigenerational effects using the historical multi-generational Rendalen database, covering the 18th and 19th centuries, and the population-based 1974–2016 Tromsø Study. The first paper discusses the effect of grandmothers' economic hardships on grandchildren's health. Economic hardships during pregnancy demonstrate transgenerational health effects that continue through generations by social class. The second paper investigates whether smoking in earlier generations is causally related to that in subsequent generations and differential maternal versus paternal grandparent effects. Maternal grandparents' smoking behavior directly reduce the probability of grandchildren smoking, whereas intergenerational transmission increases that probability. The third paper investigates the impact of intergenerational transmission of child neglect by grandparents on the long-term mental health of their grandchildren. The results implicate that the likelihood of depression in grandchildren is specifically linked to neglectful parenting by maternal grandparents, taking into account whether their own parents exhibited neglectful behaviors. In conclusion, further investigation of the effects of cultural inheritance from grandparents on future generations' health is important. The results suggest that policymakers should reevaluate and expand health policies to include grandparents.

Keywords: Multigenerational effect, Intergenerational transmission, Health shocks, Grandparental investments, Norway

JEL Classification: D64, I10, I12, I14, I18

Abstrakt

Helse er en essensiell komponent av menneskelig kapital, og helseulikheter er et betydelig problem. Mens det er gjort omfattende forskning på intergenerasjonell overføring av helse og helseatferd, er spørsmålet om hvordan besteforeldres helse påvirker barnebarna underundersøkt. Denne avhandlingen har som mål å bedre forstå mekanismene for helseulikheter ved å analysere flergenerasjonell helse og overføring av risikofylt helseatferd. Jeg bruker metoder for å belyse flergenerasjonelle effekter ved å benytte den historiske flergenerasjonelle Rendalen-databasen som dekker 18. og 19. århundre, samt den befolkningsbaserte Tromsø-studien fra 1974 til 2016. Den første artikkelen diskuterer effekten av bestemødres økonomiske vanskeligheter på barnebarnas helse. Økonomiske vanskeligheter under svangerskapet viser transgenerasjonelle helseeffekter som varer gjennom generasjoner etter sosial klasse. Den andre artikkelen undersøker om røyking i tidligere generasjoner er årsaksmessig relatert til røyking i påfølgende generasjoner og differensielle effekter av mors- og fars-siden av besteforeldre. Røykeatferd besteforeldre direkte sannsynligheten for at barnebarn røyker, mens intergenerasjonell overføring øker denne sannsynligheten. Den tredje artikkelen undersøker effekten av intergenerasjonell overføring av omsorgssvikt fra besteforeldre på barnebarns langsiktige psykiske helse. Resultatene viser at sannsynligheten for depresjon hos barnebarn er spesifikt knyttet til omsorgssvikt fra bestemødres side, med hensyn til om deres egne foreldre utviste omsorgssvikt. Konklusjonen er at det er viktig med ytterligere undersøkelse av effektene av kulturell arv fra besteforeldre på fremtidige generasjoners helse. Resultatene antyder at beslutningstakere bør revurdere og utvide helsepolitikken for å inkludere besteforeldre.

Nøkkelord: Fleregenerasjonseffekt, Intergenerasjonell overføring, Helsesjokk, Besteforeldreinvesteringer, Norge
JEL klassifisering: D64, I10, I12, I14, I18

Özet

Sağlık insan sermayesinin temel bir bileşenidir ve sağlık eşitsizlikleri önemli bir sorundur. Nesiller arası sağlık ve sağlık davranışlarının aktarımı üzerine önemli araştırmalar yapılmış olsa da, büyükanne veya büyükbabaların sağlığının torunları nasıl etkilediği sorusu henüz yeterince araştırılmamıştır. Bu tez, çok kuşaklı sağlık ve riskli sağlık davranışlarının aktarımını daha iyi anlamak için sağlık eşitsizliği mekanizmalarını analiz etmeyi amaçlamaktadır. 18. ve 19. yüzyılı kapsayan tarihsel çok kuşaklı Rendalen veritabanı ve nüfus tabanlı 1974-2016 yıllarını kapsayan Tromsø Çalışması ile çok kuşaklı etkileri daha iyi açıklığa kavuşturmak için yöntemler kullanmaktadır. İlk makale, büyükannelerin ekonomik zorluklarının torunların sağlığı üzerindeki etkisini tartışmaktadır. Gebelik sırasındaki ekonomik zorluklar, sosyal sınıfa göre kuşaklar boyunca devam eden nesiller arası sağlık etkilerini göstermektedir. İkinci makale, daha önceki kuşaklarda sigara içmenin, sonraki kuşaklarda sigara içme ile nedensel olarak ilişkili olup olmadığını ve anneyle baba soylu büyükanne veya büyükbabaların etkilerini araştırmaktadır. Anne soylu büyükanne veya büyükbabanın sigara içme davranışı, torunların sigara içme olasılığını doğrudan azaltırken, kuşaklar arası aktarım bu olasılığı arttırmaktadır. Üçüncü makale, büyükanne veya büyükbabanın çocuk ihmalinin kuşaklar arası aktarımının torunların uzun vadeli ruh sağlığı üzerindeki etkisini araştırmaktadır. Sonuçlar, torunlardaki depresyon olasılığının, kendi ebeveynlerinin ihmalci davranışlar sergileyip sergilemediği de dikkate alınarak, anne soylu büyükanne veya büyükbabaların ihmalci ebeveynlikleriyle özellikle ilişkili olduğunu göstermektedir. Sonuç olarak, büyükanne veya büyükbabaların gelecek kuşakların sağlığı üzerindeki kültürel mirasının etkilerinin daha fazla araştırılması önemlidir. Sonuçlar, politika yapıcıların büyükanneleri de içeren sağlık politikalarını yeniden değerlendirmesi ve genişletmesi gerektiğini göstermektedir.

Anahtar kelimeler: Çok kuşaklı etki, Kuşaklar arası aktarım, Sağlık şokları, Büyükannelerin yatırımları, Nineler ve dedeler, Norveç,
JEL Sınıflandırması: D64, I10, I12, I14, I18

List of Papers and Contributions

Name of candidate: Emre Sarı
The following papers are included in my PhD thesis:

Paper I: Sari, E., Moilanen, M., Leikny Sommerseth, H. (2020). Transgenerational health effects of in Utero exposure to economic hardship: Evidence from preindustrial Southern Norway. *Economics and Human Biology, 43.* https://doi.org/10.1016/j.ehb.2021.101060
Paper II: Sari, E., Moilanen, M., Lindeboom, M. (2023). Role of Grandparents in Risky Health Behavior Transmission: A Study on Smoking Behavior in Norway.– *advanced manuscript.*
Paper III: Sari, E., Moilanen, M., Lindeboom, M. (2023). Long-term Effects of Grandparental Child Neglect on Grandchildren's Mental Health: A Three-generation Study.– *advanced manuscript.*

Contributions

	Paper I	**Paper II**	**Paper III**
Idea	ES, MM	ES, MM	ES, MM, ML
Conceptualization	ES, MM	ES, MM, ML	ES, MM
Methodology	ES, MM	ES, ML	ES
Data curation	ES, HS	ES	ES
Formal analysis	ES	ES	ES
Writing original draft	ES, HS*	ES	ES
Writing – Review and Editing	ES, MM, HS	ES, MM, ML	ES, MM
Visualisation	ES	ES	ES

ES = Emre Sari; MM = Mikko Moilanen; HS = Hilde Leikny Sommerseth; ML = Maarten Lindeboom *The section of the article titled "2. Rendalen" was written by HS

Other declarations
Conflict of interest: *None.*

Contents

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Introduction

Globalization and technological and cultural advancements have all contributed to an era in which inequality has become a much more pressing issue, particularly regarding health inequality. Domestic economies are becoming more international and integrated as cross-border trade with other countries occurs daily; thus, the way the world works is changing with this rapidly growing integration ("One World", 1997). The introductory chapter of Health and Inequality (O'Donnell et al., 2015) poses a central question for economists: what motivates economists to consider health inequalities beyond the confines of their field? We know that health is a key indicator of human capital quality and a major contributor to economic well-being (Schultz, 1961). Furthermore, policymakers and public health practitioners aim to increase heath in society and decrease health inequalities.

Health inequalities are a major problem for everyone, regardless of their socioeconomic status; i.e., health inequalities do not affect only those in lower socioeconomic positions on the basis of education, income, occupation, or geographic diversity (Currie, 2009; Woodward & Kawachi, 2000). Long-term, health inequality wastes public investments and forces policymakers to increase public spending to maximize utility (Maria et al., 2017). As a result, beyond the unfavorable economic outputs, nobody benefits and everyone is affected by the harmful consequences (Woodward & Kawachi, 2000). Johnson & Schoeni (2011) state that health is an essential component of human capital. Additionally, Barro (2013) claims that initial health is a more reliable indicator of future economic growth than initial education levels.

According to human capital theory, a person's level of education and state of health are essential factors in endowments (Behrman et al., 1994). One of the central questions is what role one's family background plays in shaping one's adulthood human capital. The answer to this issue has long piqued the curiosity of social scientists since it may shed light on the extent to which social inequality is perpetuated from one generation to the next (Lundborg et al., 2018). A significant body of research has been conducted to address this problem by concentrating on the intergenerational transmission of income and socioeconomic status (see,

e.g., Adermon et al. (2021); Barone & Mocetti (2021); Black et al. (2020)). Some of this research employs twin (see, e.g., Andersen (2021); Heckley et al. (2016)) and adoption (see, e.g., Björklund et al. (2006); Lundborg et al. (2018)) designs to overcome the sensitivity to the interaction between *nature* and *nurture*.

We now have a better grasp of how human capital is passed down through generations due to this literature. The estimates mainly consider factors such as the fact that the health of the mother and the father has a significant consequence on their children's health. The importance of the intergenerational transmission of health and health behaviors from one generation to the next has been highlighted in an increasing body of research in recent years (see, e.g., Classen & Thompson (2016); Göhlmann et al. (2010); Halliday et al. (2020)). Recent studies, including those conducted by Almond et al. (2018) and Currie (2009) and others, have shed light on the significance of one's early life in determining one's health in later years. Individuals' health may also be related to their peers (see, e.g., Triyana & White (2022); Zhou & Wang (2022)), neighbors (see, e.g., Grossman & Khalil (2022); Sari et al. (2021a)), exogenous shocks (see, e.g., Aaskoven et al. (2022); Avdic et al. (2021)), and even macro variables like globalization (see, e.g., Schrecker et al. (2008)), suggesting the involvement of other environmental mechanisms.

However, our understanding of how parental health and health behaviors affect their offspring remains vague. In other words, what are the exact mechanisms involved in health transmission from generation to generation? Looking slightly further, does grandparent health impact grandchildren in the same way that parents affect children—either directly from the grandparents to the grandchildren, indirectly through the parents, or both? In this thesis, I contribute to the literature on the mechanisms underlying the transmission of health and health behaviors across more than two generations by utilizing the *Rendalen database*, comprising rich individual-level data from the 18th and 19th centuries, and the population-based *Tromsø Study*, which spans 1974 to 2016.

The rest of the thesis is structured as follows. Chapter 1 discusses the determinants of social inequalities in health and the evolution of the relevant literature. A discussion of health across generations is included in Chapter 2, along with an overview of the multigenerational transmission of health and health behaviors. This section then focuses on an economic theory and finally discusses the health inequalities in Norway. In Chapter 3, an overview of this thesis is presented, along with the aims and a conceptual framework that demonstrates how the covered studies relate to each other. Within this section, I present a synthesis of the enclosed Papers, including relevant data and methodological challenges associated with them. The overall contributions of this thesis and its political implications and limitations are

discussed in the Discussion. The research papers appended to this thesis are presented at the end.

1 Social Inequalities in Health

The Black Report (Townsend et al., 1982) was likely the first official attempt to explain increasing life expectancy gaps across different socioeconomic groups. According to this report, health inequalities have increased since 1948 in the United Kingdom. Numerous factors influence health and illness, including income, education, housing, nutrition, and employment. The Whitehall study (Marmot et al., 1984) was published after the Black Report. According to the Whitehall study, the death rate in the lowest grade levels was three times that in the highest grade levels. The lower grades have a higher prevalence of cardiovascular disease risk factors, including smoking and other coronary risk factors; however, these disparities account for only a portion of the mortality gap. In addition, the Whitehall study showed that such inequities are pervasive throughout society (Quesnel-Vallée & Jenkins, 2010). As a result, in the early 1980s, these two seminal studies on social inequalities in health drew attention to this area. These studies heralded the beginning of research that would rapidly spread and continues today.

Currently, one of the most significant challenges in public health in developed countries is health inequalities (Mackenbach et al., 2018). Health inequalities are unjust and detrimental to individuals, families, and society. Substantial developments have recently taken place in explaining these health inequalities across and within countries to better understand their underlying mechanisms and suggest better solutions. However, in-depth research into the root cause of this gap is required to develop more effective strategies. For instance, it is widely recognized that early interventions have a large and favorable impact on the outcomes of children's futures and that inadequacies in early childhood development can be linked to social inequalities in health (see, e.g., Conti et al. (2004); Currie (2020); Heckman &

Masterov (2007)). Now that demographic shifts, such as increased life expectancies, are in play, we must extend two-generational-focused research to include the grandparents to develop a comprehensive understanding of health capital effects.

Social inequalities in health are a dimensional concept referring to measurable quantities and a political concept showing moral responsibility to social justice (Kawachi & Subramanian, 2002). However, finding common ground for defining the straightforward meaning of social inequalities in health is laborious and complicated. For example, Kawachi & Subramanian (2002, p. 647) define health inequalities as "[. . .] the generic term used to designate differences, variations, and disparities in the health achievements of individuals and groups." In addition, Carter-Pokras (2002), shares the following four elements for defining "health disparities" commonly used in the USA for health inequalities: (1) environmental effects; (2) treatment access, utilization, and quality; (3) health status; and (4) a measurable health outcome that requires examination.

1.1 Determinants of health

Numerous determinants can influence one's health, and most are unlikely to be under the individual's direct control. To this end, disease causation models can never fully exclude the environment as a health determinant. Accordingly, macro-level factors such as unemployment rates and food availability are also included in the social determinants of health alongside micro-level elements, such as health-related behaviors and social support. Figure 1.1 is an excellent graphic representation of these determinants and is directly taken from Acheson (1998)'s report based on a working document by Whitehead & Dahlgren (1991). People are represented in the middle of the diagram, with age, sex, biological, and genetic features considered. It is generally accepted that the layers people are surrounded by are susceptible to change due to governmental initiatives. Individuals' own behaviors and lifestyles, such as smoking, eating habits, and physical activity, are included in the innermost layer (Quesnel-Vallée & Jenkins, 2010). These behaviors can be either good for or bad for health. The subsequent layer highlights that people do not exist independently of their surrounding social environment. Their connections with family, friends and members of their immediate network provide influences that can affect their health directly via associated stress or indirectly by the behaviors they adopt as a result of those interactions. This level and the ones it encloses are particularly relevant for understanding the impact of extended family members—or dynasty members—on health and health behaviors, including grandparents, great-grandparents, aunts, uncles, and cousins (Adermon et al., 2021). The third layer shows larger societal factors that may influence an individual's ability to maintain their health, such as living and working

conditions and welfare state provisions (or lack thereof) through education and health care. The top layer represents the socioeconomic, cultural, and environmental conditions as a whole.

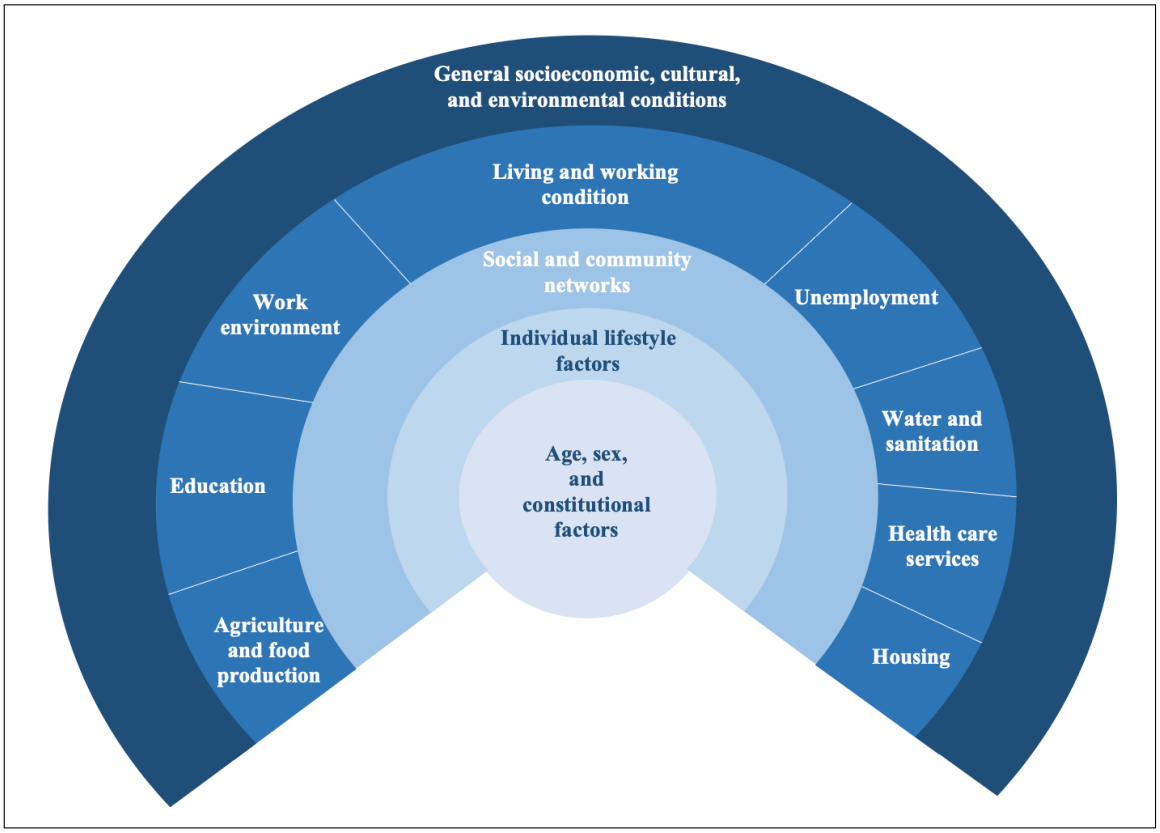

Figure 1.1 *"The main determinants of health"*
Source: Acheson (1998).

The Norwegian national perspective on health inequities is described in detail in the national strategy report (HOD, 2007). This report classifies the factors most widely acknowledged as determinants of health into five primary categories, collectively referred to as determinants of health. As in other studies, income, the conditions of childhood, work and the environment of work, health behaviors, and health services are considered factors that determine health.

Increasing evidence implies that income inequality is a major factor in health outcomes (Kinge et al., 2021; O'Donnell et al., 2015; Olsen et al., 2020). It cannot be denied that living in poverty (Bucci et al., 2019), working under adverse conditions (Belloni et al., 2022), having poor and unequally accessible health services (Cinaroglu & Çalışkan, 2022), and adverse health behaviors are detrimental to one's health. Overall, health outcomes, including mortality, morbidity, and quality of life measures, tend to worsen in tandem with decreasing material standards of living, as assessed by the aforementioned indicators. Contrary to

the often-repeated empirical evidence of these determinants, studies on the influences of extended family members on people's health and health behaviors have only recently been reported in the economics literature.

2 Health Across Generations

The intergenerational transmission of income and socioeconomic standing from one generation to the next has long been a focus of social scientists. This is because if these characteristics are sufficiently intergenerational, they would be incompatible with the premise that everyone should have the same opportunities in life (Ahlburg, 1998). The proposition that everyone should have the same opportunities in life is also valid for health and the intergenerational transmission of health. Studies on the relationship between health outcomes and a person's family history have increased in number and produced substantial evidence since the early 19th century. Most of these studies focused on a broader definition of health, including longevity and anthropometric outcomes such as height, weight, and body mass index.

Since Beeton & Pearson (1899), many have concentrated on longevity and estimated intergenerational correlations in life spans (see, e.g., Dalgaard et al. (2021); Lindeboom et al. (2010); Modin et al. (2009); Sari et al. (2021b). Galton (1886)) conducted one of the first intergenerational correlation studies using anthropometric measures. Galton found that the average height difference between a person's parents and the population mean equals two-thirds of the deviation of their parents from the population mean. Since, numerous further studies have been conducted to investigate the relationship between anthropometric measurements and overall health outcomes (see, e.g., Björkegren et al. (2022); Lahti-Pulkkinen et al. (2018); Lindeboom et al. (2009); Thomas (1994)). In addition, there is substantial methodological and quantificational variation in recent studies despite this relatively straightforward premise.

2.1 Multigenerational transmission of health and health behaviors

A recent shift in the focus of health economics to multigenerational relationships has occurred, namely, in the form of mathematical or empirical models highlighting the connection between grandparents and the long-term repercussions of that relationship. The pattern of inheritance,[1] intergenerational mobility, and extended family transmission of social and cultural capital over generations are all topics worth investigating when studying hereditary relationships, which are also relevant from a sociological perspective (Mare, 2011, 2014).

The modern nuclear family and dynasty structure has developed over the last century as a consequence of demographic fluctuations in mortality, fertility, and family formation (Song & Mare, 2019). Variations in these areas include when people get married and start families, how long people live, how much money and other resources they have, what kind of experiences they have, and how they are socialized (Coall et al., 2018). Other factors that contribute to these variations include the nature of people's relationships with their grandparents. In this context, Bengtson (2001) stresses that grandparents' duties in families were, are, and will continue to be vital despite varying demographic trends that have led to some forecasts of a decline in the value of grandparents' influence within the nuclear family in industrialized societies. Furthermore, Coall et al. (2018) summarize the various roles that grandparents play and the associated challenges within the context of the nuclear family. Some examples include families with many generations living under one roof, children with special needs, grandparents raising their grandchildren, and competing priorities between paid jobs, retirement, leisure activities, and parental care. Increased childcare intensity, the changing roles of grandfathers, and reduced institutionalized support for childcare have also been addressed.

Mare (2011) states that we should pay more attention to social connections beyond those between a parent and a child, particularly those that span three or more generations. The fields of sociology, economics, and evolutionary biology have all made significant strides toward a better understanding of these characteristics. Nevertheless, to date, they have done so mostly independently. Each field also explains the role of grandparents differently (Coall et al., 2018). In this thesis, I take an interdisciplinary approach to addressing the impact of grandparents on their grandchildren by using the literature and theories from various disciplines, including demography, population health, evolutionary biology, and economics.

[1] Similar to Stuhler (2012), I use the term "inheritance" in a wide sense. This means that it incorporates not just genetic inheritance but also other causal paths from parents to offspring, such as the method by which parents nurture their children.

2.2 Economic lens on multigenerational effects

In recent years, academics and policymakers have paid more attention to the extent to which human capital is transferred from generation to generation (Aaskoven et al., 2022). A person's health status is also an essential component of human capital and directly impacts both individual benefits and the overall functioning of the economy (Grossman, 1972). Therefore, social inequality and individual well-being are significantly impacted by the intergenerational transfer of health. To highlight the relationship between grandparents and their offspring and the long-term influence of this relationship, economists have switched their focus to multigenerational effects using mathematical or simulation models (see, e.g., Adermon et al. (2021); Barone & Mocetti (2021); Braun & Stuhler (2018); Solon (2018). In tandem, in-depth studies have recently been conducted on how the intergenerational transmission of health and health behaviors occurs through three or more generations (see, e.g., Björkegren et al. (2022); Lindahl et al. (2015); Maystadt & Migali (2021); Vanderweele (2016)).

Transfer between generations can take many forms, including inheritance, money and time. There is no universal economic model of investment by either parents or grandparents, and transfers may occur up or down the dynastic hierarchy (Coall et al., 2018). In this thesis, my interest concentrates on two possible transmission mechanisms: the direct effects that grandparents (G1) have on their grandchildren (G3) and the indirect effects that grandparents have on their grandchildren (G3) through their children (G2) (see, Figure 2.1). Solon (2018) states that the human capital production function incorporates the child's human capital endowment, regardless of the family's deliberate investment decisions. This endowment is associated over generations due to the inheritance of genetic features and cultural or environmental factors, such as the influence of parental role modeling. In addition, Heckman (2006) provides a summary of the evidence regarding early parental investments in their children and how they affect later-life human capital. The Heckman Curve highlights the fact that the life cycle is a dynamic process and that early investments have a greater effect than later ones.

Ashenfelter (1973) conducted one of the oldest studies on parental investment in economics. Ashenfelter portrayed children as home-produced durable assets from which their parents would consume a flow of services. Ashenfelter did this by using a household production function and postulates that increasing the number of children, referring to having more children, and increasing the quality of children, referring to enhancing the resource investment in existing children, are both replacements in the production function for child services that families make use of. Parents' well-being depends on the well-being of their

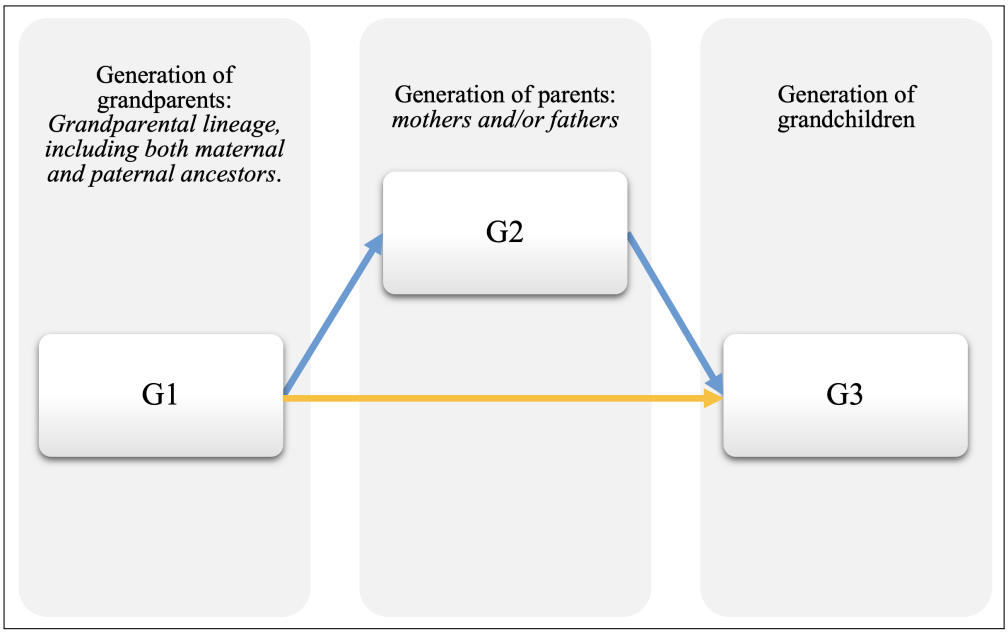

Figure 2.1 *Diagram showing the chronological order of the generations under study.*

Notes: This figure illustrates the general context of the studies in this thesis. Grandparents (G1), parents (G2), and grandchildren/offspring (G3) are the three focal generations. The inclusion of all grandparents and parents varies depending on the study setting. The light blue arrows represent the intergenerational transmission between generations. The direction of the purple arrow indicates the direct effect of grandparents on grandchildren.

children and grandchildren, as shown by Barro (1974) and Becker (1974). When determining the child's value to the parent, the utility function considers the child's potential lifetime value. According to this approach, parents provide resources to their offspring depending on the offspring's qualities, such as their ability and skill, and then employ the redistribution of resources more fairly among their offspring (Coall & Hertwig, 2011). Therefore, successive generations are connected by recursive acts of altruism; parents provide altruistic care for their children, who then pass on resources to their offspring.

One way to look at intergenerational transfers is as investments made by parents to secure their children's commitment to the family in the coming years. To improve the possibility that their children would support their parents in their time of need, parents often make capital investments in areas such as education, income, and health in the present and in the promise of an inheritance. One of the earliest studies on intergenerational transmission of health was conducted by Ahlburg (1998). According to Ahlburg, intergenerational correlations between education and health contribute to income and economic inequality across generations. People in families where ancestors had poor health or passed away at an early age are more likely to have an overly pessimistic estimate of their own morbidity and mortality and underinvest in human capital. In contrast, people in families with ancestors who were healthy and lived long lives may demonstrate the opposite tendency. The result of intergenerational transmission of health may reflect parental choices regarding health-related behaviors, unequal health

endowments, the nature of the health production function, or different positions in the labor market (Ahlburg, 1998). As a result, the outcomes of the health status of parents may have significant effects on the individual distributions of wages and income and affect the factors that determine the health of subsequent generations.

Studies that span many generations and analyze intergenerational interactions are rare in the economics literature, and most existing studies focused on the enduring influence of socioeconomic variables (see, e.g., Adermon et al. (2021); Braun & Stuhler (2018); Long & Ferrie (2018); Lundborg et al. (2018); Solon (2018)). Few studies have focused on health and health behavior transmission (see, e.g., Björkegren et al. (2022); Cook et al. (2019); Straatmann et al. (2021); van den Berg & Pinger (2016); Xu (2019)). Nevertheless, studies including more than two generations can be useful for distinguishing the effects of biological processes that occur by nature from the effects of environmental activities that occur by nurture.

As previously indicated, a wide range of empirical data supports the claim that health and social status inequalities tend to be passed down from generation to generation. One must continue to focus on the extent of causal links to understand the underlying processes of health and health behaviors. van den Berg & Pinger (2016) ask an influential question: does the existence of strong correlations between generations of health and socioeconomic status indicators mean that human capital shocks may be passed down from parents to children and even grandchildren? Early and profound influences caused by the conditions in the grandparents' generation that arise from adverse circumstances, such as early life shocks, parental behaviors, inadequate prenatal care, and poor childhood conditions, can be an important cause of poor health in adulthood. For instance, recent studies attempting to answer this question of causality have focused on the long-term effects of exogenous shocks (see, e.g., Almond & Currie (2011a); Almond et al. (2018); Avdic et al. (2021); Darden & Gilleskie (2015); Le & Nguyen (2018)) or the effects of policy shifts, such as reforms or new regulations, on the health of children (see, e.g., Altindag et al. (2021); Erten & Keskin (2020); Kong et al. (2019); Thompson (2017)). As a result, the poor health outcome of the third generation can possibly reduce the human capital of individuals who carry the burden of their dynasty.

In this thesis, I investigate the multigenerational factors that affect longevity in Paper I and then focus on adult smoking behavior in Paper II and adulthood mental health issues in Paper III. Furthermore, I divide the birth cohorts of the grandparents' generation into the 18th, 19th, and 20th centuries, respectively, in Paper I, Paper II, and Paper III. I present evidence for the multigenerational effects of health-related shocks, risky health behaviors,

and child neglect caused by environmental factors, such as cultures that cover beliefs and attitudes.

2.3 Health inequality in Norway

The health of the Norwegian population is generally considered to be good (HOD, 2007). Despite its reputation as a "welfare state," Norway is a developed nation that still demonstrates considerable health inequalities in the 21st century (Mackenbach, 2017).

Based on the annual report from the United Nations Development Programme (UNDP, 2018), Norway is among the countries with a good population health status in terms of infant and adult mortality rates, child malnutrition, and life expectancy index; Norway is also well known for its egalitarianism. What is the significance of minor health inequalities in Norway and other highly developed countries? Although the Norwegian Ministry of Health and Care Services (2012) has prioritized health inequality reduction, significant health inequalities persist (Mackenbach, 2012, 2019; Strand & Madsen, 2016). Norwegian policies on social inequalities in health are known to contribute substantially to reducing the gaps in mortality and life expectancy across counties (Skaftun et al., 2018). In the regional context, Helgesen et al. (2017) and Fosse (2022) both note that the ability to eliminate health inequities differs among municipalities, and Fosse (2022) emphasizes that municipalities are primarily accountable for service provision in preventative measures. Furthermore, Tiwari et al. (2022) found that cardiovascular disease risk factors, including smoking, drinking excessively, and lack of exercise, are more prevalent in low-income neighborhoods in the Tromsø municipality. Their results are consistent with Sari et al. (2021a)'s study showing that the body mass index, a proxy for an individual's overall health, differs significantly across different neighborhoods in Tromsø.

Overall, most studies on Norway focus on the life course of the current generation and their health outcomes. Limited research on health in Norway considers intergenerational perspectives, with the majority focusing on correlations. One of the important studies is Naess et al. (2013), which shows evidence of correlations between grandparents' cardiovascular mortality and grandchildren's birth weight. In addition, Naess et al. indicate that genetic and environmental factors may play a role, and parental smoking during pregnancy appears to be a significant variable. In another study, Grytten et al. (2014) revealed that after Norway increased the number of years of compulsory education from seven to nine, the likelihood of newborns being born with a low birth weight decreased as mothers' education level increased. Vik et al. (2013) investigated the intergenerational correlation between physical activity and anthropometric measures, such as blood lipid levels and blood pressure, between parents and

adult offspring by using the Nord-Trøndelag Health Study. Recently, Gjerde et al. (2021) and Hannigan et al. (2018) conducted analyses as part of the Norwegian Mother and Child Birth Cohort Study and found a link between maternal depressive symptoms and an increased risk for early life psychopathology in offspring.

Finally, to the best of my knowledge, only a handful of studies in health focus on causal inferences for intergenerational effects using evidence from Norway. For instance, Black et al. (2016) analyzed the impact of maternal stress due to the loss of a parent on the immediate and long-term health of their offspring using Norwegian birth register data spanning 1967-2009. Their results show a small birth effect but no evidence of any long-term adverse impact on children's labor market and education outcomes. Black et al. (2019) investigated the long-term consequences of Norwegian infant exposure to low radiation levels *in utero* due to nuclear bomb testing in the 1950s and early 1960s. They found that low doses of radiation *in utero* result in poorer IQ scores in males and lower education levels and incomes in both men and women. Finally, Aizer et al. (2022) investigated the causal effect of teenage childbearing using Norwegian register data. Their evidence suggests that being the child of a teenage mother has long-term adverse effects on height, cognitive test scores, schooling, teenage childbearing, offspring earnings at age 30, and welfare use. In summary, evidence suggests that early life experiences have a long-term influence on health in Norway. Consequently, even a wealthy welfare state can be limited in providing equal opportunities; see, for instance, Berthung et al. (2022).

3 Overview of the Thesis

3.1 Aims

The primary aim of this thesis is to contribute to our understanding of the mechanisms that underline health inequalities, with a specific emphasis on the multigenerational transmission of health and health behaviors. For this thesis to be successful in achieving its primary aim, there are three supplementary objectives:

1- *Paper I*: To investigate whether economic hardships during a grandmother's pregnancy affect their grandchildren's overall health and whether these effects differ between high and low social classes.

2- *Paper II*: To ascertain whether tobacco smoking is causally correlated with earlier generations' smoking behavior and, if it is, whether maternal versus paternal grandparents affect grandchildren differently.

3- *Paper III*: To examine the relationship between grandparental child neglect and the occurrence of similar adverse behaviors in the next generation, as well as its relation to mental health problems in adult grandchildren.

The first objective relates to whether the health of the grandchildren whose grandmothers experienced economic hardship during their pregnancy was better or worse than that of the grandchildren whose grandmothers became pregnant in relatively good financial statuses. The adverse health effect of economic hardship can be passed down via epigenetic inheritance through generations through fetal vulnerabilities caused by shocks (van den Berg & Pinger, 2016). According to the fetal origins hypothesis, also known as the Barker hypothesis and the scarring effect, exposure to stress during pregnancy increases the likelihood of the offspring having health problems later in life (Almond & Currie, 2011b; Barker, 1990). Unfavorable environmental shocks endured during pregnancy may not only leave a scar (Almond & Currie, 2011a), such as a shortened life span, but also contribute to selective fetal mortality

by increasing the risk of fetal death or death in the early stages of life, referred to as a culling effect (Fletcher, 2018). Since there is evidence that long-term effects may differ depending on grandparents' social classes (Almond et al., 2018; Barone & Mocetti, 2021; Lindeboom et al., 2010), it is essential to test the hypothesis that the negative consequences of economic hardship can vary depending on the social class of the grandmother. Consequently, Paper I anticipates that economic hardship has a more dominant scarring impact on the higher social class. In contrast, the culling effect is potentially more prevalent in the lower social class.

The second objective is to determine the causal relationship in smoking behavior among generations and to determine whether this relationship differs by dynastic lineage. Grandparents invest in their grandchildren's human capital by providing care, time, emotional and financial support (Sadruddin et al., 2019). As stated in Chapter 2.2., Solon (2018) underlines that the human capital production function includes the human capital endowment the child receives regardless of the family's conscious investment choices in the function's error term. Many studies have demonstrated a matrilateral bias in grandmaternal investment, with maternal grandmothers providing more resources than paternal grandmothers (Coall & Hertwig, 2011; Daly & Perry, 2017; Sadruddin et al., 2019). As kin/dynasty members, grandparents' investments can influence their grandchildren directly or indirectly through the child's parents. This transmission can be inherited through genetic and cultural factors (Heckman, 2006), such as parental and grandparental role modeling. In this regard, the focus is on identifying the causal relationships that drive the intergenerational transmission of smoking behavior, emphasizing the distinction between maternal and paternal lineages. To effectively examine these relationships, theoretical concepts such as social learning theory (Bandura, 1971) and the health belief model (Rosenstock et al., 1988) are employed, both of which emphasize the influence of observational learning and personal beliefs on behavior acquisition and continuation, respectively. The transmission of health behaviors, such as smoking, is understood to be deeply embedded in these social and psychological constructs. Thus, these theoretical models provide a strong foundation for the study.

The third objective is to examine the intergenerational transmission of child neglect and its correlated risk for depression, a pervasive mental health problem. Long-term consequences of parental child neglect can manifest as adult mental health problems in offspring, including depression and anxiety, with far-reaching personal and societal consequences (Norman et al., 2012). Given their broad impact on individuals, depression and anxiety disorders impose a significant economic burden on healthcare systems and society (Morrissey & Kinderman, 2020). Therefore, it is important to elucidate the underlying mechanisms of these disorders in order to develop effective preventive approaches and economically efficient policy designs (Persson & Rossin-Slater, 2018). In addition to its intergenerational and multigenerational

effects, child neglect can have profound and lasting effects on an individual's mental health, which ultimately affects their economic prospects (Currie et al., 2010). This makes it an important area of research for economists seeking to address issues related to health inequality and social mobility. This study focuses primarily on the relationship between grandparental child neglect and the mental health of their adult grandchildren (Hayslip et al., 2019). Despite being the most common form of child maltreatment, the basic mechanisms of neglect remain poorly understood (Laajasalo et al., 2023). Paper III explores grandparents' child neglect and the potential development of mental health problems in their grandchildren. The study identifies the roles of maternal and paternal grandparents in relation to the mental health outcomes of their adult grandchildren. This research takes a holistic approach by examining the cumulative risk that underlies the persistence of child neglect across generations. In contrast to previous studies that focused primarily on the relationship between parents' adverse childhood experiences and their children's mental health, the current study shifts the focus to the lasting effects of child neglect by examining whether these effects differ between neglectful maternal and paternal grandparents.

3.2 Conceptual framework

Human life expectancy has risen considerably in all countries worldwide since the late 19th and early 20th centuries. Throughout this process, humans have become healthier and taller than preceding generations. Additionally, noncommunicable diseases, as opposed to infectious diseases, are currently the leading cause of death worldwide, especially in developed countries, with the exception of the recent COVID-19 pandemic. Demographic and social shifts have occurred during this period.

As Goldin (2016) stated, increases in available resources are not the cause of the improvements in health that have been seen throughout much of history; rather, these improvements are the outcome. People become able to spend more on their own health and human capital as a result of greater resources. In other words, people can consume more calories, protein and nutritious meals when they have access to more resources. Additionally, a higher standard of living among the populace increases productivity and enables adults to work longer hours and more intensively throughout their lifetimes. The improvement of people's health is one way that investments in better nutrition may increase human capital. However, individual social inequalities have increased substantially.

Overall, time is a critical factor in studies of multigenerational transmission of health and health behaviors. Therefore, I linked the studies compiled for this thesis in chronological order based on the last generations' birth years to present findings from different time frames

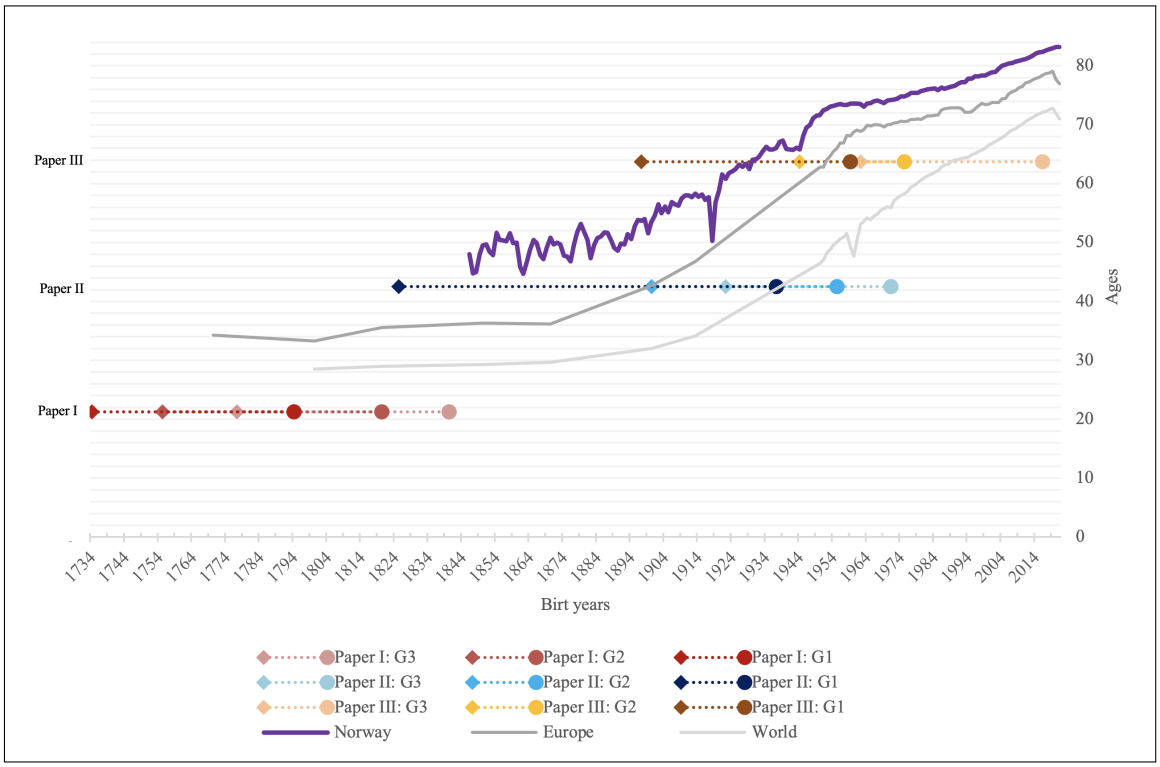

Figure 3.1 *The life expectancy at birth in Norway and the birth years covered in the Papers.*
Notes: The y-axis on the left represents the order number of the papers. The diamond at the beginning of the line denotes the generation's first birth year, while the oval at the end denotes the generation's final birth year referred to in the study. We have no information regarding the grandchildren's (G3) birth year for Paper III. Therefore, the birth years are illustrated based on the same assumption described in Paper III, footnote 16. The life expectancy at birth is plotted along the y-axis on the right. The x-axis displays the birth years covered by this thesis. Data source: UNDP (2018).

(see, Figure 3.1). These studies provide theoretical and methodological insights by deepening our knowledge of the impact of dynasty on an individual's health outcomes over time.

3.2.1 Health transmission across generations in the preindustrial era

There is no doubt that the industrial revolution had a profound impact on society and the economy. As a result of this revolution, even lower-income groups in today's industrialized countries have better access to the vast majority of material goods and enjoy longer lifespans than the higher social class did in the era before the industrial revolution. Although this improvement is a major step forward for humanity, it necessitates the consideration of numerous variables by researchers, complicating research on the role of environmental factors in intergenerational health transmission. Some characteristics shared by preindustrial cultures were low production rates, an economy based primarily on agriculture, a relatively uniform division of work, and limited social stratification (Bull, 2005). Therefore, unlike today, preindustrial revolution period data allow us to conduct research with a particular framework.

Many studies have revealed that longevity is one of the most reliable indicators of overall health status (Björkegren et al., 2022). Considering the population dynamics of the region as a whole allows speculation about the conditions in that region and provides an opportunity to draw conclusions about those dynamics. While making specific conclusions is difficult when using age at death as an indicator of health status, these conclusions are necessary to better understand the ancestor effect on an individual's overall health status. From this perspective, focusing on longevity within the 18th and 19th centuries provides an important standpoint for the subsequent detailed investigation of more specific health outcomes.

As explained in Paper I, the lack of medicines, the study region's relatively isolated geography, and the lack of modern infrastructures that improve well-being and health conditions naturally reduce some of the environmental effects. Overall, the covered period occurred before the establishment of the welfare state and important milestones, such as the Health Act (Sunnhetsloven) in 1860, in public health strategies in Norway (Nordhagen et al., 2014). Therefore, aside from being rare in the literature due to the limited number of available data sets, health research using preindustrial era data allows a clearer identification of the influence of grandmothers on the health status of their grandchildren.

3.2.2 Proactive public health initiatives in Norway and tobacco smoking

Proactive public health initiatives were initiated and promoted in Norway during the late 19th century. In 1886, the Norwegian Physicians' Association was founded to elevate medical professionals' status and provide advice and oversight to local health boards and district medical officers, focusing on public and personal sanitation. In the early 20th century, tuberculosis was Norway's leading killer, especially among young adults. The official public health institutions implemented the law and ran an extensive propaganda campaign to educate the public about infectious illnesses and correct sanitary conduct to prevent infection (Hubbard, 2006). The Norwegian Institute of Public Health was established in 1929 to foster more systematic approaches to public health policymaking and raise public knowledge of proven preventative methods (Nordhagen et al., 2014). The Norwegian welfare state after 1945 reoriented public health policy. Public health was part of this welfare state idea and was intended to be universal and equitable, ensuring that all Norwegians, regardless of their socioeconomic status or where they live, would have access to effective disease prevention measures and state-funded, high-quality medical care in the event of illness or injury (Jordåen, 2006).

Risky health behaviors are universally acknowledged to be one of the greatest threats to longevity. Tobacco smoking, unhealthy diet, alcohol use, and lack of physical activity are lifestyle risk factors that contribute to the development of cancer and cardiovascular

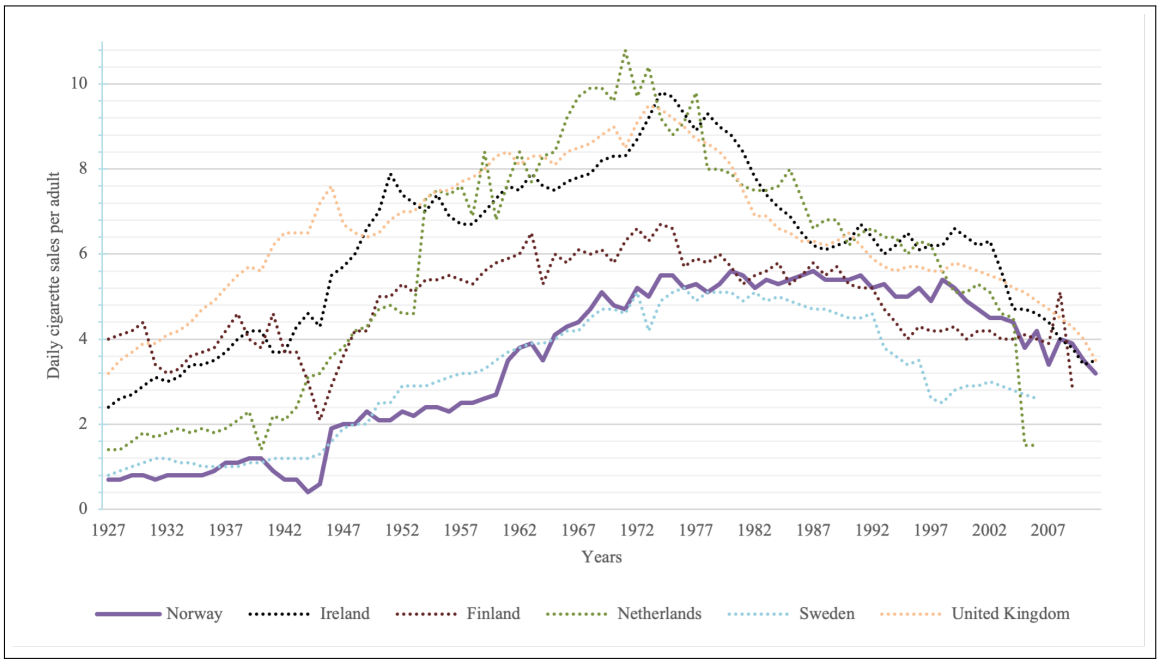

Figure 3.2 *Daily cigarette sales per adult in Norway between 1927 and 2011.*
Notes: The yellow line depicts daily cigarette sales per adult in Norway, and the dashed lines represent the five highest-ranking countries in the European and Central Asian regions in the Human Capital 2020 Index (Finland, Sweden, Ireland, the Netherlands, and the United Kingdom). Data source: Forey et al. (2016).

disease, which are responsible for two out of every three years of life lost in Norway (IHME, 2013; Nordhagen et al., 2014). Considering the reasoning that underpins the welfare state in Norway and the knowledge gained through the country's history of enacting proactive public health policies, Norway initiated a decisive and extensive public health campaign against tobacco smoking. In 1969, the government introduced a white paper to the Parliament entitled "Influencing Smoking Behavior." In the report, one of the many recommendations was a prohibition on advertising tobacco products and the requirement that all tobacco products carry explicit health warnings (Helsedirektoratet, 2021). Since 1988, smoking has been prohibited in all workplaces, and since 2004, smoking has been completely outlawed in all public places, including bars and restaurants. Norway's laws regarding the use of tobacco are still considered to be among the world's strictest, and the country has seen the positive effect of these public health measures, which it has implemented over time and in many different ways. Figure 3.2 shows that Norway has the lowest rate of cigarette sales per adult per day out of the five countries ranked highest in Europe and the Central Asia region in the Human Capital 2020 Index (World Bank, 2021), excluding Sweden. As shown, this rate has been decreasing since the 1990s.

Within the context of risky health behaviors, Paper II presents a study covering most public health interventions regarding smoking. Unlike the previous studies in the literature,

this study centers on the grandparents who smoked while they were bringing up their children and the effect that this has on their grandchildren's smoking behavior rather than directly examining the effects of the implemented policies. In summary, the period when grandparents smoked in the presence of their children corresponds to a time before Norway implemented large-scale anti-smoking policies. In addition, the period in which parents smoked while raising their children roughly covers the periods when smoking bans were widely applied. Subsequently, the smoking of grandchildren in adulthood encompasses a period in which great efforts were made in anti-smoking campaigns, which were mainly received positively in society. Therefore, these efforts make Norway a distinct region and confer the potential for the country to be used for meaningful comparisons and as a source for tobacco smoking research that can be conducted in other countries.

3.2.3 New millennium: Mental health problems in the chronic disease era

Noncommunicable diseases dominated the disease landscape at the beginning of the 21st century, and since the 1970s, technological advancements and improvements in medical treatment have impacted patient survival. Now that we are entering the third decade of the 21st century, the accomplishments made by scientists have been remarkable, especially in recent years. As a result, substantial advances have been made in the mental health field.

Emotional, psychological, and social well-being are all components of mental health (WHO, 2022), and physical and mental health are essential components of overall health. For example, some of the numerous physical health issues for which depression is a risk factor include diabetes, heart disease, and stroke (NIMH, 2021). Similar to how chronic illnesses can raise the risk for mental illness, mental illness can also increase the risk for chronic conditions. That is, a person's mental and physical health is inextricably linked to their overall health and, thus, to the likelihood of a long lifespan. It is essential to remember that a person's mental health, like their physical health, might shift throughout their lifetime depending on a multitude of factors.

The most important thing to consider in this context is the cause of mental health problems. However, one or more factors can cause various mental health problems, such as anxiety, depression, post-traumatic stress disorder, and schizophrenia. Several factors put someone at risk of developing a mental illness. A person's mental health can be affected not only by the presence of other chronic medical diseases, such as cancer or diabetes, but also by biological causes or chemical imbalances in the brain. Additionally, adverse early life experiences, such as a traumatic event, a history of abuse, child abuse and neglect, sexual assault, and

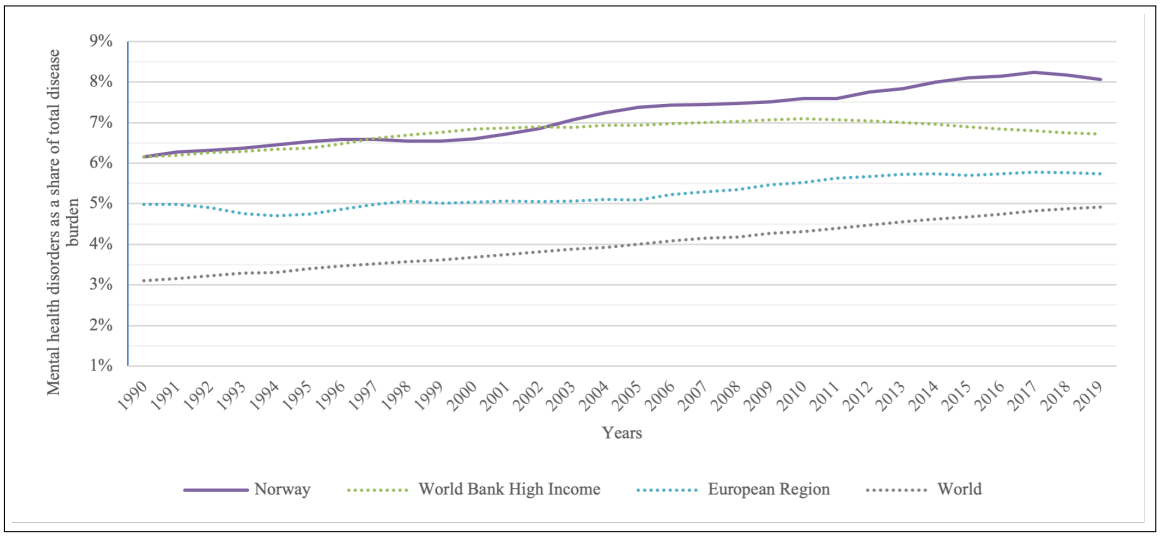

Figure 3.3 *The share of Norway's total disease burden to mental health disorders between 1990 and 2019.*

Notes: The total disease burden share attributes of mental health disorders in Norway is represented by the purple line. The average for high-income countries, according to the World Bank, is represented by the green dashed line. The light blue dashed line represents the average for countries in the European Region, while the gray dashed line represents the global average. Data source: IHME (2021).

witnessing violence, are risk factors for mental illness. Many studies have shown that the first signs of mental illness typically appear in the early stages of a person's life. It is also well known that mental health issues can have a lasting impact on one's human capital due to their inherently chronic characteristics. Even after accounting for the individual's future health and their health at the time of birth, Currie et al. (2010) demonstrated that early mental health problems have a significant potential to predict an individual's health in adulthood. In continuation, Johnston et al. (2013) went even further and presented explanatory evidence of intergenerational mental health permanence.

While the global and European average of mental health disorders as a share of the total disease burden increased between 2000 and 2010 (see, Figure 3.3), this share remained nearly stable in high-income countries between 2000 and 2010 and began to decline after 2010. Norway's trend line largely mirrors that of the European region with a higher share, diverging from the other high-income countries and continuing to rise. It should be noted that the shares of the total disease burden for mental health disorders represent the period before the COVID-19 pandemic. Therefore, it is evident that mental health disorder rates are increasing globally, and their prevalence among other diseases is also rising in Norway. In conclusion, Paper III takes a deeper look into health in general by addressing the transmission of childhood neglect across generations, mental health problems, and, thus, chronic illness.

3.3 Methodological approaches for multigenerational effect studies

The extent to which children mirror their parents due to nurture, nature, or some mix of the two remains unknown. Therefore, studying the causal relationship between parents and children presents challenges, and focusing on more than two generations further complicates the study of intergenerational transmission mechanisms.

Intergenerational mobility studies in economics mostly start with Becker and Tomes (1986) and further develop this model (Solon, 2014, 2018). The research agenda for multigenerational studies on social mobility, according to Güell et al. (2018), requires the development of empirical approaches that can be utilized to identify more exact metrics for assessing the substantive value of estimated impacts from grandparents or distant ancestors. In multigenerational health studies, adoption and twin designs are the most commonly used approaches in studies that examine health transmission across three or more generations (see, e.g., Andersen (2021); Björkegren et al. (2022); Lundborg et al. (2018)). A rising number of studies in social sciences are examining whether grandparents' health or socioeconomic position affects grandchildren's health outcomes, with or without adjusting for parental variables. However, as we go one generation deeper, the methodological needs increase and require specific solutions.

Understanding the factors associated with intergenerational transmission is vital for forming suitable public policy. Economists have moved beyond the nature versus nurture debate and are now trying to ascertain the impact of certain parental attributes on children's outcomes (Black & Devereux, 2011). Nevertheless, it is difficult to comprehend how to encourage change without knowing the underlying mechanisms. The challenge is that any specific parenting quality is frequently connected with a range of other parental and grandparental attributes, most of which cannot be detected in the data. Researchers have employed various methodological tools to better understand the underlying causes of the observed grandparent–parent-child transmissions. However, several methodological concerns have made it difficult for researchers to draw firm conclusions about whether grandparents directly influence their grandchildren (Breen, 2018).

As a result, Kawachi et al. (2010) determined that two potential issues must be addressed while building study models to obtain causal inferences from observational studies. The first is the possibility of reverse causality. Typically, researchers mostly consider transmission from parents to offspring when studying health across generations. However, we must be aware that the causality effect is not always linear and can also work in the inverse direction (see, e.g., Lundborg et al. (2018)). It is essential to identify variables and the period they span

and to ensure that their timings do not overlap to avoid this simultaneity. Selected variables for all generations in Paper II and Paper III provide clear examples of how to overcome this overlap possibility. For instance, in Paper II, we analyze whether grandparents' tobacco smoking while raising their children influences their grandchildren's smoking behavior. Grandchildren's tobacco smoking behavior cannot influence whether grandparents smoked while raising their children.

The second is omitted variable bias. Some omitted variables that impact both parents and their children, such as genetic traits, cultural environment, ethnicity, and community (Breen, 2018), make it difficult to determine the overall bias level in estimates of the direct effect of grandparents on grandchildren. Nonetheless, Breen (2018) states that even though certain countermeasures have been proposed to cope with these challenges, it is essential to seriously consider the possibility of biases and base one's interpretations on such biases.

The omitted variable bias is critical in assessing direct effects from grandparents to grandchildren and must be overcome for causal claims. Assumptions allow for some commentary on biases; however, these assumptions frequently involve relations that have not been observed and, therefore, cannot be tested. Breen (2018) highlights two main approaches to this matter. The first is a test developed by Tingley et al. (2014) to determine how sensitive estimates of a direct effect are. In Paper I, I followed Thompson et al. (2019) and used structural equation modeling with the sequential ignorability assumption, which states that residuals are uncorrelated and independent. Gunzler et al. (2013) note that structural equation modeling captures complex and dynamic connections among observable and unobserved factors. However, one of the most significant drawbacks of the methodology is the inclusion of an unmeasured confounder, which can confound the relationship between the outcomes of the parents and the grandchildren (Imai et al., 2010b). As a result, a sensitivity analysis is required to determine whether the findings are resilient if the sequential ignorability assumption is violated (Imai et al., 2010a,b). The sensitivity analysis for this technique depends on the correlation between the residuals derived from the models for the outcomes of the grandparents and the grandchildren. Such studies are especially helpful for identifying whether unmeasured confusion might significantly influence the results of both the average direct and indirect effects (Vanderweele, 2016).

The second is an instrumental variable approach. In addition to the twin and adoptee approaches, the predominantly instrumental variable estimate method has been used extensively to investigate the causal link between a parent and their children's outcomes (Black & Devereux, 2011; Lundborg et al., 2018). This approach addresses any potential endogeneity concerns, biases, and measurement errors (Currie & Stabile, 2006).

Currie & Moretti (2003) conducted the first study that used instrumental variable estimation on the subject. They measured mothers' education by the rise in the number of colleges in each state in the United States and found that college education enhanced maternal and infant health indicators, including birth weight and gestational age, and decreased maternal smoking. The Loureiro et al. (2010) study is another example. Using the instrumental variable method, they studied whether parental smoking influenced their children's smoking behavior. They found evidence that mothers have a more significant influence on their daughters' smoking behavior, while fathers' smoking behaviors are predominantly mimicked by their sons. In their study on the intergenerational transmission of education, Lindahl et al. (2014) linked great-grandparents' data to great-grandchildren's data and used the educational attainment of the great-grandparent generation as an instrumental variable. More recently, Ren et al. (2020) employed an instrumental variable estimation technique to establish the effect of education on the intergenerational persistence of unhealthy consumption. They used two institutional changes as instruments to demonstrate this influence.

The empirical estimation findings suggest that children's risky health behaviors, such as heavy drinking and tobacco smoking, are positively correlated with the unhealthy behaviors of their parents in China. Last, Helle et al. (2022) used instrumental variable regression to demonstrate more causally interpretable conclusions regarding whether grandparental investment might lessen emotional and behavioral issues in children confronting several adverse childhood experiences. When children had been subjected to several adverse childhood experiences, the involvement of maternal grandparents could lessen their emotional and behavioral difficulties. Nevertheless, it was unable to eradicate these challenges completely. According to these results, maternal grandparent involvement can increase children's wellness even in unfavorable environments.

As an alternative, we can use the control function approach or the two-stage residual inclusion (2SRI) method. This method is similar to the instrumental variable method and comparable to the two-stage least squares (2SLS) method when the models used in both the first and second stages are linear (Basu et al., 2018). Similar to 2SLS, this method has two stages, and the instrumental variable must be included in the regression's first stage. Thus, the control function approach, by definition, is an instrumental variable-based method. Then, we obtain the residuals from the first-stage regression. Second-stage regression requires the inclusion of the acquired residual from the first stage as a covariate alongside the initial endogenous variable and any relevant control variables. Wooldridge (2015) comprehensively explains the control function method used in applied econometrics. More specifically, the equation being modeled contains at least one explanatory variable that is either known to be

endogenous or is suspected to be endogenous due to the possibility of its correlation with unobservable.

Terza et al. (2008) suggested that nonlinear 2SRI is preferable to linear 2SLS for binary dependent variables because it provides more reliable estimates of the overall treatment impact. In another study, Basu et al. (2018) investigated a case with a binary result, a binary treatment, and a binary instrument. They concluded that the 2SLS approach combined with a binary instrumental variable produced the most reliable estimations in their scenario. Basu et al. also highlight that for very rare outcomes (< 5%), the least-biased estimate of the average treatment effect can be generated using 2SRI. Paper II employs a three-stage regression model within a structural equation framework that includes instrumental variables to mitigate endogeneity concerns. The method also uses a control function to deal with selection bias, bootstrapping to address potential issues related to standard errors, and various tests to ensure the validity and relevance of the chosen instrumental variables. Paper III uses ordinary least squares regression to examine the relationship between neglect in the parental and grandparental generations and mental health, specifically depression, in the adult grandchild generation. The model captures the combined effect of grandparental and parental child neglect on grandchild mental health, tests the additive risk hypothesis.

3.3.1 Data for multigenerational studies used in this thesis

Data availability is the most important and basic issue for studying multigenerational transmission (Güell et al., 2018). The availability of comprehensive and high-quality data varies across countries. In this context, Nordic countries are well known for high-quality data based on both historical records and contemporary studies. Norway is one of the Nordic countries and has available data sources. Nevertheless, studies on the multigenerational effects on health are very rare. Under the scope of this thesis, I used both historical and contemporary data sets to study the multigenerational transmission of health.

Rendalen database

From a historical perspective, the municipality of Rendalen[1] is one of the most researched places in Norway and has a vast historical archive to help researchers (Skotte & Hagen, 2018). Thanks to Sølvi Sogner's extensive work and personal efforts, the Norwegian Historical Data Centre made the data available for researchers to study the 18th and 19th centuries (Sommerseth, 2019). The primary data sources for this data set are church records of

[1]Extensive details about Rendalen are provided in the Rendalen section of the first study. For more practical details, please visit https://rhd.uit.no/.

baptisms, marriages, confirmations, and burials (Bull, 2006). The data span the years 1733 to 1925, covering the lives of individuals and their descendants. The data set was formed from data sets developed by linking the censuses (1801, 1865, 1875, 1900, and 1910), parish registers, baptism, and cadastral records that cover the period between 1733 and 1925 in Rendalen.

The significance of the Rendalen data lies in the length of time it covers. This data set is one of the oldest that can be utilized for studying health inequalities that span multiple generations. A further consideration is that the observed period occurred prior to the development of advanced medical infrastructure in Norway (Saunes et al., 2020). The fact that it occurred before the introduction of antibiotics is of substantial importance (Zaffiri et al., 2012). Meanwhile, there are a few obstacles to overcome when conducting research on the multigenerational transmission of health outcomes. The first obstacle that needs to be overcome is reorganizing the Rendalen data set to create links between different generations. To overcome this challenge, I merged the three successive generations based on their given ID numbers in the maternal line. This allowed me to maintain a complete data set while minimizing the risk of losing significant information. As a result, the merged data consist of three-generation chains: grandmothers who had given birth to at least one mother, mothers who had had at least one child, and children survived for at least twelve months after birth. Paper I provides more detailed explanations.

The Tromsø Study

Tromsø is the largest municipality in northern Norway (2022: 77,700 residents),[2] with a mix of urban and rural regions (90%). The majority of the population is of Norwegian origin, and most have jobs in the service industry or the medical and social care sectors. The Tromsø Study is an important multipurpose population-based cohort study[3] in Norway. This study has been used to answer many research questions in several disciplines, such as medicine, healthcare, and social sciences. The Tromsø Study is a comprehensive health investigation focusing on chronic and communicable illnesses and the associated risk factors. The study seeks to enhance the long-standing monitoring of illness and risk factors in the community (Hopstock et al., 2022).

The user engagement process of the Tromsø Study, especially in Tromsø7, is quite inclusive and is the product of the collective mind of professionals in many health-related fields. In addition to researchers, users of the Tromsø Study include the municipality,

[2]The statistics were obtained from https://www.ssb.no/kommunefakta/tromso/.

[3]Epidemiological studies in the "population-based cohort" category are distinguished from other types of studies because they include the continuous monitoring of a predetermined population to determine the relationships between an exposure and an outcome (Sorlie & Wei, 2011).

county, health authorities, healthcare professionals, participants, and the general public. The Norwegian Institute of Public Health, the Northern Norway Regional Health Authority, Troms County, the Tromsø municipality, the University Hospital of North Norway, and the Norwegian Health Association in Tromsø7 are among these users (Hopstock et al., 2022). In addition, the Tromsø Study has allowed the development of a wide variety of epidemiological and clinical research designs, one of which is the linking of registries (Eggen et al., 2013); thus, this study has attracted the attention of social scientists.[4]

Even though it was theoretically possible to construct the family and generational linkage within the Tromsø Studies, it was initially impossible to do so in practice. Therefore, this linkage had not previously been constructed in the Tromsø Studies. Torbjørn Wisløff, leader of the interdisciplinary strategic project High North Population Studies at UiT the Arctic University of Norway, requested the Tromsø Study participants' ID numbers from the Norwegian Tax Administration for this doctoral project under the Social Inequality in Health research group. After receiving the requested information, it became possible to identify family relationships for the Tromsø Study. Afterward, I constructed the family and two generational linkages for the first time to utilize them in Paper II and Paper III. At the end of the linkage, the final data set covered mothers, fathers, and their offspring, including siblings for both generations. Here, it is worth noting that the definition of generations differs between Paper II and Paper III. In Paper II, I used parents (Generation 2) and their offspring (Generation 3) from the Tromsø Study participants; I included grandparents' (Generation 1) tobacco smoking while raising their children from the reports of Generation 2 on their parents' smoking. I could then compile the results from three successive generations. For Paper III, neglect experienced by Generation 3 (adult offspring) during their childhood was measured from the Tromsø Studies, with neglect being defined as the failure of their parents (Generation 2) to provide them with adequate care. Similarly, data on neglect experienced by Generation 2 during their childhood was obtained from the Tromsø Studies and attributed to the failure of their parents (Generation 1 or grandparents) to provide adequate care. The mental health status of Generation 3 was assessed on the basis of their self-reported experience of depression. Thus, by integrating parental responses linked within the Tromsø Study, I was able to collect and analyze data from four generations within the same study.

[4]For more details about the Tromsø Study's cohort profile, the data collection process, how to address ethics and privacy issues, and communication strategies for the recruitment process are comprehensively addressed in Eggen et al. (2013), Hopstock et al. (2022), and Jacobsen et al. (2012). For more practical details, please visit https://uit.no/research/tromsostudy.

3.4 Synthesis of studies

3.4.1 Paper I: Transgenerational health effects of *in utero* exposure to economic hardship: *Evidence from preindustrial Southern Norway*

In Paper I, we studied whether a grandmother's *in utero* exposure to economic hardship during her pregnancy has a transgenerational influence on the overall health status of her grandchildren. We utilized one of the earliest data sets available in transgenerational research within the social sciences, which included rich data on the individual level from the 18th and 19th centuries from the Norwegian municipality of Rendalen. Individual-level data spanning three generations also allowed us to determine the socioeconomic status of households. Only a handful of studies have examined how exposure to an external shock *in utero* might influence more than two generations, and the findings have been contradictory. Therefore, many unsolved problems merit investigation. To the best of our knowledge, no research on the transgenerational effects of *in utero* shocks has examined the significance of the relative intensity of the culling effect as selective fetal mortality (Fletcher, 2018) and the scarring effect in conjunction with the fetal origins hypothesis (Almond et al., 2018).

In this study, we anticipated two primary mechanisms through which economic hardship during the grandmother's pregnancy with her daughter is connected with a grandchild's life span: a positive culling impact and a negative scarring effect through the mother's health state. We utilized mediation analysis with the structural equation model approach (Thompson et al., 2019). One of the most notable findings from our study was that there was a positive and substantial association between economic hardship during the grandmother's pregnancy and the grandchild's life duration among grandchildren born to lower social class families. This finding provides evidence of a positive culling effect in the context of transgenerational transmission. In addition, the data revealed that economic hardship affected those in lower social classes and had a detrimental scarring effect on succeeding generations of those in higher social classes. These findings shed light on the three-generation route that was investigated to see how *in utero* economic adversity affects the health of subsequent generations and social inequalities in health.

In conclusion, the strength of the net relationship between fetal shocks and subsequent generations' health is an empirical question and depends on the balance between culling and scarring. Since survivors are subjected to intense positive selection during periods when culling is the dominant force, we may not see any scarring. When scarring is predominant, a detrimental effect is passed down through the mother, shortening the third generation's life span.

3.4.2 Paper II: Role of grandparents in risky health behavior transmission: *A Study on smoking behavior in Norway*

Paper II investigates the intergenerational transmission of risky health behaviors, with an emphasis on tobacco smoking. In this study, we examined the probability of direct transmission of smoking behavior from grandparents to grandchildren and the probability of a differential impact between maternal and paternal lineage grandparents. We used the individual three-generational Tromsø Study data set from Norway to examine kin selection theory (Coall & Hertwig, 2010) and the transmission of risky health behaviors. We used a two-stage residual inclusion approach to estimate causal relationships (Breen, 2018; Wooldridge, 2015). Few studies have analyzed the intergenerational transmission of smoking behavior, and even fewer have focused on the relationship between grandparents and grandchildren (Sadruddin et al., 2019). This study adds to the literature on the intergenerational transmission of risky health behaviors and provides a deeper understanding of the mechanisms underlying health capital. To the best of our knowledge, this is the first study of its kind conducted in Norway.

We hypothesized that maternal grandparents would have a more significant impact on their grandchildren's decision to forego smoking than paternal grandparents. In addition, we anticipated that grandparents' smoking behavior would influence the grandchild through their influence on the parents; socio-emotional transmission may also indirectly affect the grandchild beyond the direct effects. In contrast to the paternal lineage, our findings demonstrated that only maternal grandparent smoking behavior has a significant direct negative effect on the offspring's smoking probability. The findings of our study foster an interesting discussion between social learning theory and the health belief model. Additionally, our results indicate that grandparents' smoking is transmitted to their grandchildren via the smoking behavior of their parents. This indirect effect positively influences the probability of offspring smoking, thus offsetting the negative effects of grandmothers on grandchild smoking.

In conclusion, families, which are the most immediate social setting, have an undeniable influence on the initiation of tobacco use in their children because of their proximity. How individuals choose to spend their time and take risks can influence the course of their lives. However, positive changes in these preferences over time may influence their grandchildren not to smoke when they become grandparents, unlike their grandparents. Raising awareness of the powerful influence that parents, and to a lesser extent grandparents, have on their children's behavior will, in the end, result in improved health outcomes and increased economic well-being.

3.4.3 Paper III: Long-term effects of grandparental child neglect on grandchildren's mental health: *A Three-generation study*

Paper III focuses on understanding the relationship between grandparental child neglect and the mental health outcomes of their grandchildren. Using data from the Tromsø Study, the researchers employ a linear probability model with ordinary least squares regression to estimate the likelihood that grandparents' child neglect affects the mental health of their adult grandchildren. This approach allows for a comprehensive examination of the intergenerational transmission of child neglect, taking into account grandparents from both sides of the family.

The results of the study support the additive risk hypothesis, which suggests that the combined effects of childhood maltreatment by neglectful maternal grandparents and parents have a more severe influence on the mental health of grandchildren than maltreatment by parents alone. This suggests that the cumulative effect of intergenerational neglect increases the risk of mental health problems in grandchildren. These findings highlight the importance of considering the role of both grandparents and parents in understanding the intergenerational transmission of child neglect. Besides, the study provides insight into the mechanisms underlying the intergenerational transmission of neglectful behavior. It suggests that neglectful grandparents may influence the parenting behaviors of their children, who, in turn, may perpetuate the same parenting style with their children. This intergenerational cycle of neglect is driven by social learning processes in which children learn by observing the behavior of their parents and grandparents. The study highlights the potential role of attachment theory, suggesting that neglect may disrupt the emotional bonds between parents and children, leading to adverse outcomes in the mental health of grandchildren. The study also acknowledges the potential differences between the influence of maternal and paternal grandparents on their grandchildren. Maternal grandparents, who traditionally play a more involved caregiving role, may have a greater impact due to their closer relationship and investment in the lives of their grandchildren. This distinction is consistent with evolutionary theories that account for the certainty of biological ties and the differential investment of grandparents based on their genetic relatedness (Coall & Hertwig, 2010, 2011).

In conclusion, this study contributes to our understanding of the intergenerational transmission of child neglect and its influence on grandchildren's mental health. By considering both grandparents and parents, the research provides a comprehensive view of the cumulative effect of neglect across generations. The findings highlight the importance of interventions aimed at breaking the cycle of neglect and promoting the mental well-being of future generations.

Discussion

There are four broad inferences to be made from this thesis. First, despite the country's relatively high equality and well-established welfare-state structure, Norway still has health inequalities (Mackenbach, 2017, 2019), calling for a closer examination of the factors that led to these inequalities. Studying a multigenerational effect based on environmental causes of adverse health outcomes reveals a more profound entrenched inheritance that goes beyond the interactions between parents and their children through biological persistence (Black et al., 2020). Expanding on previous literature (see, e.g., Black et al. (2019); Erten & Keskin (2020); Halliday et al. (2020); Thompson et al. (2019), I employed causal methods to examine various outcomes using historical and contemporary data, expanding our understanding of the role of nurture on multigenerational effects. Using the Papers included in this thesis, I provide convincing evidence that public health policies focusing on the early years of individuals and addressing parental and grandparental issues are essential. While these findings add to the growing empirical investigations since Ahlburg (1998) and Ashenfelter (1973), they also shed light on the dynastic origins of social inequalities in health, which have received great attention after Townsend et al. (1982).

Health outcomes are influenced by numerous variables, including the societal demographics of the time and place (Mare, 2014; Solon, 2018); thus, it is crucial to remember that as society evolves, these aspects also change. In this context, the second contribution is that the grandparents themselves could be both the root of the problem and the solution to it. Maternal grandparents, paternal grandparents or both can be both the cause and the cure in different instances.

The third contribution relates to the methodology used. As stated in Paper II, I used a control function approach (Breen, 2018; Wooldridge, 2015) to estimate the causal effects of grandparents on their grandchildren's health outcomes. I expand our estimation beyond the conventional two-stage residual inclusion to a three-stage residual inclusion. This was performed to prevent potential reporting and response bias (Kinge et al., 2021; Lindeboom & van Doorslaer, 2004) in addition to the omitted variable bias (Breen, 2018; Solon, 2018). In

other words, I used a novel and practical approach in light of previous research on possible biases associated with multigenerational studies and reported health behavior outcomes.

Finally, to the best of my knowledge, this thesis is one of the most comprehensive studies in economics that focuses on multigenerational effects on health in Norway. Norway is a Nordic welfare nation with a robust public sector, including free higher education and healthcare for all citizens, a rich welfare system, and progressive policies promoting gender equality (Mackenbach, 2017). Consequently, as Aaskoven et al. (2022) stated for Denmark, it can be argued that Norway also constitutes a 'best-case' scenario, providing likely lower bounds for the grandparental effects on grandchildren's health outcomes compared to other developed countries.

Policy implications

Ahlburg (1998) states that social scientists have long been interested in the intergenerational transmission of income and socioeconomic status. This interest has subsequently grown exponentially, and its growth is expected to continue. This line of study is inspired by concerns that allowing one's family background to determine one's economic prospects can run counter to the principle of equal opportunity (Solon, 2018). In addition, along with demographic transitions over time (Song & Mare, 2019), it is necessary to understand the multigenerational causes of adverse health outcomes affecting human capital accumulation and to find more effective public policies as a current necessity of this transition. This perspective covers our family roots, not just biologically but also via culture and behavior. Therefore, there is a need to examine cultural inheritance from parents and grandparents more closely. I argue that for the healthy development of future generations, policymakers need to re-evaluate and expand current health policies by including family members, including grandparents.

Limitations and suggestions for further studies

Mare (2014) highlights the importance of time and timing as a limitation of intergenerational studies on social mobility. This limitation also applies to multigenerational studies of health and health behaviors. According to Mare, the investigation of impacts that span several generations makes it possible to identify complex and nuanced relationships between the passage of time and health outcomes. Even in models of health transmission that only include two generations, the ages of the parents and the children still provide significant interpretational challenges. Because of this, the "generation gap" between parents and their children may vary in size depending on the parents' birth years and the ages of their

children. The age of the parents may affect the accomplishments of their children. For instance, Aizer et al. (2022), using Norwegian register data, investigated a causal effect of teenage childbearing. Their evidence suggests that being the child of a teenage mother has long-term adverse effects on height, cognitive test scores, schooling, teen childbearing, offspring earnings at age 30, and welfare use. In addition, older parents may earn more money, potentially making them more aware of the significance and consequences of their health-related behaviors (Case & Paxson, 2002), or vice versa. The same thing can be said about the grandparents' generation; depending on the ages at which grandparents and parents birth their children, the age gap between a grandparent and grandchild may be as short as 50 years. If everything else remains the same, young grandparents have many more potential years ahead of them in which they may appreciate their grandchildren, and their grandchildren also have much more time ahead of them in which they can get acquainted with their grandparents. Fomby et al. (2014) investigated the potential impacts of the ages of grandparents when grandchildren were born on the cognitive results of those grandchildren. In summary, this kind of inquiry is a promising beginning to a more extensive collection of timing studies, which may be intriguing in their own right but are crucial for accurately comprehending the processes that take place throughout several generations.

Furthermore, the Tromsø Study is a prospective, ongoing study (Hopstock et al., 2022) with no predetermined conclusion date, which is encouraging in terms of future research opportunities. The upcoming Tromsø8 and the inclusion of collected biological samples will allow researchers to investigate in more depth and cover a more comprehensive range of issues from the point of view of multigenerational transmission.

In addition, we do not know how many children were in the families of those who participated in the Tromsø study or how many siblings each participant had. However, I believe that linking the register data with studies such as The Trøndelag Health Study (HUNT), The Finnmark Study, Fit Futures, and the Tromsø Study will provide excellent opportunities to investigate deeper dynastic ties for the health outcomes of individuals today. In this regard, research on children's education by Black et al. (2005) demonstrates that it is feasible to examine the effects of birth order using Norwegian registration data while also making a trade-off between the quantity and quality of children within a household. Additionally, Cools & Kaldager Hart (2017) investigate the influence of sibling number on adult fertility using Norwegian register data from the 1960s in Norwegian households with at least two children.

Consequently, combining population-based cohort studies from Norway and linking them to nationally registered data is likely to yield broader results; however, this is not something that can be accomplished within the Ph.D. program due to the time and resources needed.

Nevertheless, the findings to date are of sufficient importance and provide directions for future research. Consequently, I intend to continue my research in this area.

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Paper I

Transgenerational health effects of *in utero* exposure to economic hardship: *Evidence from preindustrial Southern Norway*

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Transgenerational health effects of in utero exposure to economic hardship: Evidence from preindustrial Southern Norway

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ABSTRACT

We studied whether *in utero* exposure to economic hardship during a grandmother's pregnancy has a transgenerational effect on her grandchildren's health condition. We used an individual-level three-generation data set covering people born between 1734 and 1840 in the municipality of Rendalen in Norway. We found a culling effect in which grandchildren whose grandmothers gave birth in years of economic hardship lived approximately ten years longer than grandchildren whose mothers were born in years of economic well-being. This impact was only observed among the grandmothers who belong to the lowest social classes. Our results also showed that in higher social classes, economic hardship during a grandmother's pregnancy deteriorated her grandchildren's health by "scarring" the mother's health.

1. Introduction

A large body of economic literature has shown that shocks *in utero* can have a persistent impact on later life outcomes (Almond and Currie, 2011, 2018; Bruckner and Catalano, 2018; Currie, 2020; Lee, 2014; Lindeboom et al., 2010; Menclova and Stillman, 2020; Thompson et al., 2019; Van den Berg et al., 2009a, 2009b). Animal studies suggest that the effect of negative exogenous factors persists across multiple generations (Skinner et al., 2010), but studies spanning more than two generations are still rare in the social sciences (Van Den Berg and Pinger, 2016).

In our paper, we studied whether the grandchildren of women who suffered economic hardship were healthier or less healthy. Fetal vulnerabilities due to shocks are some of the cardinal components of adverse epigenetic[1] inheritance across generations (Franklin and Mansuy, 2010; Skinner et al., 2010). These adverse conditions not only affect prenatal development *in utero* but also remain active in the first year of life through early-life exposures (Barker, 1990). Adverse environmental shocks experienced *in utero* may do more than leave a "scar" (Almond and Currie, 2011). They can also increase fetal and early-life mortality rates. Therefore, those who are exposed to these circumstances *in utero* but survive may also be potentially selected in cases where selection is endogenous to the same adverse event as the scarring effect. As fetal mortality tends to eliminate fetuses that are in poor health, survivors of adverse fetal events are generally positively selected. One of the main issues addressed thus far in this framework is the direction and scale of the transmission of *in utero* exposure to exogenous shocks across generations (Almond and Currie, 2011). According to Almond and Currie (2011), survivors of adverse fetal events are usually positively selected, a process known as culling, because mortality tends to eliminate unhealthy fetuses. For this process to be detected, the positive impact of selection *in utero* among survivors over generations must be strong enough to dominate the negative scarring effects (Almond and Currie, 2011; Blum et al., 2017; Bruckner and Catalano, 2018; Deaton, 2007).

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[1] Skinner et al. (2010) "define epigenetics as 'molecular factors and processes around DNA that are mitotically stable and regulate genome activity independent of DNA sequence".

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Therefore, the net association between shocks *in utero* and the health of children and grandchildren is an empirical issue: it depends on the relative strength of culling and scarring. When the effect of culling dominates, we may observe no scarring because survivors are highly positively selected. When scarring dominates, a negative association between a shock and health is observed.

Only a limited number of studies have addressed how *in utero* exposure to external shock can affect subsequent generations, and their results are mixed. Lee (2014b) demonstrates that women's stress during the Kwangju uprising (1980) in South Korea negatively affected their grandchildren's birth weights. Cook et al. (2019) and Richter and Robling (2013) found that grandmothers' *in utero* exposure to the 1918 influenza pandemic lowered their grandchildren's educational performance. Conversely, Kaati, Bygren and Edvinsson's (2002) study conducted in Överkalix in Sweden found a lower ratio of cardiovascular disease-related deaths in children whose fathers had difficulties obtaining food early in their lives than in children whose fathers did not have such difficulties. Moreover, Van Den Berg and Pinger (2016) show that a grandparent's exposure to famine had a positive effect on their grandchildren's mental health. There are still many unanswered questions that warrant inquiry about whether there are positive or negative influences of *in utero* exposure to external shocks for subsequent generations. To our knowledge, research on the transgenerational effects of *in utero* shocks has not focused on the role of the relative strength of culling and scarring. Our research will help to fill this gap.

Our paper makes several contributions. First, we use rich data from the 18th and 19th centuries to look at the long-term and transgenerational health impacts on individuals. The time period we studied predated the establishment of modern medical infrastructure in Norway (Saunes et al., 2020) as well as the use of the first antibiotics (Zaffiri et al., 2012). Our study uses one of the oldest data sets available for multigenerational studies: the oldest individuals in our data set were born in 1743, and the youngest were born in 1840. The resulting simplicity of the health-related exogenous conditions within this time frame is an advantage of our data.

Second, with our individual-level data, we are able to identify families according to their social class. Exogenous shocks *in utero* can hit vulnerable groups more heavily by way of selection, while in other groups, they may yield scarred cohorts (Bruckner and Catalano, 2018). The scope for selective fetal mortality (i.e., culling) is generally found to be more prominent in situations where baseline health is poor (Almond and Currie, 2011). Therefore, we focus on the landless as the lowest social class who lived closest to the margin and therefore were not only most vulnerable to economic hardship but also had the lowest possibility of counteracting its negative effects. We thus would expect that short-term economic hardship led to higher selective mortality among this social class.

We also analyzed landowner families, who had relatively more opportunities to counter the negative effects of economic hardship. Children born to this group might suffer from the negative effects of economic hardship for life. The fetal origins hypothesis and selection *in utero* coincide with the scarring effect and similarly assume that *in utero* exposure to maternal stress increases the likelihood of having adverse health conditions later in life (Bruckner and Catalano, 2018). We anticipate that short-term economic hardship resulted in a greater scarring effect among the higher social class.

Instead of examining the direct association of exogenous shocks, health indicators, and other outcomes considered in previous literature, we use more interpretable disaggregated measures. Using mediation analyses, we explore whether the effects of economic challenges faced during pregnancy persist across generations by following the maternal line. We use the health conditions of the mother as a mediator for both high and low social classes. We show that *in utero* exposure to economic hardship plays an important role in the third generation's health condition beyond early life and adulthood. As a result, in addition to the scarring effect that is passed down through generations, we are able to present evidence of the existence of selection.

Our results suggest that the effects of *in utero* exposure to economic hardship during pregnancy persist across multiple generations. The impact of exposure differs according to the social class to which the grandmother belonged: among lower social classes, exposure to economic hardship increases longevity, which may reflect the effects of selective mortality. The most vigorous mothers survived and recovered during their infancy and childhood, leading to positive selection among the surviving grandchildren. As a result, overall negative health conditions later in life dominated the stronger portion of the grandchildren's birth cohort. However, among higher social classes, the grandchildren were scarred, and exposure decreased the longevity of the grandchildren.

This article is organized as follows. Section II provides background on our study area, the municipality of Rendalen. Section III presents the data and discusses the variables we used in the analysis. Section IV provides the methodology. Section V outlines the results. Section VI provides a discussion of our findings. Section VII presents the conclusion.

2. Rendalen

Rendalen is an inland parish in southeastern Norway, close to the Swedish border. Its population was approximately 1000 in 1733 and 2000 in 1840. Throughout the period under examination in our study, it was a vast and sparsely populated parish, with an area of more than 4000 km^2.

The main industries of the region were agriculture, animal husbandry, and forestry. Situated in a highland area with farms located 250–540 m above sea level and summer pastures located up to 940 m above sea level, the harvest seasons were significantly shorter here than further south, which had a more favorable agricultural potential. Additionally, according to the parish priest, who left a note when he finalized the 1801 census of Rendalen's population, enough grain could be cultivated only in the most fertile years. Most of the time, local people had to buy grain and spend endless hours collecting moss, brushwood, and leaves as winter fodder for livestock (Sogner, 1979, p. 260).[2]

As the population increased, farms were divided, and new settlements were built in outlying areas by the so-called *rydningsmenn* (settlers). This resulted in increased social cleavage between family households with taxable land (farmers and *rydningsmenn*) and those who lacked any such property (cotters and lodgers) (Sogner, 1979). Rendalen, according to the 1801 population census, comprised 43% farmers, 7% settlers (*rydningsmenn*), and 35% cotters, lodgers 15% (Sogner, 1979, p. 273), making Rendalen, with its two-class society, an interesting case for further elaboration of plausible mechanisms across generations. Overall, between 1733 and 1828, cotters/lodgers had lower fertility rates than farmers, but the former had a prenuptial birth rate twice as high as the latter.

In the archives of Rendalen, we can find documented examples of how higher prices led to economic hardship. During the struggles of 1742 and 1773, local granaries functioned as banks by lending out grain (Sogner, 1979, p. 433). Apart from the fact that this was necessary for survival, it also shows that the residents of Rendalen depended on cash income to pay off these loans. During both crises, the same number of grain barrels was distributed, but the price of grain was 120% higher during the latter crisis. The list of arrears shows that all loans were paid

[2] During years of hardship, bark (outer covering of a tree) was used as substitute for grain (Östlund et al., 2009). According to medical reports from the beginning of the 19th century, bark bread that contained a mix of bark meal and other types of flour did not have any observed negative consequences for a person's health. However, an unbalanced diet of bark bread made of solely bark meal could result in loss of energy, edema, and "narrow chest," and it could be fatal in some cases.

back only weeks after the 1742 crisis, but the list was still full two years after the 1773 crisis. Obviously, the poor were hit the hardest by economic hardship; 60 households, vast majority of those listed, were occupied by cotters, many of whom were registered as destitute.[3]

3. Data

The original Rendalen database covers individual life trajectories that are recorded over the generations from 1733 to 1900. Its main sources are baptism, marriage, and burial entries from church books, and individuals have been manually linked across different sources in accordance with Louis Henry's family reconstitution method (Henry, 1970). In addition to the main sources, information about confirmations, smallpox vaccinations, stillbirths, inbound and outbound migration and decennial nominative population censuses and cadastral registers were included. The construction of the database took place in several stages. First, handwritten family reconstitution cards, which were gathered and carefully stored in shoeboxes by Sølvi Sogner, formed the basis of her doctoral dissertation in 1979 (Sogner, 1979). The database was digitalized and documented in approximately 2000 in a funded project led by Sogner (Gjeldseth, 2000), and it was expanded with data for the period after 1900 by the Norwegian Historical Data Centre in approximately 2010. Today, the Rendalen database is a part of the Norwegian Historical Population Register (Norwegian Historical Data Centre and the National Archive of Norway, 2019).

To understand the association between economic hardship during pregnancy and grandchildren's health condition, we formed a unique dataset of three generations that includes 2070 children, their mothers, and grandmothers (see Fig. 1). We limited our analysis to individuals born before 1840, as the exact ages at death of the individuals who were born later are not known. The ages at death fell dramatically after 1840, as shown in Fig. 1. Consequently, the sample used for the analysis contains 798 grandchildren, 271 mothers, and 170 grandmothers, all of whom were born and died in Rendalen.

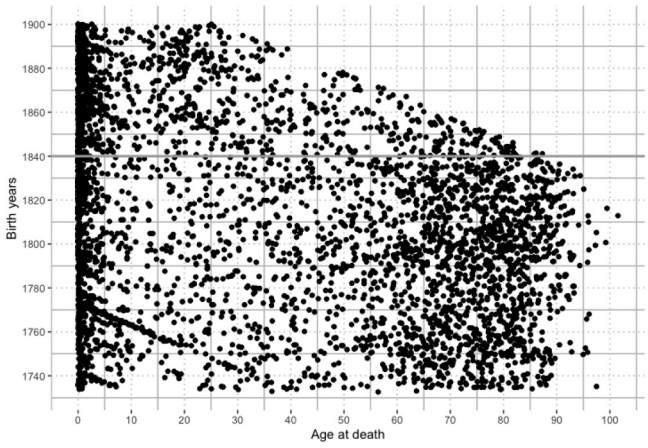

Fig. 1. Birth years and ages at death of three generations. Note: The area under the gray horizontal line displays the individuals in the sample; their ages are between 0 and 103 years, and their birth years were between 1733 and 1840. Our dataset includes 1239 individuals, of whom 798 were grandchildren, 271 were mothers, and 170 were grandmothers.

[3] More specifically, we are talking about three farmers, three settlers, 43 cotters (including five widows), and three lodgers. The likelihood of collecting on the loans was considered slim: 4 of the recipients were dead, 21 of them were described as having a moderate condition, eight were poor, and 23 o were extremely poor (Sogner, 1979, p. 434).

To determine the level of economic hardship faced during the pregnancies of the grandmothers and mothers,[4] we used the annual inflation rates between 1734 and 1840 taken from Grytten's (2018) comprehensive consumer price index (CPI) study for Norway. His study is the most recent publicly available source of data on the Norwegian historical CPI. For the period between 1736 and 1816, his main sources were the Proviant Office, price currents, merchant archives and Wedervang Archive records. Some of these sources were also located near Rendalen (see Appendix A). To establish the price index, he constructed subindices in the structure of commodity and expenditure group series. He weighted[5] commodity and expenditure groups, and 70% of this weighting was food prices (see Appendix A). Therefore, the annual inflation rates were used as an indicator of economic hardship during the period under examination.

3.1. Variables

Our data consist of three generations along the maternal line. The first generation, *grandmothers,* gave birth to at least one daughter who survived into adulthood to give birth to the second generation (*mothers*) and had at least one *grandchild* (third generation).

Rendalen was a rural two-class society in the 18th and late 19th centuries (Bull, 2005; Sogner, 1979). To understand whether the effect of *in utero* exposure to economic hardship during the grandmother's pregnancy on the health of her grandchildren differed by social class, we used the HISCLASS[6] scheme to divide our sample group into two main categories: lower and higher grandparental social class. The lower grandparental social class included mainly lower-skilled and unskilled farmworkers and cotters/lodgers. Farmer grandparents who owned land and other members of the upper classes are classified as part of the higher social class (see Appendix B).

In Table 1, we present the list of variables with their descriptive statistics.[7]

3.1.1. Dependent variable: grandchild's health

We used the age at death as a proxy for the health condition of the mothers and grandchildren: a higher age at death represents a healthier person, and a lower age indicates poor health. This measure is widely used in the literature as an indicator of quality of health (for example, see Kaati et al., 2002 and Lundborg and Majlesi, 2018). We calculated the individuals' ages by subtracting their birth dates from their death dates.

[4] Missing dates and age heaping are well-known challenges when working with historical population data. Therefore, we focused on the year of birth and called that year "during pregnancy." Approximately 10% of the mothers and grandchildren in our sample group were born in January.

[5] The figures regarding commodity and expenditure groups cover the years between 1736 and 1816, and they are presented here as percentage values in parentheses: Grain (12%); flour and bread (14%); vegetables, fruits and berries (5%); dairy products (7%); meat (13%); fish (14%); beverages and tobacco (6%); colonial goods (salt) (5%); clothing and footwear (16%); and fuel and lighting (8%).

[6] "HISCLASS is an international historical class scheme, created for the purpose of making comparisons across different periods, countries and languages". *Maas, I., & van Leeuwen, M. (n.d.). HISCLASS. Retrieved December 12, 2019, from* http://www.hisma.org/HISMA/HISCLASS.html.

[7] We examined correlation indexes to determine the correlations between all of the variables and to detect whether they are all insignificant. We have a multigenerational set of variables, which can pose the risk of multicollinearity. To be on the safe side, we conducted variance inflation factor (VIF) analysis for all models to check the probability of multicollinearity risk in our study. The results of the VIF analysis ranged from 1.016 to 2.491. As a result, we concluded that multicollinearity is not a risk factor in our analysis since the results are close to the smallest possible value for VIF (Purkayastha, 2018).

Table 1
Variable descriptions and summary statistics.

	Lower grandparental social slass					Higher grandparental social class				
	N	Mean	Standard deviation	Min	Max	*N*	Mean	Standard deviation	Min	Max
Health										
Grandchild's health	408	48.6	33.08	0	95.89	390	48.09	32.39	0	101.51
Mother's health	408	70.55	14.57	27.25	96.09	390	70.07	15.42	18.08	93.24
Grandmother's health	408	72.52	13.13	33.65	97.47	390	72.01	15.21	32.75	95.82
Macrolevel variables	*N*	%				*N*	%			
Economic hardship during grandmother's pregnancy										
No (ref)	218	53.4				192	49.2			
Yes	190	46.6				198	50.2			
Economic hardship during mother's pregnancy										
Low annual inflation (ref)	115	28.2				137	35.1			
High annual inflation	293	71.8				253	64.9			
Local-level variables										
Disease environment during grandmother's pregnancy	408	2.97	3.06	0.28	21.2	390	2.88	2.87	0.28	21.2
Disease environment during mother's pregnancy	408	2.14	0.84	0.84	5.29	390	2.11	0.86	0.84	5.29
Individual-level variables										
Parental social class										
High (ref)	122	29.9				325	83.3			
Low	286	70.1				65	16.7			
Illegitimacy of mother										
No (ref)	387	94.9				377	96.7			
Yes	21	5.1				13	0.3			
Illegitimacy of grandchild										
No (ref)	352	86.3				339	86.9			
Yes	56	13.7				51	13.1			
Vaccinated grandchild (smallpox)										
No (ref)	224	54.9				207	53.1			
Yes	184	45.1				183	46.9			
Grandchild's gender										
Male (ref)	215	52.7				212	54.4			
Female	193	47.3				178	45.6			

Note: We divided our sample group based on the grandparent's social class, as defined by HISCLASS. Those whose occupational classification was between 1 and 8 were included in the higher social class, and all others were grouped into the lower social class (see Appendix B.1.). We determined economic hardship by dividing the annual inflation rates between 1736 and 1840 into quartiles; years above and below the upper and lower quartile rates were designated as economic hardship years. The variable of child mortality rate only covers the births and deaths of children between 0 and 9 years old in Rendalen. Vaccination refers to smallpox and indicates the grandchildren who were vaccinated during their life span.

3.1.2. *Independent variables*

Our study focuses on determining whether *in utero* exposure to economic hardship during a grandmother's pregnancy has either a positive culling effect or a transgenerational negative scarring effect on her grandchildren's health conditions. Similar to Van den Berg et al. (2009a, 2009b), we used the annual inflation rates from 1734 to 1840 from Grytten's (2018) study as a primary explanatory variable for understanding the transgenerational mechanism of exogenous shocks from the grandmother to her grandchild as our proxy for economic hardship. We used annual inflation rates outside the interquartile range to determine the years of economic hardship during our time frame. We defined economic hardship as an annual inflation rate above the 3rd quartile (6.9%) or below the 1st quartile (−3.4%) (see Fig. 2 and Appendix A).

We controlled for the disease environment in Rendalen during grandmothers' and mothers' pregnancies (Borrescio-Higa et al., 2019; Quintana-Domeque et al., 2011) using the annual childhood mortality rate[8] (CMR). We also controlled for other individual-level factors known in the literature to affect health and life span, such as parental social class (Currie and Vogl, 2013), illegitimacy[9] of the mother and grandchild (Edvinsson et al., 2008; Lust, 2020; Modin et al., 2008), whether the grandchildren had received a smallpox vaccine[10] (Steckel, 2009; Van den Berg et al., 2009a, 2009b; van Dijk, 2019), and gender (Classen, 2010; Classen and Thompson, 2016). We used baptism records to determine the mother's and grandchildren's illegitimacy and used confirmation records to determine the grandchildren's smallpox vaccination status.

4. Methods

In our study, we hypothesized that there are two main mechanisms by which economic hardship during the grandmother's pregnancy with her daughter can be associated with a grandchild's life span: a positive culling effect and a negative scarring effect through the mother's health condition. Hence, we used mediation analysis (Gunzler et al., 2013; Imai et al., 2010; Thompson et al., 2019). We followed Thompson et al.'s (2019) method and employed the structural equation model (SEM)

[8] Appendix C presents the annual CMR calculation, and Appendix C.1 shows the CMR fluctuations between 1741 and 1900.

[9] Illegitimate: "not recognized as lawful offspring" *The Merriam-Webster.com Dictionary, Merriam-Webster Inc.,* https://www.merriam-webster.com/dictionary/illegitimate. *Accessed December 12, 2019.*

[10] In 1810–11, the smallpox vaccine was mandated by law, and no one could get married or be confirmed in church or attend school unless they had a certificate of vaccination or had contracted smallpox naturally (Jensen, 2009). The church kept confirmation records after children turned 15; therefore, we do not know precisely when the children were vaccinated. Due to uncertainty regarding the children's ages at vaccination, we used the smallpox vaccination variable in our model only for those aged 15 years and older (see Appendix D).

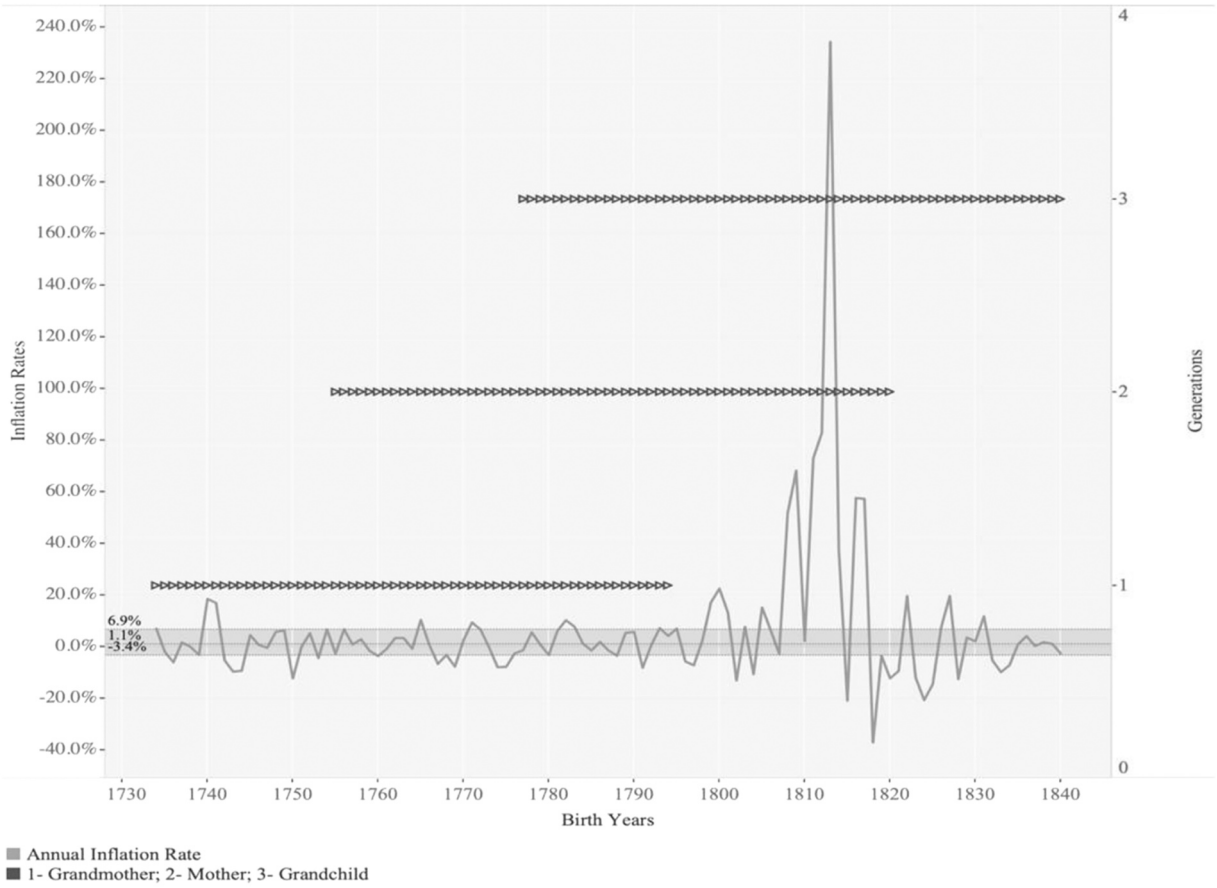

Fig. 2. Annual inflation rate and generations, from 1734 to 1840. Note: Triangles represent the birth years of the generations; 1 shows grandmothers born between 1734 and 1794, 2 shows mothers born between 1755 and 1820, and 3 shows grandchildren born between 1777 and 1840. The line graph shows the annual inflation rates over the years covered by this study. These inflation rates were calculated by the CPI data taken from Grytten's (2018) research. We divided the annual inflation rates into quartiles: the 1st quartile was − 3.4%, the 3rd quartile was 6.9%, and the median annual inflation rate was 1.1%. We used annual inflation rates outside of these quartiles, namely, below the 1st quartile and above the 3rd quartile, to determine the years of economic hardship.

approach under the sequential ignorability assumption.[11] As Gunzler et al. (2013) highlight, SEM is a conceptual model for capturing the complex and dynamic relationships within the network of observed and unobserved variables. The logic of the model is to use a path diagram and a system of linked regression-style equations. As a result, SEM simplifies the testing of transgenerational transmission of economic hardship because it is designed to test more complicated mediation models in a single analysis (Gunzler et al., 2013).

We examined the influence of the mother's health condition on the grandchild's health by separating the total effect of *in utero* exposure to economic hardship during the grandmother's pregnancy on the grandchild's health condition into two determinants: the culling effect, reflected by the average direct effect, and the scarring effect, expressed by the average mediation effect in the SEM. Thus, we fitted two models: a first ordinary least squares regression model that regressed the mother's health condition on economic hardship during the grandmother's pregnancy (*a*-path) and a second ordinary least squares regression model that regressed the grandchild's health condition on economic hardship during the grandmother's pregnancy (c'-path) and the mother's health condition (*b*-path). The models are presented below:

$$Y = i_1 + cX + e_1 \quad (1)$$

$$Y = i_2 + c'X + bM + e_2 \quad (2)$$

$$M = i_3 + aX + e_3 \quad (3)$$

$$Y = (i_2 + bi_3) + (c' + ab)X + (e_2 + be_3) \quad (4)$$

The basic conceptual framework of a mediation process is illustrated in Fig. 3. The outcome variable, *Y*, denotes the grandchild's health condition; the explanatory variable, *X*, represents economic hardship during the grandmother's pregnancy; and the mediator variable, *M*, represents the mother's health condition. In the Eqs. (1)–(3), the intercepts are denoted by i_1, i_2, and i_3. *c* indicates the coefficient between *X*

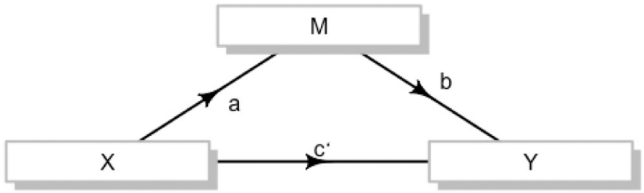

Fig. 3. The conceptual mediation model path diagram. Note: X denotes an explanatory variable, economic hardship during grandmother's pregnancy; M represents the mediator, the mother's health condition; and Y represents the outcome, the grandchild's health condition. c′ is for a direct impact on the coefficient between X and Y, fitted for M. a indicates the coefficient between X and M. b represents the effect of M for the explanatory variable on the adjuster, Y.

[11] "The treatment is firstly assumed to be ignorable given the pre-treatment covariates, and then the mediator variable is assumed to be ignorable given the observed value of the treatment as well as the pre-treatment covariates" (Imai et al., 2010).

and Y, and the total effect, $c^{'}$, indicates the average direct effect on the coefficient linkage of X on Y, fitted for M. a indicates the coefficient between X and M. b represents the effect of M for the explanatory variable on the adjuster Y. According to the sequential ignorability assumption, residuals ($e_1, e_2,$ and e_3) are not correlated with the variables, and they are independent of one another (*the results of the sensitivity analysis also support this; see Appendix E*) (Imai et al., 2010; Zhang et al., 2016). M is a subset of Y, and to obtain the mediation analysis equation in Eq. (4), we replaced M in Eq. (2) with Eq. (3). In Eq. (4), the product coefficient of ab is a denotation of an average mediation effect. We calculated the total effect by multiplying the a-path coefficient by the b-path coefficient and adding the $c^{'}$-path coefficient ($c^{'} + ab$). Like Thompson et al. (2019), we used 5000-simulation bootstrapping to increase estimation accuracy at 95% confidence intervals since the average mediation effects tend to be skewed.

With this approach, we tested the existence of a mediation effect that fits the three linear regressions separately and tests the null hypothesis; $a = 0$, $b = 0$, $c = 0$ and $c^{'} = 0$ (Imai et al., 2010). To conclude that the transgenerational effect of economic hardship during a grandmother's pregnancy affects consecutive generations as a culling effect, we must determine that the c'-path p-value is significant and that the coefficient is greater than zero. Conversely, to determine the transgenerational scarring effect through the mediating effect of the mother's health, the model must meet the following criteria: First, the confidence interval for the scarring effect should not contain zero; second, the p-values of the a-path and b-path must be significant; and third, the p-value of the c'-path must be insignificant (Thompson et al., 2019).

5. Results

As a result of the mediation analyses, we found evidence of both the positive culling effect in the lower social class and the negative scarring effect in the higher social class. As shown in Fig. 4(A), the average life span of grandchildren whose mothers were in the lower social class and were born in years of economic hardship was 10.2 years longer than that of grandchildren in the lower social class whose mothers were born in economically better-off years. Notably, in our model, we found a significant result on the c'-path, but not a significant effect on the a-path or the b-path. Hence, we see that the effect of economic hardship during the grandmother's pregnancy "skipped" the mother's generation, which provides evidence in support of the culling effect.

However, first, we need to state that unlike in Fig. 4(A), Fig. 4(B) shows a negative and significant relationship between economic hardship during the grandmother's pregnancy and the grandchild's health condition for the higher social class. For the higher social class, the average life span of a grandchild whose grandmother endured economic hardship during her pregnancy was 1.3 years shorter than that of a grandchild whose grandmother was not pregnant during an economically challenging year. Second, we see that economic hardship during the grandmother's pregnancy had a significant and negative impact on her daughter's life span, shortening it by 4.2 years. Third, we found that a one-unit increase in the mother's health condition could result in a 0.3-year increase in the life span of her child. A sensitivity analysis yielded similar results (see Appendix E). As a result, the criteria for the transgenerational scarring effect were met by significant a-path and b-path and a nonsignificant c'-path. This result shows a transgenerational negative scarring effect of economic hardship *in utero* for the grandchild of a grandmother from the higher social class.

We also obtained some other expected results from regression models (see Table 2). We found that within the higher social class, a worse disease environment during the grandmother's pregnancy resulted in a decrease of the grandchild's average life span by 1.1 years, while greater *in utero* exposure to disease environment during the mother's pregnancy resulted in a decrease of the grandchild's average life span by 5.3 years. In addition, being born out of wedlock had a significantly negative impact on grandchildren's life span, regardless of their grandmother's social class. Additionally, the impact of being a woman born into a lower social class was statistically significant; their average life span was 6.5 years longer than that of men.

6. Discussion

In our study, we used rich 18^{th-} and 19th-century individual-level data from the municipality of Rendalen in Norway, one of the oldest data sets used in transgenerational studies in the social sciences. We were able to identify families' social classes using individual-level data that includes three generations, and we investigated the transgenerational effect of *in utero* exposure to economic hardship as an exogenous shock during the grandmothers' pregnancy. Our study provides evidence to support the hypothesis that economic hardships during a grandmother's pregnancy have a transgenerational impact on her grandchildren's health condition by focusing on the role of the relative strength of culling and scarring.

Our results indicate that for a grandchildren born to a family in the lower social class, there is a positive and significant relationship between economic hardship during the grandmother's pregnancy and the grandchild's health. To test the robustness of our finding, we reduced our sample size by age at death, based on the work of Lindeboom and van Ewijk (2015) (see Appendix D). In conclusion, considering our findings in conjunction with the results of parallel testing of subsets of samples, it appears unlikely that the culling effect occurred solely by chance.

The question is as follows: when a mother is born in a year marked by economic hardship, why did low-status grandmothers' grandchildren live longer? In the literature, the results of most intergenerational studies (e.g., Lee, 2014b; Cook et al., 2019) show the consecutive effects of exogenous influences across generations. Additionally, as Van Den Berg and Pinger (2016) stated, it is crucial to distinguish this transgenerational influence from parental effects. Following this, one plausible explanation is that the observed economic hardship resulted in a positive selection among grandmother births in the lower social class,

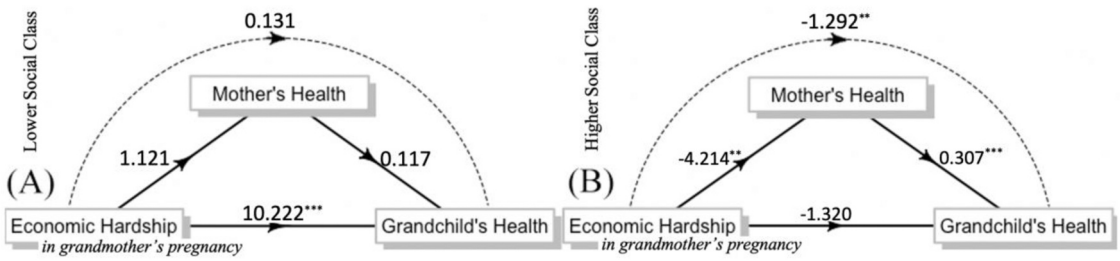

Fig. 4. The results of the mediation analysis for all ages presented in the models. Note: (A) presents a positive culling effect of economic hardship during the grandmother's pregnancy on the grandchildren's health condition for the grandmothers in the lower social class; (B) illustrates the transgenerational scarring effect of economic hardship during the grandmother's pregnancy in the higher social class. Dashed lines demonstrate the result of multiplying the a-path coefficient by the b-path coefficient. *p < 0.1; * *p < 0.05; * **p < 0.01.

Table 2
The intergenerational effect of *in utero* exposure on health conditions.

	Dependent variable: Grandchild's health		*Dependent variable: Mother's health*	
	Lower social class (1)	Higher social class (2)	Lower social class (3)	Higher social class (4)
Grandmother → Grandchild				
Economic hardship	10.221***	-1.320		
	(3.935)	(3.648)		
Grandmother's health	0.099	-0.075		
	(0.125)	(0.114)		
Disease environment	-0.288	-1.072*		
	(0.536)	(0.585)		
Parental social class	-0.460	-0.976		
	(3.506)	(4.301)		
Illegitimacy of the mother	-1.097	-12.642		
	(7.198)	(8.901)		
Birth year time trend	0.505*	-0.022		
	(0.284)	(0.278)		
Mother → Grandchild				
Economic hardship	-1.807	-0.654		
	(3.618)	(3.526)		
Mother's health	0.117	0.307***		
	(0.109)	(0.105)		
Disease environment	0.814	-5.265***		
	(2.015)	(1.927)		
Illegitimacy of the grandchild	-25.965***	-18.723***		
	(4.817)	(4.911)		
Female	6.517**	-2.948		
	(3.161)	(3.185)		
Birth year time trend	-0.823***	-0.353		
	(0.281)	(0.284)		
Grandmother → Mother				
Economic hardship			1.121	-4.214**
			(1.774)	(1.782)
Grandmother's health			0.181***	0.080
			(0.056)	(0.055)
Disease environment			0.356	-0.376
			(0.242)	(0.287)
Illegitimacy of the mother			2.068	-4.918
			(3.249)	(4.365)
Birth year time trend			-0.118**	0.048
			(0.058)	(0.061)
Constant	73.938***	82.739***	62.440***	64.236***
	(19.658)	(18.178)	(4.968)	(4.849)
Observations	408	390	408	390
R^2	0.128	0.114	0.037	0.029
Adjusted R^2	0.102	0.086	0.025	0.017
Residual std. error	31.350 (df = 395)	30.970 (df = 377)	14.387 (df = 402)	15.290 (df = 384)
F statistic	4.839*** (df = 12; 395)	4.034*** (df = 12; 377)	3.112*** (df = 5; 402)	2.310*** (df = 5; 384)

Note: We examined two different sets of models based on the differences in dependent variables; one was the grandchild's health condition, and the other was the mother's health condition. According to the grandparents' social class, we divided the sample groups into higher and lower classes. Grandmother's health, mother's health, and disease environment were continuous variables. We used the smallpox vaccination variable in our model only for those aged 15 years and over (see Appendix D). Standard errors are presented in parentheses;
* $p < 0.1$;
** $p < 0.05$;
*** $p < 0.01$.

where the subsequent birth-cohort outcomes (mothers) were improved by eliminating the weakest individuals. According to Almond and Currie (2011), survivors of adverse fetal events are generally positively selected because mortality tends to remove those in poor health, and the positive culling effect among survivors was strong enough to dominate the negative effect of scarring. Another potential explanation for the transgenerational effect in the lower social class is that the grandmother's exposure to economic hardship during pregnancy has nonbiological consequences, such as effects on behaviors toward offspring, education within the household, and the model of upbringing adopted (Van Den Berg and Pinger, 2016). However, it is challenging to conclude that this transmission from grandmother to grandchild is due to biological or cultural inheritance.

For the higher social class, economic hardship during the grandmother's pregnancy had a significant and negative effect on the grandchild's health condition. This result shows evidence of the negative scarring effect. If a grandmother from a high social class gave birth during a year of economic hardship, and her daughter could survive beyond reproductive age, then the adverse effect of economic hardship might be expected to be passed on to the grandchild. We can argue here that this negative scarring effect is in line with the fetal origin hypothesis (Almond et al., 2018; Bruckner and Catalano, 2018) regarding a stress mechanism during pregnancy. Here, the transgenerational transmission of health is consistent with Classen and Thompson (2016) and Coneus & Spiess (2012). Additionally, in Serpeloni et al. (2017), they underline the biological mechanism that has a mediating role in the transmission of stress to subsequent generations. Here, epigenetic inheritance can be described as the development of germ cells (future grandchild) in the fetus (mother), which develop in the body of the grandmother.

On the other hand, because our mediation models were unlikely to account for all potential confounders, we provide evidence of

associations rather than causal relationships. Although the literature claims that mediation analysis is based on the assumption that no confounders influence both the mediator and the outcome (Coffman, 2011), mediation analysis has the same assumptions as standard regression (Thompson et al., 2019). As Thompson et al., (2019, p. 113) said, "while a more nuanced, less certain interpretation is warranted, it does not mean that mediation analyses are ipso facto not worth undertaking". Even if we do not have the results to present causal chains based on the mediation analysis in this study's findings, the associations we found will support future research on transgenerational studies.

Furthermore, broader and more current data covering multiple generations may help to establish a higher degree of accuracy and provide new evidence on the effects of earlier generations' environmental exposure on their descendants.

7. Conclusion

Our study investigated whether *in utero* exposure to economic hardship during a grandmother's pregnancy has a transgenerational effect on her grandchildren's health condition. We used rich historical individual data collected for the Rendalen municipality in Norway in the 18th and 19th centuries. One of the most important findings to emerge from our study is that among grandchildren born to a family from the lower social class, there was a positive and significant relationship between economic hardship during the grandmother's pregnancy and the grandchild's life span. This discovery is significant evidence of a positive culling effect in the context of transgenerational transmission. Additionally, the findings show that economic hardship had an impact not only on the lower social class but also had a negative scarring effect on subsequent generations of the higher social class. These findings provide important insights into the three-generation pathway that was studied to show how *in utero* economic hardship influenced consecutive generations' health conditions. Future research can also provide new evidence regarding the impact of previous generations' environmental exposure on their descendants by using broader and more recent data covering several generations.

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CRediT authorship contribution statement

Emre Sari: Conceptualization, Methodology, Formal analysis, Data Curation, Writing - Original Draft, Visualization, Project administration. **Mikko Moilanen:** Conceptualization, Supervision, Resources, Writing - Review & Editing. **Hilde Leikny Sommerseth:** Resources, Data Curation, Writing - Review & Editing.

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Declarations of interest

None.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.ehb.2021.101060.

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Appendix to:

Transgenerational Health Effects of *In Utero* Exposure to Economic Hardship: Evidence from Preindustrial Southern Norway

Appendix A. Annual Inflation Rates from 1734 to 1840 and Economic Hardship Years

This study used the annual inflation rates between 1734 and 1840 that were shared in Grytten's (2018) study on Norway's comprehensive CPI for the period between 1492 and 2017. His study is the most recent publicly available source of data on Norway's historical CPI.

Grytten (2018) used the private archives of merchants from 13 different locations: Tromsø, Oslo, Stange, Bergen, Trondheim, Grundset, Kristiansand, Ringsaker, Fredriksvern, Akershus, Fredrikstad, Kongsberg, and Halden (Grytten, 2003). Rendalen is approximately 120 km from Stange and approximately 90 km from Grundset and Ringsaker by air. Additionally, Grytten weighted[1] commodity and expenditure groups, including grain (12%); flour and bread (14%); vegetables, fruits and berries (5%); dairy products (7%); meat (13%); fish (14%); beverages and tobacco (6%); colonial goods (salt) (5%); clothing and footwear (16%); and fuel and lighting (8%). He took into account products such as refined sugar, plain sugar, salt, vinegar, rice, peas, rye, rye flour, barley, barley flour, fresh cod, and stockfish (Grytten, 2018). In total, in his study, he took into account the prices of rye, wheat, barley, oats, potatoes, peas, flax, hemp, salt, iron, spirits, tar, wool, herring, and stockfish (dried cod). Among these, all but iron and tar were essential consumption goods during the period (Grytten, 2003).

Grytten (2018, p. 50) characterizes the period between 1700 and 1820 as "turbulent economy- and inflationary-wise". Additionally, recognizing that price stability is the norm for Norway, Qvigstad (2005) divided economic conditions into two categories, normal and abnormal, from a historical perspective (1667-2004). His abnormal scenario covers both inflation and deflation. He defined abnormality as an annual inflation rate above 5% or an annual deflation rate above 5%. However, instead of using a predefined static cutoff point to identify economic condition, we used annual inflation rates beyond the interquartile range to determine the years of economic hardship during our time frame of interest.

[1] The commodity and expenditure group figures, which cover the period of 1736-1816, are shown as percentages in parentheses.

Appendix B. Division of Social Class: Lower & Higher

The occupation information for the grandparents and parents was gathered from censuses (1801, 1845, 1865, 1875, 1900), parish registers (1733-1900), and baptism records (1733-1900) in Rendalen (Norwegian Historical Data Centre, the National Archive of Norway, et al., extracted in 2019). We linked these datasets based on personal and family ID numbers and identified every possible piece of occupational information for the included individuals. To thoroughly identify this information, we went through all of the missing variables line by line and filled in the missing variables with marriage records (1733-1900). Considering the conditions of the period of interest, we mainly had information for men, but farming was a joint husband-wife enterprise. Therefore, most of the occupational information obtained was for grandfathers. However, in cases where variables were still missing after the linkage and control process, we also used the occupational information of grandmothers, if any was available, for very few observations.

On the other hand, in some cases, we obtained more than one piece of occupational information for an individual as a result of linking various datasets that covered long- and short-term. We used the occupational information for the period closest to the mother's pregnancy year. Therefore, for a grandparent who had more than one grandchild, occupational information could vary according to the grandchildren's birth years. Finally, we had adequate social class data according to HISCO-based classification schemes, or HISCLASS, which is a 12-category occupational classification scheme based on skill levels (Bull, 2005; Dribe & Helgertz, 2016; Van Leeuwen & Maas, 2011). In Appendix B.1., occupational classes are presented by the grandparent's occupational classes for each grandchild's birth year.

Appendix B.1. Social class distinctions based on HISCLASS in Rendalen among grandparents.

HISCLASS	Grandparental Social Class	Definitions	Grandchildren
1	Higher Level	Higher Managers	8
2	Higher Level	Higher Professionals	11
3	Higher Level	Lower Managers	2
4	Higher Level	Lower Professionals	4
5	Higher Level	Lower Clericals	0
6	Higher Level	Foremen	14
7	Higher Level	Skilled Workers	14
8	Higher Level	Farmers	337
9	Lower Level	Lower-Skilled Workers	7
10	Lower Level	Lower-Skilled Farm Workers	205
11	Lower Level	Unskilled Workers	3
12	Lower Level	Unskilled Farm Workers	193
		Total	**798**

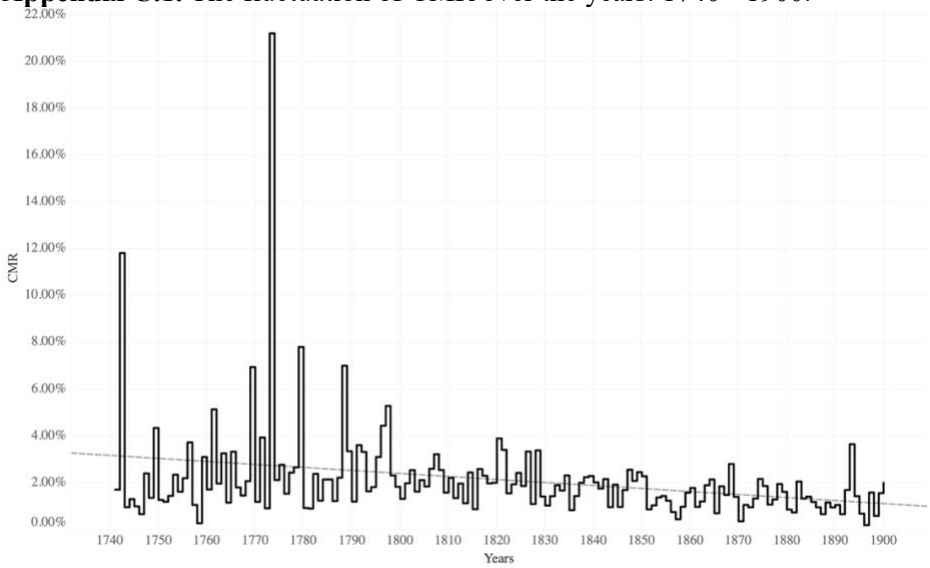
Appendix C. Mortality among Children Aged 0-9 Years: Childhood Mortality Rate

Similar to Borrescio-Higa et al.'s (2019) study, we tested et disease environment in childhood by using the childhood mortality rate (CMR) as a proxy during and around the pregnancy year. The CMR was calculated by using both burial (1733-1900) and baptism (1733-1900) records from Rendalen. In this study, we focused on mortality in children aged 0 to 9 years to cover all-cause mortality during childhood (Yu et al., 2016). Yu et al. (2016) addressed certain conditions within this age range in their study, such as neoplasms, diseases of the nervous system, and transport accidents, as well as conditions originating during the prenatal period, congenital malformations, and sudden infant death syndrome. This provided us with a better understanding of the disease environment during childhood. CMR was calculated by dividing the total childhood (0-9 years) deaths in a given year (D_{0-9}) by the total births in the preceding nine years (T_{0-9}), including the year of death, and then multiplying the outcome by 100.

$$CMR = \frac{\sum D_{0-9}}{\sum T_{0-9}} \times 100$$

The annual CMR variables are not related to economic hardship years during and around the grandmother's and the mother's pregnancies. The correlation results for these variables are nonsignificant: -0.18 for the grandmother's pregnancy year and -0.15 for the mother's pregnancy year.

Appendix C.1. The fluctuation of CMR over the years: 1740 - 1900.



Note: The dashed line is drawn to show the annual CMR trend.

Appendix D. The Results of Mediation Analysis for the Subsamples

Empirical analyses were performed to discuss the mechanisms underlying health persistence across generations. First, we tested the whole sample group, including infant deaths, and presented and discussed our empirical findings in the Results section. To control for later-life effects, we narrowed our sample size based on their age at death, and we present the results here. Using the same procedure as Lindeboom & van Ewijk (2015), we excluded infants who died before the age of 1 year to focus on the long-term health conditions of the grandchildren rather than the immediate effects. Additionally, to determine the effect of the explanatory variable on a grandchild's later-life health condition, we considered 15 years, which was the average age at beginning work and getting married during the period of interest, as the threshold age (Rahikainen, 2001).

The results of all of the mediation analyses are shown in Appendix D.1. The results for all age groups of grandchildren whose grandparents were from lower social classes show that there is a positive culling effect from the grandmother to the grandchild. For children aged one year and older and those aged 15 years and older in the lower social class, we found significant culling effects; economic hardship during and around the mother's birth increased the child's life span by 6.0 and 7.1 years, respectively. On the other hand, we did not find any significant difference between better-off and more challenging economic years for the age-constrained sample groups whose grandparents were from the higher social class. Additionally, we obtained another interesting result: Having received a smallpox vaccination was significantly associated with a 7.5-year increase in the life span of grandchildren whose grandparents were from a lower social class.

Appendix D.1. Average mediation effect, average direct effect, and total effect of economic hardship during and around the grandmother's pregnancy.

		Lower grandparental social class		Higher grandparental social class	
		Estimate	95% Confidence Interval	Estimate	95% Confidence Interval
All ages included	Average Mediation Effect	0.131	-0.353 – 0.810	-1.292**	-2.953 – 0.100
	Average Direct Effect	10.222***	2.709 – 17.860	-1.320	-8.542 – 5.880
	Total Effect	10.353***	2.802 – 18.050	-2.612	-9.850 – 4.470
	Prop. Mediated	0.013	-0.047 – 0.110	0.494	-4.544 – 4..860
	N	408		390	
Aged one and older	Average Mediation Effect	0.260	-0.424 – 1.190	-0.544	-2.076 – 0.610
	Average Direct Effect	5.963*	-0.610 – 12.760	-2.429	-8.652 – 3.540
	Total Effect	6.223*	-0.284 – 13.050	-2.973	-9.140 – 2.840
	Prop. Mediated	0.042	-0.159 – 0.390	0.183	-2.396 – 2.990
	N	340		318	

Aged 15 and older	Average Mediation Effect	0.134	-0.517 – 0.950	0.350	-0.645 – 1.690
	Average Direct Effect	6.798***	1.993 – 11.830	-2.576	-7.611 – 2.220
	Total Effect	6.932***	2.130 – 11.830	-2.226	-7.275 – 2.670
	Prop. Mediated	0.019	-0.092 – 0.170	-0.157	-2.089 – 2.290
	N	296		289	

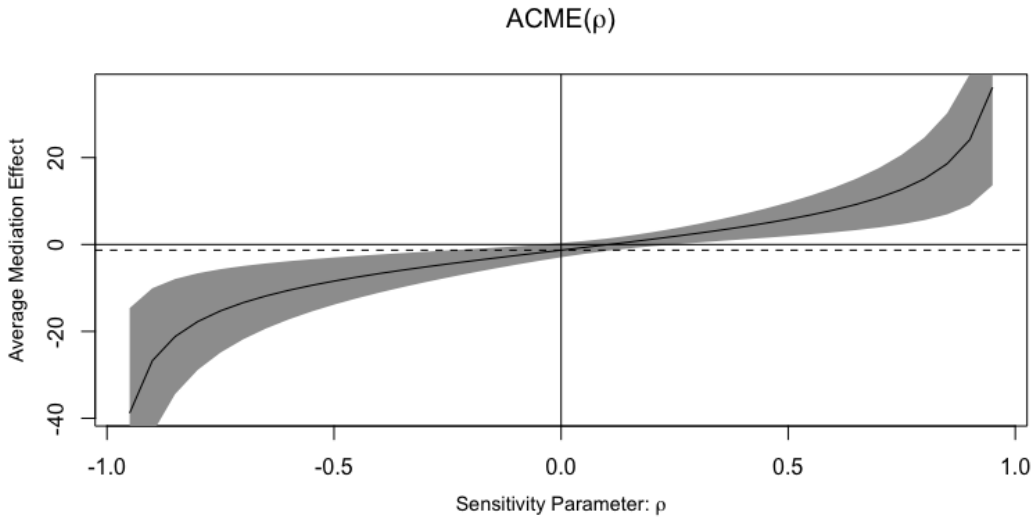
Note: The criteria for mediation are met for the "all ages included" sample group with higher grandparental social class. The significant results for the average direct effect show that economic hardship during the mother's birth year affects the grandchild's life span, as mediated by the mother's life span. Simulations: 5000. Nonparametric bootstrap confidence intervals with the percentile method. $^{*}p<0.1$; $^{**}p<0.05$; $^{***}p<0.01$

Appendix E. Sensitivity Analysis

As previously mentioned, one major drawback of the approach used for our research is the presence of an unmeasured confounder, which might confound the relationship between the mediator and outcome (Imai, Keele, & Yamamoto, 2010). Therefore, we needed to conduct a sensitivity analysis to examine whether the results were robust against the violation of the sequential ignorability assumption (Imai, Keele, & Tingley, 2010; Tingley, Yamamoto, Hirose, Keele, & Imai, 2014; Zhang et al., 2016). Such analyses are particularly useful for determining whether unmeasured confusion could have a significant impact on the outcomes of both the average direct effect and average mediation effect (VanderWeele, 2016). In the context of the SEM, a sensitivity analysis is based on the correlation between the residuals of the mediator (e_3) and the outcome (e_2).

Appendix E.1., presents the result of a sensitivity analysis referring to residual correlation with all ages included in a model of higher grandparental social class. It shows that the conclusion regarding the direction of the ACME under the sequential ignorability assumption will be preserved unless p is more significant than 0.15. The confidence interval covers only the zero value when $-0.896 < p < 0.565$, and this result holds even after the sampling variability is taken into account. Therefore, we can say that the original finding of positive ACME is robust despite the violation of sequential ignorance in the context of the SEM.

Appendix E.1. Sensitivity analysis for the health outcome model of the higher social class.



a

Note: The graph illustrates the point by plotting the estimated ACME and its 95% confidence intervals. The solid line presents the estimated ACME at different p-values. The dashed line is drawn at the point estimate of the mediation effect for $p = 0$. The gray region represents the 95% confidence interval for each value at each value of p.

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Paper II

Role of grandparents in risky health behavior transmission: *A Study on smoking behavior in Norway*

Role of Grandparents in Risky Health Behavior Transmission: A Study on Smoking Behavior in Norway

By EMRE SARI, MIKKO MOILANEN, AND MAARTEN LINDEBOOM*

Exploring the role of grandparents in the intergenerational transmission of risky health behaviors, specifically smoking, this study aims to examine the differential influence of maternal and paternal grandparents on their grandchildren's smoking behavior in adulthood. Utilizing the Tromsø Study's unique three-generational dataset from Tromsø, Norway, we employ a control function approach to establish causal relationships. The findings show a significant matrilateral bias, revealing that maternal grandparents' smoking behavior has a notable negative direct effect on the probability of their grandchildren's smoking. No such influence is observed in the case of paternal grandparents. Moreover, an indirect transmission of grandparental smoking behavior from grandparents to grandchildren through parents is identified, increasing on grandchildren's smoking probability. These results underscore the necessity of incorporating the influential role of grandparents, in crafting public health policies and family-centered interventions for tobacco use.

JEL: I10, I12, J24, Z13

Keywords: Intergenerational transmission, Risky health behaviors, Tobacco smoking, Grandparents' influence, Matrilateral bias

I. Introduction

Lifestyle diseases, characterized by unhealthy habits such as poor diet, excessive alcohol consumption, and smoking, have overtaken infectious diseases as the leading cause of death in high-income countries like Norway (Cappelen et al., 2020; Ritchie & Roser, 2018). In contrast to low-income countries battling infectious diseases, high-income countries contend with chronic diseases induced largely by daily habits that compromise public health (Cawley & Ruhm, 2012). Smoking, in particular, is a major health risk that profoundly affects individual health and the economy at large (Qin et al., 2016). Remarkably, smoking accounts for 1.7 years of the 8-year difference in life expectancy between

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the lower and upper-income quartiles in men and 1.2 years of the 6-year difference in women in Norway (Kinge et al., 2019).

However, the understanding of these behaviors is not just about their impact but also their origins. Economic theory often views these behaviors as underpinned by differences in time and risk preferences, representing a trade-off between immediate gratification and future well-being (Cutler & Glaeser, 2005; Miura, 2019). Furthermore, these preferences and behaviors can be transmitted across generations, from parents to children (Brown & van der Pol, 2015). For instance, children observe the social functionality of smoking through the habits of their parents or friends (El-Amin et al., 2015).[1] Consequently, if at least one parent smokes, children may develop an inclination towards smoking out of curiosity, even if they don not fully comprehend the consequences (Bantle & Haisken-DeNew, 2002).

The mechanisms behind the intergenerational transfer of these behaviors are multi-faceted, involving both genetic and environmental factors. Specific genes have been associated with time and risk preferences (Brown & van der Pol, 2015), and shared genetic factors contribute to the relationship between parental and child smoking (Aydogan et al., 2021; Duarte et al., 2016). Concurrently, cultural factors such as values, beliefs, and attitudes significantly contribute to the intergenerational transmission of behaviors (Brown & van der Pol, 2015). Parents have the ability to influence these cultural factors, which in turn influence socio-emotional development (Zeng & Xie, 2014). Two prominent explanations behind this transmission are altruism and self-interested interaction (Brown & van der Pol, 2015).

As an extension of the parental investment concept (Trivers, 1972), the grandparental investment theory describes the resources—care, time, emotional support, and financial assistance—provided by grandparents to their grandchildren (Danielsbacka et al., 2015).[2] These investments can directly or indirectly benefit their grandchildren, serving as vital factors in human capital endowment. As Solon (2018) postulates, this comes not only through genetic inheritance but also through cultural transmission, with parental and grandparental role modeling playing a critical role.

Grandparents hold a unique position in the familial structure, especially when it comes to passing on cultural norms and behaviors, including smoking. Given that children exposed to their parents' second-hand smoke are more likely to smoke themselves (Gottfredson et al., 2017), grandparents' role in influencing their grandchildren's behavior and thus contributing to the transmission of smoking across generations becomes an interesting area of exploration. Grandparents' altruistic roles not only directly influence their grandchildren but also indirectly shape the next generation's norms and behaviors, establishing a pattern of intergenerational transmission (Lindahl et al., 2015; Solon, 2014).

Many studies show that maternal grandmothers provide more care and resources than

[1] El-Amin et al. (2015) find that there is a strong association between both maternal and paternal grandmothers' smoking and their offspring's smoking behavior. They conduct a mediation analysis to determine the effect of grandparents' smoking on their children's smoking behavior.

[2] Danielsbacka et al. (2015) draw attention to the fact that natural selection tends to reward actions or behaviors that have a basis in genetics and improve overall inclusive fitness.

paternal grandmothers, and numerous studies have demonstrated this matrilateral bias in contact, childcare, and emotional intimacy (see, e.g., Bishop et al. (2009); Coall & Hertwig (2010); Daly & Perry (2017); Lehti (2020); Sadruddin et al. (2019); Tu et al. (2021)). This asymmetry is the matrilateral bias in grandmaternal investment. Coall & Hertwig (2010) emphasize that the kin selection theory can explain the matrilateral bias in grandparental investment. According to the kin selection theory, also known as Hamilton's rule, psychological adaptations may have evolved to regulate investment in grandchildren in response to genetic certainty (Bishop et al., 2009; Coall & Hertwig, 2010). Parental uncertainty can impact how parents and grandparents invest in the next generations. This uncertainty is especially significant from the perspective of fathers and paternal grandparents, who face a double risk (Heijkoop, 2010). Fathers may have doubts about their children's paternity, and paternal grandparents might question whether their son's children are indeed their genetic descendants.

On the other hand, mothers and maternal grandparents typically face less uncertainty about genetic lineage, as they can be confident their children and grandchildren are their direct descendants. However, factors beyond genetic certainty, such as cultural norms and societal expectations, can influence their behaviors and choices regarding the next generations (Danielsbacka et al., 2015; Lehti, 2020). Based on these earlier studies, we anticipate that maternal grandparents exert a stronger influence on their grandchildren than paternal grandparents and that this influence has a negative effect on the probability of smoking.

The trend of increased life expectancy has amplified grandparents' role in their grandchildren's lives, offering more opportunities for interaction and influence (Vandewater et al., 2014). However, research on the transmission of smoking across three generations is sparse (Danielsbacka et al., 2015). Existing literature establishes the correlation between parental and offspring's smoking behavior (Gottfredson et al., 2017; Hübler & Kucher, 2016; Kalmijn, 2022; Ren et al., 2020; Rodríguez-Planas & Sanz-de Galdeano, 2019), but comprehensive evidence to discern whether smoking behavior is directly transferred from grandparents to offspring, or indirectly via parental smoking, is still lacking. While past research has extensively explored the influence of parental smoking on their children (Duko et al., 2021; Leonardi-Bee et al., 2011), the role of grandparents remains inadequately examined. The current study seeks to fill this gap by addressing the research question: *Do grandparents, specifically maternal grandparents, significantly influence their grandchildren's smoking behavior?* We address this question by examining the direct influence of grandparents' smoking behavior during the time they were raising their own children. The key hypothesis is that the adult smoking behavior of grandchildren may be directly influenced by their grandparents' past smoking behavior, independent of their parents' smoking behavior.

Central to our study are two primary theoretical foundations that shape the transmission of smoking behavior from grandparents to grandchildren: Social learning theory and health behavior models. Social learning theory, advanced by Bandura (1971), is grounded in the principle that individuals acquire new behaviors by observing and modeling the actions of others. This theory breaks from the conventional understanding of

learning as a direct result of conditioning, positing instead that much of human learning occurs in a social context (Bandura, 2001). Social learning theory is based on several key concepts, including attention, retention, reproduction, and motivation. For learning to take place, individuals must first pay attention to the observed behavior. They must then be able to remember what they have observed, be capable of reproducing the behavior, and must have sufficient motivation to carry out the behavior. Social learning theory provides valuable insights into how smoking behaviors can be adopted by children observing their parents, friends, or even grandparents (Simons-Morton & Farhat, 2010). When children observe adults smoking, they may perceive it as normal, adult-like behavior and may be motivated to try it themselves out of curiosity or a desire to emulate adult behaviors, even if they do not fully understand the potential health consequences (Bantle & Haisken-DeNew, 2002; Conrad et al., 1992; Gugushvili et al., 2018; Purohit, 2022).

On the other hand, health behavior models have significant implications for understanding the potentially harmful effects of smoking behavior across generations, particularly by emphasizing the role of personal beliefs, attitudes, and perceptions in shaping health-related behaviors. Two important frameworks in this category are the Health Belief Model (Rosenstock et al., 1988) and the theory of planned behavior (Ajzen, 1991). The health belief model helps to examine the direct influence of grandparents' smoking on their grandchildren and suggests that people's health-related actions depend on their beliefs about health problems, perceived benefits and disadvantages, and barriers to action (Rapoff et al., 2023). In the case of smoking, seeing their grandparents smoke could make grandchildren aware of the adverse health effects of smoking. This awareness might discourage them from smoking, but on the other hand, they might also normalize smoking by seeing it as a regular habit of their grandparents, despite being informed about its health risks. Meanwhile, the theory of planned behavior complements our understanding by positing that smoking behavior and intentions are dictated by attitudes toward the behavior, subjective norms, and perceived behavioral control (Rapoff et al., 2023). Consequently, grandchildren's reactions could range from disliking smoking and considering it harmful to accepting it as a model, mainly if they've observed their grandparents' smoking. These theories thus provide a basis for examining how grandparents' past smoking behavior might directly discourage or indirectly influence their grandchildren's adult smoking behavior, a central aspect of our research question.

These three models offer a theoretical framework to comprehend better how grandchildren might develop an improved awareness of the related health risks after observing the harmful health consequences of smoking on their grandparents. This increased understanding could lead them to decide to abstain from smoking. For instance, seeing a grandparent suffer from a smoking-related health condition can enhance a grandchild's perception of the severity and susceptibility to health risks associated with smoking. This, coupled with a strong belief in the benefits of not smoking and perceived control over their actions, may dissuade them from picking up the behavior (Purohit, 2022; Weinberger et al., 2010). Overall, these theories offer a robust and comprehensive understanding of how observational learning, personal health beliefs, and social norms con-

tribute to the transmission and prevention of smoking behaviors across generations. They allow us to perceive the multifaceted ways in which familial and social contexts influence individual behaviors and health outcomes.

Combining these theoretical perspectives, our study utilizes a unique three-generational dataset from Tromsø, Northern Norway. Our empirical methodologyleverages a sophisticated system of equations within a structural equation modeling framework reminiscent of the control function approach (Wooldridge, 2015). This methodology allows us to estimate the structural parameters consistently. Norway offers a unique context for this study as it has robust investments in human capital, which can greatly influence the dynamics of health behavior transmission, such as smoking, providing a distinctive framework for investigating these patterns. To the best of our knowledge, our study is the first to thoroughly examine this transmission over three generations within the Norwegian context, thereby expanding the understanding of health capital mechanisms (Currie, 2020; Halliday et al., 2020).

Our findings reveal a distinct matrilateral bias in health behavior transmission, which may alter the conventional perspective of familial influence and individual health choices. This discovery of a matrilateral bias is an important contribution, indicating a new dimension in the intricate interplay of familial influence and individual health behaviors. These novel findings make this research a significant contribution to understanding the multigenerational transmission of health behaviors and contribute valuable insights into the nuanced interplay of family dynamics. For real-world implications, our findings not only shed light on familial influence on health behaviors but can also inform public health interventions and policy design, focusing on reducing smoking prevalence by addressing the identified matrilateral bias in health behavior transmission and its associated dynamics.

II. Data

A. *The Tromsø Study*

Tromsø is the largest city in Northern Norway and has about 77,000 inhabitants. The Tromsø Study[3] is a cohort study in which residents of the municipality of Tromsø participate. The study started in 1974 initially to support reducing Norway's high cardiovascular disease (CVD) mortality rates. It has also focused on various chronic diseases and disorders, in addition to focusing on CVD mortality and prevention (see, Jacobsen et al. (2012)). The study spans the years 1974 to 2016, and has had seven waves. The core interviews were with people aged 20 and older. The percentage of participation has varied from 64.7 to 78.5 among the waves. Most of the study population is representative of the adult population in Norway (Olsen et al., 2020).

In this study, we implemented stringent measures to establish the first family linkage of the Tromsø Study, ensuring data accuracy and reliability. We used rich data to estimate

[3]The study has approval from Regional Committees for Medical and Health Research Ethics, and the informed consent of each participant was obtained before they were accepted into the study (Olsen et al., 2020). https://uit.no/research/tromsostudy

intergenerational transmission in risky health behaviors. As presented in Figure 1, we first identified the offspring (G3) and then determined whether both parents (G2) participated in the study. Afterward, we selected our sample based on whether the offspring's parents responded to questions about their parents (G1) smoking during childhood. To ensure data accuracy and reliability, our linkage was constructed using anonymized identifiers within the Tromsø Study database and key family identification numbers obtained from the Norwegian Tax Administration. This dual approach allowed us to cross-verify the data within the dataset to rule out inconsistencies and ensuring our linkages' robustness. Despite the potential for minor inaccuracies due to factors beyond our control, we maintain confidence in the integrity of our findings. The logical and consistent patterns observed in our data, coupled with their congruence with existing literature, further attest to our linkages' validity.

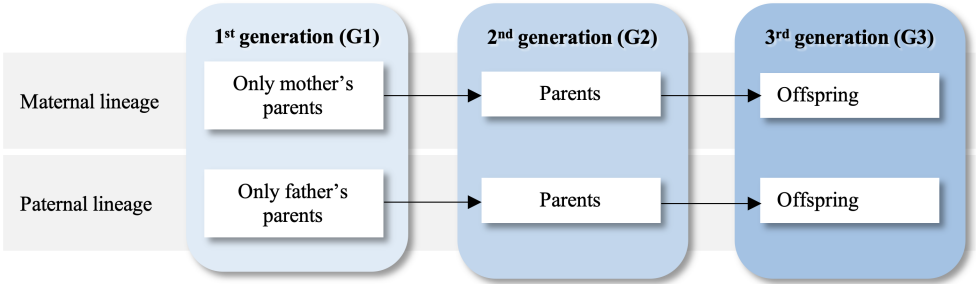

FIGURE 1. DIAGRAM AND DEFINITION OF GENERATIONS.

Note: The generation order begins with the grandparent's generation, labeled as G1. Maternal G1 indicates the mother's parents, maternal lineage, while paternal G1 indicates the father's parents, paternal lineage. The second generation represents the offspring's parents, who participated in the Tromsø Study.

Table 1 provides an overview of the descriptive statistics for the three generations in our study, categorized by maternal and paternal lineage. The smoking rates for the G3 generation appear to be slightly lower than for the G2 and G1 generations. In the maternal lineage, our sample size was 5717, and 4057 in the paternal lineage.

B. Dependent variable: smoking behavior of offspring

To better understand the multigenerational pathways of smoking behavior, we focus on intergenerational transmission mechanisms for three generations.We constructed the 'smoking' variable differently for each generation. For G3, we used responses from multiple smoking-related questions across the Tromsø Studies (see, Appendix A.1 Table A.1) to determine whether they have ever smoked occasionally or regularly. Based on these responses, we constructed a binary smoking status of the offspring into two categories: non-smokers and smokers.

Unlike previous studies on intergenerational transmission of smoking behavior for three generations, we focus on adulthood smoking behavior in the last generation rather

TABLE 1—DESCRIPTIVE STATISTICS FOR THREE GENERATIONS.

	Maternal lineage	Paternal lineage
Third generation (G3) – Offspring		
Smoking as adult	0.65 (0.48)	0.64 (0.48)
Year born	1960 (7.51)	1962 (6.94)
Female	0.52 (0.5)	0.52 (0.5)
Household economic conditions during childhood	0.79 (0.41)	0.83 (0.37)
Second generation (G2) - Parents		
Smoking during G3's childhood	0.78 (0.41)	0.77 (0.42)
Year born	1932 (9.53)	1931 (9)
Household economic conditions during childhood	0.66 (0.47)	0.58 (0.49)
First generation (G1) - Grandparents		
Smoking during G2's childhood	0.65 (0.48)	0.70 (0.46)
Number of observations (N)	*5,725*	*4,057*

Note: Table values represent means (and median for year-born), with standard deviations in parentheses. 'Maternal lineage' refers to the mother and her parents, 'paternal lineage' to the father and his parents. 'Household economic conditions during childhood' refers to the perceived financial situation during childhood, with higher values indicating better conditions.

than adolescent ages (see, e.g., Duarte et al. (2016); El-Amin et al. (2015); Escario & Wilkinson (2015); Vandewater et al. (2014)). Adult behavior, as opposed to child behavior, is a more informative indicator of how widespread smoking has been among generations (Bantle & Haisken-DeNew, 2002; Duarte et al., 2016). Additionally, while previous studies have focused on whether the adolescents' parents and grandparents smoked at least one period of their lives, our study's exposure variable unequivocally indicates that the parents and grandparents smoked while parenting during their child's growth period.

Our main goal is to investigate whether G2 and G1's smoking affects G3's smoking behavior. We derived the smoking behavior of G2 from the responses of G3 to the following question: *'Did any of the adults smoke at home while you were growing up?'* We used the response of G2 to the same question to determine whether G1 was smoking. These variables (the reference value includes non-smokers), therefore, capture the smoking environment during the upbringing of G3 and G2, respectively.

C. Control variables

In our models, we controlled for demographic and socioeconomic characteristics that can influence smoking behavior for G2 and G3. While we have not explicitly adjusted for these confounding variables for G1 due to the lack of sufficient data, we believe that the household environment captured by the smoking behavior of adults during G2's upbringing provides a proxy measure for these factors.

Various scholars have argued that different measures of socioeconomic characteristics have different health-related pathways and mechanisms (Braveman et al., 2005; Kawachi et al., 2010). To this end, we incorporate the variable 'household economic conditions during childhood', which serves as a proxy for the socioeconomic status (SES) during both G2's and G3's respective formative years.[4] The significance of this variable cannot be overstated, given the compelling evidence linking childhood SES to health behaviors, including smoking (Gilman et al., 2003; Jefferis et al., 2004; Tian et al., 2019). By controlling for childhood SES, we mitigate potential confounding of the observed relationship between parental and offspring smoking (Tian et al., 2019). Moreover, this control allows us to discern the effect of parental smoking from the broader context of intergenerational disadvantage. Gilman et al. (2003) and Tian et al. (2019) findings show that lower SES during childhood can leave a lasting impact on health behaviors, potentially leading to smoking in adulthood. In our study, we have deliberately refrained from including additional covariates like education or parental education to minimize the risk of endogeneity. Our choice to focus on household economic conditions during G3's childhood is a more direct and relevant measure of the family environment which could influence smoking behaviors (Braveman et al., 2018). We collected data on 'household economic conditions during childhood' for both G2 and G3 generations based on their responses to specific questions within the Tromsø Study that related to their perceived economic status during their upbringing.

Furthermore, we controlled for the gender of the G3 in our models, as gender can have significant effects on smoking behavior (Gugushvili et al., 2018; Hübler & Kucher, 2016; Rodríguez-Planas & Sanz-de Galdeano, 2019). While we could not adjust for the gender of G1 and G2, we believe that the household smoking environment largely captures the gender influences from these generations during their respective child-rearing periods. We used the year born to control for the exogenous changes in the dependent variable in different periods; for more details, see Figure 2. For example, in 1960, 53% of Norwegian women aged 16 to 74 years considered themselves homemakers, 95% of all children were born to married couples, and low divorce rates (Syltevik, 2017).

[4]We intentionally did not include other SES-related variables, such as G3 educational status, in our analysis to avoid potential endogeneity problems. Adult SES-related variables could be influenced by past smoking behavior, creating a circular relationship that could bias our estimates. The selected control variable provides a measure of SES during childhood, a period that is less likely to be influenced by an individual's smoking behavior, thus minimizing the risk of endogeneity.

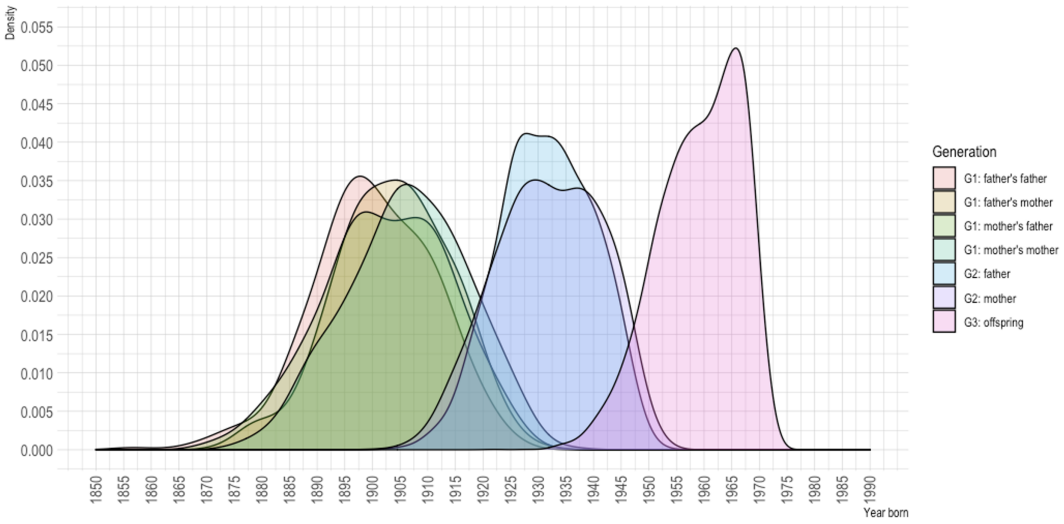

FIGURE 2. YEAR-BORN DENSITY GRAPH.

Note: The density graph illustrates the individuals' birth years in the study by generation. The first grandparent was born in 1825, the parent in 1900, and the offspring in 1922.

III. Empirical Methodology

We employ a comprehensive system of equations in a structural equation framework, reminiscent of the control function (CF) approach (Wooldridge, 2015). Our model allows the consistent estimation of the structural parameters, and we leverage individual sources of variation as instrumental variables for the smoking behaviors of the parents (S_2) and grandparents (S_1). Our goal is to estimate their combined effect on the smoking behavior of the grandchildren (S_3). In order to accurately capture the nuances of our research, we use probit regressions with average marginal effects in our empirical analysis. We apply this to both our main analysis and the evaluation of indirect effects, aiming to enhance the depth of our insights into the intergenerational transmission of smoking behavior.

We observe smoking behaviors across three generations: S_1, S_2, and S_3. Each binary variable denotes the smoking behavior of the grandparents (G1), parents (G2), and grandchildren (G3), respectively. We analyze maternal and paternal G1 smoking behaviors separately, so-called maternal lineage model and paternal lineage model in our study, allowing us to examine the distinct effects of each grandparental lineage smoking on the G3 smoking behavior. The system of equations is defined as follows.

First-stage regression.

We begin by estimating the probability of smoking behavior in G1, S_1, with the instrumental variable represented by Z_1:

$$P(S_1 = 1|Z_1) = \Phi(Z_1\alpha_1 + w) \tag{1}$$

where $\Phi(\cdot)$ is the cumulative distribution function of the standard normal distribution, and w is the error term.

To address potential endogeneity, we use the Consumer Price Index (CPI) for beverages and tobacco in the year of G2's birth in Norway,[5] as Z_1 represents our instrumental variable in the first equation. This choice is based on theoretical literature that consistently links cigarette prices to smoking behavior (French & Popovici (2011); Wooldridge, 2010, p. 309). This is also common practice in empirical research, cigarette prices or taxes have been widely used to gauge the impact of smoking on various outcomes (see, e.g., Cotti et al. (2022); Felsinger & Groman (2022); Nguyen et al. (2021)). To facilitate our analysis, we convert the annual CPI into a binary form using the third quartile as a threshold, utilizing a methodology based on the birth year of G2.[6] For robustness, a sensitivity analysis was conducted using the continuous form of the CPI. The average marginal effect of the maternal and paternal lineage models on the significance of G1 smoking remains similar, reinforcing the validity of our model.[7] Our decision to use a binary form of CPI is grounded in its potential to better capture non-linear relationships and threshold effects. A more comprehensive discussion of the instrumental variable, Z_1, can be found in Appendix A.2.

Once we have estimated this equation, we predict $\hat{S}_1$ from S_1, and then calculate the inverse Mills ratio (λ_1) (IMR) which is defined as:

$$\lambda_1 = \frac{\varphi\left(\hat{S}_1\right)}{\Phi\left(\hat{S}_1\right)} \tag{2}$$

where, $\hat{S}_1$ is the predicted probability of G1 smoking from the first-stage probit model. φ represent the probability density function of the standard normal distribution. We calculate the $IMR-1$, which serves as a key component in addressing potential issues of sample selection bias and endogeneity in our model. By incorporating the IMR into our analysis, we are able to correct for non-random sampling of smoking behavior, ensuring that our estimates accurately reflect the population dynamics of smoking behavior across generations. This adjustment is particularly important given that individuals with a family history of smoking are potentially more likely to be smokers themselves, making the sample susceptible to inherent selection bias. The IMR therefore facilitates a more robust analysis of underlying intergenerational smoking patterns by allowing us to control for unobserved factors that might otherwise bias our results (Murtazashvili & Wooldridge,

[5]The CPI for cigarettes and drinks for Norway, covers the period 1492-2017 (1913=100) and is based on a representative sample of products and services purchased by the typical Norwegian family. It is released annually by Grytten (2018). The transformation of the annual CPI into a binary indicator was achieved by considering the CPI values within the birth year timeframe of G2. Based on this, we classified values above the third quartile as 'high CPI' (represented as '1'), while all other values were classified otherwise.

[6]Given that G1's smoking variable encompasses G2's childhood —roughly 18 years—we prefer using CPI in the year of G2's birth to rule out any possible association between CPI for beverages and tobacco and G2's smoking behavior.

[7]While the use of binary variables can sometimes result in the loss of some information in the data, we have undertaken a sensitivity analysis using the continuous form of the CPI. The results are presented in Table A.2 in Appendix A.2, which provides additional assurance on the robustness of our results. The sensitivity analysis shows that our approach is not sensitive to the way the instrument is coded.

2016; Wooldridge, 2015, 2014).

Following the initial estimation of the probabilities of G1 smoking behavior, we proceed to the second stage to analyze G2 smoking behavior, taking into account the influence of G1. To mitigate potential standard error concerns in the second and third stages of our CF model, we implement a bootstrapping technique. This method involves repeatedly resampling the data and recalculating the estimates. It produces a distribution of estimates that allows for robust derivation of standard errors, as suggested by Terza (2016). We perform this procedure in both the second and third stages, with 10,000 iterations and a robust variance-covariance matrix option, which produces more precise standard error estimates. Marginal effects were then calculated based on these corrected models, increasing the reliability and precision of our results.

Second-stage regression.

In the second stage, we model the smoking behavior of G2, S_2, conditional on the smoking behavior of G1, a set of covariates X, an instrumental variable Z_2, and the $IMR-1$ (λ_1) from the first-stage:

$$P(S_2 = 1 \,|S_1, X, Z_2, \lambda_1) = \Phi(S_1\beta_1 + X\beta_2 + Z_2\beta_3 + \lambda_1\beta_4 + v) \tag{3}$$

where X presents background characteristics for G2, such as household economic conditions during childhood and birth year, and v is the error term for the second stage.

In the second stage, we use the influence of the official statement on smoking and health made by the Norwegian Director of Health,[8] Karl Evang, in the Journal of the Norwegian Medical Association (Kjønstad et al., 2000; Lund et al., 2018), as an instrumental variable for G2 smoking behavior during the upbringing of their G3 children (Z_2). The release of Evang's statement in 1964 suggests that this instrument is exogenous to individual behaviors. Furthermore, Lund et al. (2018) highlight that this report played a seminal role in increasing public awareness about the health risks associated with smoking, leading to a significant decline in smoking prevalence in the subsequent years. Additionally, the statement served as a prelude to a series of public health measures, including stricter regulations on tobacco advertising and educational campaigns aimed at discouraging smoking. Therefore, we use 1964 as a cut-off year and define the control group as G2 individuals who became parents before Norway implemented stricter smoking regulations and the treatment group as those who had their G3 children after the regulations were in place. This categorization enables us to estimate the causal effects of G2 smoking behavior on the health of their G3 offspring. Besides, we incorporate control variables into our model, X, thereby enhancing its robustness and validity. For a more in-depth discussion on the validity of this instrument, we refer readers to

[8] Karl Evang's official statement in 1964 was a turning point in Norwegian public health, echoing the findings of the U.S. Surgeon General's Report (Surgeon General's Advisory Committee on Smoking and Health, 1964) released in the same year. Both reports were instrumental in establishing the link between smoking and diseases such as lung cancer and heart disease, drawing from a broad range of biomedical and epidemiological studies. These findings significantly heightened public awareness of smoking's health risks in Norway (Lund et al., 2018)

Appendix A.3.

From Equation 3, we obtain the predicted probabilities ($\hat{S}_2$) from S_2 and calculate the $IMR-2$ (λ_2):

$$\lambda_2 = \frac{\varphi\left(\hat{S}_2\right)}{\Phi\left(\hat{S}_2\right)} \tag{4}$$

With the behavioral model for G2 in place, we then turn our attention to the third generation, incorporating the outcomes of the first two stages and additional variables to evaluate G3 smoking behavior.

Third-stage regression.

Finally, we regress the adult G3 smoking behavior on G1 and G2 smoking, a set of exogenous control variables for G3 (W), and the $IMR-1$ and -2 from the first and second stages:

$$P(S_3 = 1 \,|S_1, S_2, W, \lambda_1, \lambda_2) = \Phi(S_1\gamma_1 + S_2\gamma_2 + W\gamma_3 + \lambda_1\gamma_4 + \lambda_2\gamma_5 + u) \tag{5}$$

W stands for household economic conditions during childhood, gender, and birth year for G3. u is the error term for the third stage.

In this final stage, we regress S_3 on S_1, S_2, λ_1, and λ_2 to obtain unbiased estimates of ($\hat{\gamma}_1$) and ($\hat{\gamma}_2$) with robust standard errors and seek to elucidate the potential causal pathways between the smoking behavior of the G1 and G3. The parameter estimate ($\hat{\gamma}_1$) represents the socio-emotional influence of G1 smoking behavior on G3 smoking behavior (Zeng & Xie, 2014; Zhang et al., 2021), after controlling for G2's influence and possible selection bias. As Zeng & Xie (2014) also underlines, it encapsulates the cultural, attitudinal, and behavioral impacts of G1 smoking behavior on G3. As the genetic influences and the main socioeconomic influences are generally mediated through G2, the direct influence of G1 smoking behavior is hypothesized to be mainly socio-emotional. For example, G1's attitudes towards smoking might shape the family's overall view of smoking and thus influence G3's behavior. This method allows us to understand the unique impact of the grandparent's smoking habits, disentangling it from the parent's influence. In order to estimate the direct influence of G2 on G3 smoking behavior, we incorporate a direct link from S_2 to S_3 in our model. By doing this, we enhance the model's ability to capture the nuanced pathways of intergenerational smoking transmission.

To verify the effectiveness of our instrumental variables, we execute numerous tests and exhibit their results in Table 2. High F-test results from both stages for maternal and paternal lineage models confirm the relevance and strength of our instruments. Residuals' covariances with our instruments are almost zero, suggesting their exogeneity. Anderson-Rubin tests for endogeneity and the significance of the Wooldridge test statistic (p-value$<$0.01) indicate that using instrumental variable methodology is suitable to handle potential endogeneity. Further, we replicate the placebo test as done by Liu et al. (2022), generating random placebo variables for both lineage models, running regressions, and calculating p-values. After 500 iterations, high p-values lead us not to

reject the null hypothesis that the placebo instruments have no effect, reinforcing the validity of our original instrumental variables and decreasing the likelihood of bias due to endogeneity in the results for both lineage models.

The indirect influence of G1 smoking on G3 smoking through G2 smoking is calculated using Sobel's product of coefficients approach (Sobel, 1982). This indirect effect, within the scope of this research, explicates the extent to which G1 smoking behavior is transmitted to G3 via G2 smoking. Also, we calculate the total effect, which captures the combined influence of both the direct and indirect effects of G1 smoking on G3 smoking. To obtain robust standard errors for the indirect effect, we conducted a Monte Carlo simulation with 10,000 replications. Monte Carlo methods are particularly advantageous for complex computations such as these, as they provide accurate, empirical estimations of standard errors, thereby enhancing the reliability of our results. The indirect and total effects are estimated through the application of Sobel's methodology, requiring the multiplication of the partial regression coefficient of S_2 on S_3 (notated as γ_2) with the coefficient of S_1 (β_1).[9] Mathematically, this is represented as:

$$Indirect\ Effect = \gamma_2 * \beta_1 \tag{6}$$

$$Total\ Effect = \beta_1 + (\gamma_2 * \beta_1) \tag{7}$$

IV. Results

Our study investigates the complex, multigenerational transmission of smoking behavior. Utilizing data from the Tromsø Study, spanning from 1974 to 2016, we distinguish the influences of maternal and paternal lineages on this transmission. Panel A of Table 2 presents the direct effects of the grandparents' (G1) and parents' (G2) smoking behavior on the grandchild generation's (G3) smoking behavior. The control function (CF) approach allows us to disentangle the complex web of direct and indirect influences of maternal G1 smoking behavior. Table 2 presents both CF and naïve OLS regression results for the maternal lineage.[10]

[9] While this approach allows us to quantify indirect and total effects, the model assumes linear and additive relationships between variables. Non-linear effects or interactions between variables could provide additional insights into the complex dynamics of intergenerational smoking behavior. Since the main focus of this study is the direct effect of G1 smoking behavior on G3, we recommend it be considered in future studies.

[10] Naïve ordinary least squares (OLS) estimates were also presented to provide a comparative perspective on the results derived from the control function (CF) approach. Naïve OLS regressions, despite their simplifying assumptions, are widely recognized for their interpretability and ease of understanding, especially when comparing marginal effects. Moreover, presenting the naïve OLS estimates alongside the CF analysis highlights the discrepancy in estimates due to uncontrolled endogeneity in the former and allows for a clearer illustration of the contribution of the CF approach in addressing such concerns.

TABLE 2—INVESTIGATION OF THE INFLUENCE OF GRANDPARENTAL SMOKING ON SUBSEQUENT GENERATIONS.

Third-step: G3 Smoking	Maternal lineage		Paternal lineage	
	Marginal effects (CF)	Naïve *OLS*	*Marginal effects (CF)*	Naïve *OLS*
Variables	(1)	(2)	(3)	(4)
Panel A				
Maternal G1 Smoking	-0.053***	0.001		
	(0.020)	(0.013)		
Paternal G1 Smoking			-0.017	0.039**
			(0.033)	(0.017)
G2 Smoking	0.118***	0.126***	0.118***	0.123***
	(0.015)	(0.015)	(0.017)	(0.018)
Control variables	Yes	Yes	Yes	Yes
$IMR-1$	Yes		Yes	Yes
$IMR-2$	Yes		Yes	
Observations	5,725	5,725	4,057	4,057
R^2		0.026		0.026
Akaike Inf. Crit.	7,272.684	0.126***	5,195.554	
$Cov(\widehat{w}, Z_1)^{\dagger}$	0.011		0.005	
$Cov(\widehat{v}, Z_2)$	-0.005		-0.006	
$Cov(\widehat{w}, \widehat{v})$	0.019		0.027	
$Cov(Z_1, Z_2)$	0.064		0.054	
$Cov(\widehat{u}, \widehat{w})$	0.002		0.035	
$Cov(\widehat{u}, \widehat{v})$	0.032		0.014	
F-test of excluded instrument in first-stage	68.053***		13.130***	
F-test of excluded instrument and $IMR-1$ in second-stage	21.387***		19.484***	
Anderson-Rubin test statistic for endogeneity	6.335***		4.934***	
Wooldridge test statistic	643.920***		596.082***	
Placebo test result (p-value)	0.948		0.900	
Likelihood ratio test (p-value)	0.001		0.042	
Panel B				
Indirect effect of G1 on G3	0.007***		0.015***	
Total effect	-0.045**		-0.002	

Note: Panel A presents the results of control function (CF) approaches and naïve OLS for both maternal and paternal lineages; Panel B presents the results of indirect and total effects. $IMR-1$ and $IMR-2$ refer to the Inverse Mills Ratios from the first and second stage regressions, respectively. The CF approach uses the first and second stage $IMR-1$ and -2 in the third step (column (3)) and the first stage $IMR-1$ in the second step (column (1)). All models include control variables. These ratios are important in our CF approach to control for potential selection bias and endogeneity in our analysis. The CPI for beverages and tobacco in G2's birth year (Z_1) and Norway's first official smoking and health statement in 1964 (Z_2) are used as instrumental variables in our analysis. Covariances between residuals and two instrumental variables are close to zero, indicating exogeneity of the instruments. F-tests confirm that the instruments are not weak; the Anderson-Rubin test verifies the endogeneity of G1 and G2 smoking behavior. Wooldridge's test supports the use of instrumental variables for endogeneity. The placebo test confirms the robustness of the third stage CF against endogeneity bias. The p-value of the likelihood ratio test indicates a better fit for the third-stage CF including $IMR-1$ and -2. Robust standard errors are shown in parentheses. Akaike Inf. Crit. is Akaike Information Criterion. More details can be found in Appendix D, Table D.1 and Table D.2.

*** Significant at the 1% level.

** Significant at the 5% level.

* Significant at the 10% level.

In the maternal lineage, the control function (CF) model shows a statistically significant negative effect of G1 smoking on G3 smoking behavior, with a marginal effect of -0.053 (column (1)). This result suggests that maternal G1 smoking during the upbringing of G2 reduces the likelihood of G3 smoking in adulthood. This negative direct effect may reflect evolving social attitudes and health consciousness regarding smoking. It is also plausible that exposure to the health consequences of smoking in maternal G1 leads to an aversion to smoking in G3. For the paternal lineage, the situation differs. The CF model does not identify a statistically significant effect of G1 smoking behavior on G3 smoking behavior (column (3)). This finding suggests that the socio-emotional influence of paternal G1 on G3's propensity to smoke is not statistically detectable in our sample. In other words, it suggests that paternal G1 attitudes toward smoking and related family smoking norms may not have a noticeable direct effect on G3 smoking behavior. As a result, the paternal lineage findings differ from the maternal lineage findings, where a direct effect of maternal G1 smoking on G3 smoking was observed, suggesting a potential matrilateral bias.[11] Contrastingly, the naïve OLS analysis reports a positive and statistically significant relationship between paternal G1 smoking and G3 smoking, reinforcing the importance of correcting for potential endogeneity when investigating these intricate intergenerational relationships.

In the case of G2 smoking behavior, both lineages exhibit a strong, positive association with G3 smoking. This effect remains statistically significant across all models. The consistency of this finding emphasizes the pivotal role G2 smoking behavior during G3's childhood plays in shaping G3 smoking behavior in adulthood, regardless of lineage. These results provide evidence that the smoking behaviors of G2 significantly related to G3 smoking behavior, with a notable degree of stability. These findings suggest that the smoking behavior of G2 during the G3's childhood is a significant predictor of G3 smoking behavior in adulthood, regardless of the lineage.

Panel B of Table 2 provides additional insights into the indirect and total effects of G1 smoking behavior on their G3 smoking behavior. First, the indirect effect refers to how the smoking behavior of G1 affects G3 through the mediating behavior of G2. The results indicate a statistically significant and positive indirect effect for both maternal and paternal lineages. This suggests that if G1 smokes, it increases the likelihood of G2 also smoking, which in turn increases the likelihood that G3 smokes. This pattern holds true for both maternal and paternal lineages.However, when considering the total effect, which includes both the direct and indirect influences of G1 smoking behavior on G3 smoking behavior, a different pattern emerges. For the maternal lineage, the total effect is statistically significant and negative. This suggests that although G1 smoking behavior may increase the propensity of G2 and subsequently G3 to smoke (positive indirect effect), there is another influencing factor at play when we examine the grandparent-grandchild relationship directly (without considering G2 behavior). This influence appears to be strong enough not only to counteract the positive indirect effect, but also to

[11] In this study, 'matrilateral bias' refers to the observation that the maternal line (mother to daughter) seems to have a stronger influence on the smoking behaviors of subsequent generations compared to the paternal line. This bias towards the maternal lineage suggests a stronger intergenerational transmission of smoking behaviors from mothers to their children and grandchildren.

reverse it, resulting in an overall negative effect on the likelihood of G3 smoking. For the paternal lineage, however, the total effect of G1 smoking on G3 smoking remains statistically insignificant. This suggests that the direct influence of G1 smoking behavior on G3 smoking behavior is not strong enough to establish a significant total effect, despite the significant positive indirect effect through G2.

These findings underscore the intricacies of multigenerational smoking behavior, reflecting the nuanced dynamics between direct and indirect familial influences and their ultimate impact on smoking prevalence in subsequent generations. Detailed results are available in Appendix A.4, Table A.3 and Table A.4.

A. Robustness checks

Robustness checks are additional analyses conducted to confirm the reliability and validity of our primary findings. By examining potential variations across different social groups, considering gender-specific influences, and extending the analysis to other risky behaviors, we aim to ensure that our conclusions are not sensitive to specific model assumptions or measures. Similar to our previous analyses, this test confirms the robustness of our main findings and the significant influence of intergenerational factors on smoking behavior.

THE POTENTIAL DIFFERENTIAL COHORT EFFECTS ACROSS SOCIAL GROUPS

Differential cohort effects refer to potential variations in our main findings across different social groups. These robustness checks explore whether factors like G3's household economic conditions during childhood may influence the relationship between G2 and G3 smoking. Specifically, we look at how the interaction between G2 smoking and G3 household economic conditions during their childhood might influence G3 smoking. Our analysis maintains the significance and direction of the key variables in both maternal and paternal lineages, reaffirming the robustness of our initial findings.

As shown in Table 3, column (1), G2 smoking remains significantly positive at the 1% level, even with a stronger effect size of 0.146 compared to 0.125 in the original analysis in the maternal lineage. The adverse influence of maternal G1 smoking on G3 smoking also persists, confirming the intergenerational transmission of smoking behavior. Conversely, the effect of G3's household economic conditions and the interaction between G2 smoking and G3's household economic conditions are not significant. This could imply that the intergenerational transmission of smoking behavior in the maternal lineage may not be influenced by G3's economic conditions during childhood.

TABLE 3—INVESTIGATION OF THE POTENTIAL DIFFERENTIAL COHORT EFFECTS ACROSS SOCIAL GROUPS FOR THE INFLUENCE OF GRANDPARENTAL SMOKING ON SUBSEQUENT GENERATIONS.

Third-step: G3 Smoking	Maternal grandparents *Marginal effects (CF)* (1)	Paternal grandparents *Marginal effects (CF)* (2)
Maternal G1 Smoking	-0.054***	
	(0.020)	
Paternal G1 Smoking		-0.017
		(0.033)
G2 Smoking	0.146***	0.155***
	(0.039)	(0.053)
G3 household economic conditions during their childhood (G3_econ)	-0.014	0.004
	(0.036)	(0.050)
G2 Smoking X G3_econ	-0.025	-0.035
	(0.040)	(0.054)
Other control variables for G3	Yes	Yes
$IMR-1$	Yes	Yes
$IMR-2$	Yes	Yes
Observations	5,725	4,057
Akaike Inf. Crit.	7,272.325	5,197.138

Note: The robustness checks for the potential differential cohort effects across social group analysis were conducted in the same manner as for the overall sample, using control function (CF) methods and including the first- and second-stage residuals, as well as other control variables for G3. $MR-1$ refers to the first-stage inverse Mills ratio, used to account for the sample selection bias in the relationship between grandparent (G1) and parent (G2) smoking. $IMR-2$ is the second-stage inverse Mills ratio, which controls for the sample selection bias in the relationship between G2 and child (G3) smoking. First- and second-stage *IMRs* are used in the control function (CF) method in the third-step (columns (1) and (2)). Robust standard errors are shown in parentheses. *** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

In the paternal lineage (Table 3, column (2)), G2 smoking continues to have a significant influence on G3 smoking, again with a stronger effect size (0.155). However, similar to the main analysis, paternal G1 smoking shows no significant impact on G3 smoking. Moreover, G3's household economic conditions and the interaction of these conditions with G2 smoking are not significant, which aligns with the maternal lineage results.

Overall, our main findings about the intergenerational transmission of smoking behaviors remain robust after accounting for potential differential cohort effects across social groups. These findings underscore the generalizability of our conclusions and the importance of a nuanced understanding of lineage-specific influences and socio-economic factors in such intergenerational patterns.

THE GENDER-SPECIFIC GRANDPARENTAL INFLUENCES (G1)

Gender-specific grandparental influences examine whether the impact of G1 and G2 smoking on G3 smoking differs between males and females. This analysis helps us understand whether the gender of the third generation influences the transmission of smoking behaviors. Several studies have investigated gender-based differences in grandparental investment in developed countries (Coall & Hertwig, 2010; Tu et al., 2021; Wang & Chen, 2019). While the outcomes of these studies differ, following Tanskanen et al. (2011), we explore whether grandparents' investment varies between female and male grandchildren in our sample. Our intention is to delve deeper into the matrilateral effect of grandparental investment and ascertain whether the evolutionary significance of intergenerational transmission that we observe in our findings remains valid.

In both lineages, G2 smoking maintains its positive and significant influence on G3 smoking at the 1% level, similar to our original findings (Table 4, columns (1) and (2)). This implies that the effect of G2 smoking on G3 smoking is robust, regardless of the gender of G3. In the maternal lineage, the influence of maternal G1 smoking on G3 smoking persists but has reduced in magnitude compared to the main analysis (-0.048 vs. -0.053) and is now significant only at the 10% level. The interaction term between maternal G1 smoking and the female gender of G3 is not significant, suggesting that the intergenerational transmission of smoking behavior from the maternal G1 does not vary by the gender of the G3.

TABLE 4—ASSESSING THE GENDER-SPECIFIC EFFECTS OF G1 AND G2 SMOKING ON G3 SMOKING.

Third-step: G3 Smoking	Maternal grandparents *Marginal effects (CF)* (1)	Paternal grandparents (2)
Maternal G1 Smoking	-0.048*	
	(0.025)	
Paternal G1 Smoking		-0.002
		(0.038)
G2 Smoking	0.124***	0.123***
	(0.016)	(0.019)
Female	-0.053**	-0.039
	(0.021)	(0.027)
Maternal G1 Smoking X Female	-0.012	
	(0.027)	
Paternal G1 Smoking X Female		-0.029
		(0.033)
Other control variables for G3	Yes	Yes
$IMR-1$	Yes	Yes
$IMR-2$	Yes	Yes
Observations	5,725	4,057
Akaike Inf. Crit.	7,272.512	5,196.765

Note: The robustness checks for the potential differential cohort effects across social group analysis were conducted in the same manner as for the overall sample, using control function (CF) methods and including the first- and second-stage residuals, as well as other control variables for G3. $MR-1$ refers to the first-stage inverse Mills ratio, used to account for the sample selection bias in the relationship between grandparent (G1) and parent (G2) smoking. $IMR-2$ is the second-stage inverse Mills ratio, which controls for the sample selection bias in the relationship between G2 and child (G3) smoking. First- and second-stage *IMRs* are used in the control function (CF) method in the third-step (columns (1) and (2)). Robust standard errors are shown in parentheses. *** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

In the paternal lineage, the paternal G1 smoking effect is still insignificant, consistent with our original results, indicating that paternal G1 smoking behavior may not significantly influence the G3 smoking propensity. Similar to the maternal lineage, the interaction term between paternal G1 smoking and the female gender of G3 is not significant, suggesting no differential effect of paternal G1 smoking on granddaughters compared to grandsons.

In terms of control variables, the 'Female' variable is significant at the 5% level in the maternal lineage model, implying that female G3 individuals are less likely to smoke, independent of their G1 smoking behavior. However, this gender effect is not significant in the paternal lineage model. In summary, our original findings about the transmission of smoking behaviors across generations hold robust even when we consider gender-specific effects. The lack of significance for the interaction terms with gender suggests that the intergenerational effects of smoking do not differ between males and females in the third generation.

THE PRESENCE AND SURVIVAL OF GRANDPARENTS (G1)

In previous studies of intergenerational relationships, geographic distance has been considered the most apparent factor influencing the opportunity for frequency of contact among generations (Danielsbacka et al., 2015; Lundborg & Majlesi, 2018; Tanskanen et al., 2011). Many correlational studies include comprehensive ethnographic reports that not only emphasize potential behaviors that promote positive impact, but also explore correlations between the presence of kin and survival, growth, and development (Cisco, 2017; Coall & Hertwig, 2010; Koster, 2018). We assume that G1's proximity to G3 gives G1 greater investment opportunities in terms of time, care, interaction, etc. Such interactions could create stronger socio-emotional ties and potentially influence G3 smoking behavior. To check the robustness of our findings regarding distance, we classify the birthplace of our sample G2 as a Tromsø and outside, also excluded observations when G1 died in the year of birth of G3. We consider G2's place of birth as a proxy for G1's potential distance from G3, and we restrict the subsample only to include people who were born in Tromsø.

Table 5, presents a robustness check on the influence of G1 smoking on G3 smoking behavior, focusing on both maternal and paternal grandparents separately. This findings reinforce the robustness of our primary empirical results. Specifically, the marginal effect of maternal G1 smoking on G3 smoking behavior is -0.051 (column (1)), which closely aligns with the main findings, where the coefficient was -0.053 (Table 2, column (1)). This minor difference in magnitude and level of significance confirms the stability and robustness of our main findings for the maternal lineage. As for the paternal lineage, there is a non-significant direct effect of paternal G1 smoking on G3 smoking behavior, with a coefficient of -0.018 (column (2)). This result is consistent with the main findings (Table 2, column (3)) where a similarly non-significant direct influence is reported. This consistency further underscores the reliability of our main findings, affirming the lack of a direct, discernible impact of paternal G1 smoking on G3 smoking behavior.

TABLE 5—ASSESSING THE GENDER-SPECIFIC EFFECTS OF G1 AND G2 SMOKING ON G3 SMOKING.

Third-step: G3 Smoking	Maternal grandparents *Marginal effects (CF)* (1)	Paternal grandparents (2)
Maternal G1 Smoking	-0.051**	
	(0.023)	
Paternal G1 Smoking		-0.018
		(0.033)
G2 Smoking	0.123***	0.123***
	(0.018)	(0.019)
Control variables for G3	Yes	Yes
$IMR-1$	Yes	Yes
$IMR-2$	Yes	Yes
Observations	4,605	4,057
Akaike Inf. Crit.	5,832.811	5,195.554

Note: The robustness checks for the potential differential cohort effects across social group analysis were conducted in the same manner as for the overall sample, using control function (CF) methods and including the first- and second-stage residuals, as well as other control variables for G3. $MR-1$ refers to the first-stage inverse Mills ratio, used to account for the sample selection bias in the relationship between grandparent (G1) and parent (G2) smoking. $IMR-2$ is the second-stage inverse Mills ratio, which controls for the sample selection bias in the relationship between G2 and child (G3) smoking. First- and second-stage *IMRs* are used in the control function (CF) method in the third-step (columns (1) and (2)). Robust standard errors are shown in parentheses. *** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

On the other hand, there can be various reasons behind not finding difference between main results. For instance, even though G1 and G3 may not live in close proximity, they could still have had substantial interactions during holidays or regular visits, maintaining strong socio-emotional ties. It is also possible that non-physical forms of interaction, such as through shared family narratives, attitudes, and values, played a more crucial role in influencing G3 smoking behavior. Therefore, we acknowledge that measuring socio-emotional ties directly would be challenging, and using proxies might not fully capture their complexity. However, using G2's birthplace as a proxy for G1's potential distance from G3 allows us to examine the role of geographic proximity within the limits of our data. Yet, the results of the robustness check closely align with our primary findings, confirming the stability and validity of the results.

Overall, the result from our robustness checks not only validates the primary findings of our study but also enhance our understanding of the intergenerational transmission of risky health behaviors. We can confidently conclude that our main findings are robust across multiple dimensions - across gender lines, across different socioeconomic conditions, and even when considering presence and survival of G1.

In addition to the aforementioned robustness checks, we conduct an additional analysis to further examine the influence of G1's smoking behavior on other risky health behaviors, focusing specifically on G3's alcohol consumption. This additional robustness check aims to examine whether G3 smoking behavior has a significant impact on G3's alcohol abuse behavior. By extending our analysis to examine the impact of ancestral smoking habits on other risky health behaviors in future generations, we aim to provide a comprehensive understanding of the broader implications. The detailed results of this supplemental analysis are presented in Appendix A.5 for a more comprehensive review.

V. Discussion

Our research highlights the significance of intergenerational transmission of risky health behaviors, in this case, smoking, emphasizing the role of maternal grandparents in mitigating this risk in the context of Northern Norway, Tromsø. The underlined transmission mechanisms between grandparents and their grandchildren's smoking behavior were explored in a comprehensive three-generational sample, reinforcing the existence of matrilateral bias in risky health behaviors. The most notable finding is that maternal grandparents have a significant negative direct effect on their offspring, even after controlling for parental smoking, which translates to a reduced risk of their grandchildren taking up smoking. We do not, however, find the same statistical significance in the paternal lineage. This finding is important evidence for the existence of matrilateral bias in risky health behaviors.

To place this within the wider scholarly context, earlier studies show evidence that tobacco use appears to be transmitted from grandparents to grandchildren through parental smoking regardless of maternal and paternal lineage (see, e.g., Duarte et al. (2016); El-Amin et al. (2015); Vandewater et al. (2014)). However, these studies have no focus on the direct effect of grandparents' smoking on their offspring. El-Amin et al. (2015) and

Vandewater et al. (2014) found that while grandparents' smoking does indirectly influence grandchildren's tobacco use, this influence often lost its significance when considering the grandparents' smoking direct effect on their grandchildren, unlike our findings. Meanwhile, Duarte et al. (2016) found a significant association between students' smoking and their mothers', fathers', and grandparents' smoking, but the study suggests a gendered impact, unlike our results, which indicate a matrilateral bias regardless of the grandchildren's gender. Our findings thus distinguish themselves by emphasizing the specific role of maternal grandparents. This divergence is novel and offers a different perspective on the dynamics of intergenerational health behavior transmission, which may be valuable for interventions aimed at reducing risky health behaviors like smoking.

This research is pivotal for several reasons. It pushes the boundaries of our understanding of the intergenerational transmission of risky health behaviors, a field that has been somewhat overshadowed in the broader realm of behavioral studies. By specifically focusing on smoking, a widespread and detrimental health behavior, the research underscores the gravity of such transmissions and their repercussions on public health. Most significantly, our study illuminates the role of grandparents, which has been underexplored in previous research. There is a tendency to focus on parents when studying behavioral influence and transmission within a family. Our research challenges this perspective by highlighting the substantial impact of grandparents on their grandchildren's behavior, particularly the maternal grandparents. This understanding reshapes the narrative of familial influence on health behaviors and suggests a broader, more inclusive view of the family's role in shaping an individual's health choices. The aspect of the study that distinguishes it from previous research and makes a novel contribution to the literature is the examination of matrilateral bias in risky health behaviors.

While existing research recognizes that behaviors can pass down through generations, the assertion of a bias towards the maternal lineage in this transmission is a relatively new and intriguing finding. This understanding of matrilateral bias can have profound implications for understanding how health behaviors are inherited and how interventions could be designed for maximum impact. Additionally, our research leverages a unique dataset from Northern Norway, Tromsø, which encompasses a comprehensive three-generational sample. This allows for a robust analysis of transmission mechanisms across generations in a specific geographical and cultural context, thereby enriching the literature in this field. Furthermore, the methodological rigor of this study, employing a control function approach, strengthens the validity of the findings and provides a replicable framework for future research.

The findings of our study create an interesting conversation between the social learning theory and the health belief model, necessitating a more comprehensive approach to understanding the multigenerational transmission of health behaviors. This nuanced understanding of grandparental investment and the strong evidence of matrilateral bias delineates a paradigm shift in the multigenerational transmission of health behaviors. The social learning theory (Bandura, 1971) traditionally emphasized parental influence, focusing on the immediate nucleus of the family. Children, according to this theory, tend to emulate the behaviors and attitudes they observe in their parents. However, our re-

search offers a more layered understanding. It posits that the influence is not just vertical (parent to child) but can be traced back horizontally (grandparent to grandchild) and is especially pronounced in the maternal lineage. This shift in the locus of influence underscores the importance of including grandparents in discussions about the family's role in shaping a child's health behaviors (Sadruddin et al., 2019). This extension of the theory prompts a reconsideration of the influential figures in a child's life, suggesting a need for broader family-based interventions.

Conversely, the health belief model (Rapoff et al., 2023; Rosenstock et al., 1988) emphasizes individual beliefs and perceptions about health risks and benefits. Applied to our context, grandchildren might have observed the ill effects of smoking on their grandparents, cultivating a belief about the serious health risks associated with smoking. Concurrently, they recognize the benefits of a smoke-free lifestyle, leading to a personal decision to refrain from smoking. This highlights the role of individual agency and informed decision-making in shaping health behaviors (Bandura, 2001). The interplay between the social learning theory and the health belief model in our study forms a compelling narrative. It underlines the importance of an intergenerational and individual perspective in understanding health behaviors. While the social learning theory suggests the role of observed behaviors in the family, especially those of the maternal grandparents, the health belief model emphasizes the individual perception of risk and benefits formed through these observations. The confluence of these two theories creates a more holistic understanding of the multigenerational transmission of health behaviors. It proposes that while grandparents, particularly maternal ones, have a strong influence on their grandchildren's health behaviors, the grandchildren also possess individual agency guided by their personal beliefs and experiences. The coexistence and interaction of these two theories can help develop more nuanced and effective interventions, considering both the family environment and individual beliefs.

Moreover, the significance of our research is amplified by the increasing life expectancy in industrialized societies like Norway. This extended lifespan affords grandparents a greater opportunity to impact their grandchildren's lives (Coall & Hertwig, 2010; Roser et al., 2013). Their role in disseminating knowledge about the detriments of risky health behaviors and nurturing healthier habits underscores the cultural inheritance that transcends mere genetic transmission. It presents the opportunity to harness this grandparental investment as a resource for health interventions and policymaking. Also, Norway presents an apt context for our study, owing to its declining cigarette sales compared to other developed countries (Forey et al., 2016; World Bank, 2021). The extrapolation of our findings could indicate a more severe scenario for these other countries. By establishing a causal relationship between grandparents' smoking and offspring's behavior in adulthood through the control function approach, our study contributes to more accurate policy implications.

A. Policy Implications

Our findings have significant policy implications, particularly in health promotion and tobacco control. Considering the profound impact of maternal grandparents on their

grandchildren's smoking behavior, interventions designed to reduce smoking could potentially benefit from targeting this influential group. Current strategies are often parent-focused, particularly directed toward mothers during the prenatal and postnatal periods (Chamberlain et al., 2017). However, our research suggests that including grandparents in such interventions might enhance their effectiveness. Health professionals could conduct educational sessions for grandparents about the detrimental effects of smoking and the role they can play in preventing their grandchildren from adopting this harmful habit. These sessions could be included in existing smoking cessation programs or community health initiatives. Also, social campaigns can be designed to enhance awareness about the role of grandparents in shaping health behaviors, stressing their influence on grandchildren. Such interventions could encourage grandparents to model healthy behaviors and actively participate in discouraging risky health behaviors like smoking. This grandparent-focused approach could complement existing parent-focused strategies, providing a more comprehensive and effective approach to reducing smoking in the younger generations.

B. Limitations and Future Directions

Although our research presents novel insights into the intergenerational transmission of smoking behaviors, it is not without its limitations. As the Tromsø Study is representative of Norway as a whole, our findings are inherently specific to this particular context. However, the geographical specificity of our sample - Northern Norway, Tromsø - might limit the generalizability of our findings. Societal norms and cultural practices regarding smoking and familial relationships can vary greatly across different regions and societies. Therefore, caution must be taken when extrapolating our results to other geographical or cultural contexts. Also, our study primarily relies on self-reported data for smoking behavior, subject to recall and social desirability biases. Individuals may underreport or misreport their smoking habits, which could potentially impact the accuracy of our findings. While the study provides data on the smoking behavior of parents who cohabited with their offspring during childhood, our data do not include information on whether a grandparent resided in the same household during the offspring's childhood, a factor that could potentially influence smoking behaviors (Duarte et al., 2016). Furthermore, while our study constructs a family link based on registered information, this may not entirely exclude the possibility of non-genetic fathers being identified as the paternal figure, although we anticipate that this limitation is unlikely to significantly bias our results given our emphasis on family environment over genetic ties.

Future research can expand on our findings by incorporating a more diverse geographic and cultural sample, which would enhance the generalizability of the results. Additionally, studies could attempt to gather more nuanced data on family dynamics, such as the presence of grandparents in the household and the specific roles of each parent in the family, to provide a deeper understanding of intergenerational transmission mechanisms. Furthermore, future research could also investigate other potential confounding factors, such as socioeconomic status, education level, and exposure to other forms of substance abuse, which were not fully explored in our study. It would be interesting

to examine whether these factors could moderate or mediate the relationship between grandparental and grandchild smoking behaviors. Lastly, as our study relied primarily on self-reported data, future research could benefit from incorporating more objective measures of smoking behavior, such as cotinine levels, from reducing potential biases associated with self-reporting.

VI. Conclusions

In conclusion, our study brings forth a novel understanding of the intergenerational transmission of smoking behaviors, underlining the critical role of maternal grandparents in mitigating this risk. Our research invites a broader view of familial influence on health behaviors, extending beyond parents to include grandparents, particularly from the maternal lineage. Furthermore, it calls for reconsidering influential figures in a child's life, underscoring the need for broader family-based interventions.

The strong evidence of matrilateral bias in our study is a novel addition to the academic discourse on health behavior transmission, indicating the profound implications for how health behaviors are inherited and how interventions could be designed for maximum impact. In light of increasing life expectancy in industrialized societies like Norway, our research underlines the importance of harnessing grandparental investment as a resource for health interventions and policymaking. Our study provides a crucial stepping stone toward understanding the dynamics of intergenerational health behavior transmission and designing effective strategies to combat risky health behaviors like smoking. By drawing attention to the significant influence of maternal grandparents on their grandchildren's smoking behaviors, we hope to inspire further research and innovative intervention strategies in health promotion and tobacco control.

We conclude that families, as children's immediate environments, have an undeniable influence on the initiation of tobacco use. Individuals' time and risk preferences impact their lives, but they also affect their children's and grandchildren's preferences, as demonstrated by the strong correlation between grandparents' and grandchildren's smoking behavior. Raising parents', to a lesser extent, grandparents', awareness of their powerful influence on their offspring's behavior will result in improved health outcomes and increased economic welfare. As a result, direct investments can help reduce the unfortunate costs of risky health behaviors and positively impact the health capital of future generations.

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APPENDIX

APPENDIX

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Role of Grandparents in Risky Health Behavior Transmission:

A study on Smoking Behavior in Norway

by

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A.1. Defining smoking and non-smoking offspring, parents, and grandparents

We assessed offspring's smoking status as smoker and nonsmoker with these questions (Table A.1): "Do you smoke cigarettes daily?" with the options "No" and "Yes";"Do you smoke daily at present?" with the options "No" and "Yes"; "Do you smoke?" with the options "Yes, daily," "Yes, sometimes," and "No, never"; "Do you smoke sometimes, but not daily?" with the options "No" and "Yes"; "Do you smoke, or have you smoked sometimes, but not daily?" with the options " Never," "Yes, now," and "Yes, previously"; and "Do you/did you smoke daily?" with the option "Yes, now," "Yes, previously," and "Never." Additionally, we assessed at least a parent who smokes during raising their child with the following question: "Did any of the adults smoke at home while you were growing up?" with the options "No" and "Yes." For parents smoking, we used offspring's answer, and for grandparents smoking, we used offspring's parents' answer to this question.

TABLE A.1—SURVEY QUESTIONS ABOUT TOBACCO SMOKING INCLUDED IN OUR STUDY.

Survey question	Answer	Tromsø1 % / (N)	Tromsø2 % / (N)	Tromsø3 % / (N)	Tromsø4 % / (N)	Tromsø5 % / (N)	Tromsø6 % / (N)	Tromsø7 % / (N)
Do you smoke cigarettes daily?	No	16.09 (N=716)	2.03 (N=164)	1.3 (N=128)	63.9 (N=17161)	32.8 (N=1021)	N/A	N/A
	Yes	83.91 (N=3733)	97.97 (N=7923)	98.7 (N=9710)	36.1 (N=9694)	67.2 (N=2092)	N/A	N/A
Do you smoke daily at present?	No	39.89 (N=2619)	51.03 (N=8430)	54.64 (N=11852)	N/A	N/A	N/A	N/A
	Yes	60.11 (N=3946)	48.97 (N=8091)	45.36 (N=9839)	N/A	N/A	N/A	N/A
Do you/did you smoke daily?	Yes, now	N/A	N/A	N/A	N/A	28.06 (N=2245)	20.41 (N=2609)	13.9 (N=2903)
	Yes, previously	N/A	N/A	N/A	N/A	37.6 (N=3008)	42.3 (N=5407)	44.29 (N=9248)
	Never	N/A	N/A	N/A	N/A	34.35 (N=2748)	37.29 (N=4767)	41.81 (N=8731)
Do you smoke?	Yes, daily	N/A	N/A	N/A	N/A	25.99 (N=1849)	N/A	N/A
	Yes, sometimes	N/A	N/A	N/A	N/A	7.29 (N=519)	N/A	N/A
	No, never	N/A	N/A	N/A	N/A	66.72 (N=4747)	N/A	N/A
Do you smoke sometimes, but not daily?	No	N/A	N/A	N/A	N/A	N/A	87.64 (N=9982)	N/A
	Yes	N/A	N/A	N/A	N/A	N/A	12.36 (N=1408)	N/A
Do you smoke, or have you smoked sometimes, but not daily?	Yes, now	N/A	N/A	N/A	N/A	N/A	N/A	7.28 (N=1447)
	Yes, previously	N/A	N/A	N/A	N/A	N/A	N/A	18.97 (N=3772)
	Never	N/A	N/A	N/A	N/A	N/A	N/A	73.75 (N=14665)
Did any of the adults smoke at home while you were growing up?	No	N/A	N/A	N/A	27.59 (N=7413)	30.98 (N=2472)	N/A	N/A
	Yes	N/A	N/A	N/A	72.41 (N=19451)	69.02 (N=5508)	N/A	N/A

Note: These figures represent participation before family linkage construction in the relevant Tromsø Studies.

A.2. *Further Discussion on the Instrumental Variable* Z_1

The instrumental variable (IV) Z_1 represents the Consumer Price Index (CPI) for beverages and tobacco in the year of G2's birth in Norway. This IV is a crucial component of our first-stage regression model, where we estimate the probability of G1 smoking behavior.

Instrumental variables are employed to mitigate the effect of endogeneity in regression analysis, a common issue that arises when an explanatory variable is correlated with the error term (Wooldridge, 2015). This correlation can lead to biased and inconsistent estimates, thus necessitating the use of an instrumental variable. An ideal IV must satisfy two essential conditions: relevance and exogeneity. The relevance condition requires the instrumental variable to be correlated with the endogenous explanatory variable, and the exogeneity condition mandates that the instrumental variable is uncorrelated with the error term in the regression equation.

The selection of the CPI for beverages and tobacco as our instrumental variable, Z_1, is informed by the economic theory of demand. The theory suggests that the price of a product, such as cigarettes, is expected to influence consumer demand for that product (Cotti et al., 2022), hence satisfying the relevance condition. As for the exogeneity condition, the CPI for beverages and tobacco is determined by broad market factors and governmental policies, which are generally independent of an individual's behaviors. Therefore, it should not be correlated with the error term. We opted to represent the CPI in a binary form to better capture non-linear relationships and threshold effects that might be overlooked if a continuous form were used. This decision was substantiated by a sensitivity analysis conducted using the continuous form of the CPI, the results of which are detailed in Table B.1. The robustness of the binary representation of the CPI is evidenced by the consistent average marginal effect of the maternal and paternal lineage models on the significance of G1 smoking, even when using the continuous form (French & Popovici, 2011). This consistency confirms the validity of the binary form of CPI representation.

TABLE A.2—DETAILED RESULTS FROM THE INVESTIGATION OF THE INFLUENCE OF GRANDPARENTAL SMOKING ON SUBSEQUENT GENERATIONS: A MATERNAL PERSPECTIVE.

	First-step: G1 Smoking		Second-step: G2 Smoking		Third-step: G3 Smoking	
	Maternal lineage	*Paternal lineage*	*Maternal lineage*	*Paternal lineage*	*Maternal lineage*	*Paternal lineage*
	Marginal effects (CF)					
Variables	(1)	(2)	(3)	(4)	(5)	(6)
Maternal G1 Smoking			0.074***	0.109***	-0.055***	-0.019
			(0.012)	(0.015)	(0.020)	(0.033)
G2 Smoking					0.125***	0.122***
					(0.016)	(0.019)
G2 household economic conditions during their childhood			-0.003	-0.035***		
			(0.012)	(0.013)		
G2 Year born			0.001	0.000		
			(0.001)	(0.001)		
G3 household economic conditions during their childhood					-0.034**	-0.024
					(0.016)	(0.020)
G3 Year born					-0.002**	-0.005***
					(0.001)	(0.002)
Female					-0.060***	-0.059***
					(0.013)	(0.015)
$IMR-1$			0.110***	0.298***	-0.106***	-0.116
			(0.025)	(0.082)	(0.033)	(0.112)
$IMR-2$					0.145***	0.105**
					(0.040)	(0.052)
$IV-2$: Tobacco legislation in Norway: 1964			-0.096***	-0.072***		
			(0.016)	(0.017)		
$IV-1$: CPI at G2's birth year (continuous variable)	0.001***	0.000***				
	(0.000)	(0.000)				
Observations	5,725	4,057	5,725	4,057	5,725	4,057
Akaike Inf. Crit.	7,358.360	4,978.018	5,909.868	4,341.184	7,269.020	5,197.676

Note: CPI for beverages and tobacco at G2's birth year (Z_1) used as a continues variable. Robust standard errors are shown in parentheses.
*** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

Moreover, the results from the F-test and the Anderson-Rubin test for endogeneity further endorse the relevance and exogeneity of our instrumental variable (Ren et al., 2020). The F-test suggests the joint significance of the instruments in the regression, while the Anderson-Rubin test verifies the presence of endogeneity. Both tests collectively validate our choice of the CPI as an instrumental variable. In sum, using the CPI for beverages and tobacco as an instrumental variable offers a robust strategy to account for endogeneity and facilitates a reliable estimation of the structural parameters in our model . This careful application of the instrumental variable allows us to capture a more accurate representation of the relationship between G1 smoking behavior and its influence on subsequent generations.

A.3. Further Discussion on the Instrumental Variable Z_2

The instrumental variable (IV) Z_2 represents the influence of the official statement on smoking and health made by the Norwegian Director of Health, Karl Evang, in the Journal of the Norwegian Medical Association. We utilize this instrumental variable in the second stage of our regression model to estimate the smoking behavior of G2, conditional on the smoking behavior of G1, a set of covariates X, and the inverse Mills ratio (λ_1) from the first stage.

As discussed in Appendix A.2, instrumental variables are tools to correct endogeneity issues in regression analysis. In this context, Z_2 satisfies the essential criteria for an effective IV, i.e., relevance and exogeneity. The relevance condition requires the IV to be correlated with the endogenous explanatory variable. In this case, the 1964 official statement is plausibly correlated with the smoking behavior of G2. This document has historical significance as it marked a turning point in public awareness about the health risks associated with smoking in Norway. Karl Evang's statement, backed by the authority of the Director of Health, sparked a significant shift in public opinion, leading to a decline in smoking prevalence in the subsequent years (Kjønstad et al., 2000; Lund et al., 2018). It also served as a precursor to stricter regulations on tobacco advertising and educational campaigns aimed at discouraging smoking. As such, the correlation between this landmark event and the smoking behavior of G2 individuals during the upbringing of their G3 children is plausible, hence satisfying the relevance condition. Exogeneity, the second condition, requires the IV to be uncorrelated with the error term in the regression equation. In our case, the release of Evang's statement can be considered an exogenous event since it was a top-down decision made by health authorities independent of individual behaviors. Its unexpected release in 1964 suggests that this instrument is exogenous to individual behaviors. Furthermore, the measures and regulations resulting from the statement had broad societal impacts, not tied to individual characteristics or decisions. Thus, we argue that Z_2 satisfies the exogeneity condition.

In the regression model, we use 1964 as a cut-off year to define control and treatment groups for G2 individuals. This choice allows us to estimate the causal effects of G2 smoking behavior on their G3 offspring and to isolate the impact of evolving social norms and regulatory measures. As with Z_1, the relevance and exogeneity of Z_2 as an IV are supported by the F-test and the Anderson-Rubin test for endogeneity. The re-

sults indicate the joint significance of the instruments in the regression and the presence of endogeneity (Wooldridge, 2015). In conclusion, the use of the official statement as an instrumental variable in the analysis provides a robust strategy to address endogeneity and enhances the validity of our estimates on the intergenerational transmission of smoking behavior.

Historical reference: Historisk oversikt over tobakk i Norge 1619-2018.[12]

A.4. Detailed Results from Maternal and Paternal Perspectives with a Focus on Control Variables

Table A.3 and Table A.4 elucidate the impact of control variables in the study of the intergenerational transmission of smoking behaviors from a maternal and paternal perspective, respectively. These tables extend our principal findings by encompassing the impact of control variables: household economic conditions during childhood, female, and year born.

MATERNAL PERSPECTIVE, TABLE A.3

The results indicate a significant relationship between G2 smoking and maternal G1 smoking (0.075 and 0.077 in columns (3) and (4), respectively). However, the effect on G3 smoking is not consistent with an unexpected negative effect observed in column (5) (-0.053) and a negligible impact seen in column 6 (0.001). Our analysis reveals that the household economic conditions of G2 during their childhood do not significantly influence the propensity of G2 to smoke (columns (3) and (4)). Similarly, these conditions do not significantly impact G3 smoking behaviors (columns (5) and (6)). The G2 year born variable exhibits a significant positive effect on G2 smoking (0.002 in column (3)), suggesting a higher propensity for smoking among those born later. However, this effect is not observed in the naïve OLS model (column (4)). In contrast, the G3 year born variable shows a significant negative effect on G3 smoking, indicating a lower likelihood of smoking among those born later. The female variable shows a significant negative effect on G3 smoking (-0.059 and -0.062 in columns (5) and (6), respectively). This implies that females in G3 are less likely to smoke than their male counterparts.

[12] https://www.fhi.no/nettpub/tobakkinorge/tobakk-i-historien/historisk-oversikt-over-tobakk-i-norge-1619-2018/

TABLE A.3—DETAILED RESULTS FROM THE INVESTIGATION OF THE INFLUENCE OF GRANDPARENTAL SMOKING ON SUBSEQUENT GENERATIONS: A MATERNAL PERSPECTIVE.

	First-step: Maternal G1 Smoking		Second-step: G2 Smoking		Third-step: G3 Smoking	
	Marginal effects (CF)	Naïve *OLS*	*Marginal effects (CF)*	Naïve *OLS*	*Marginal effects (CF)*	Naïve *OLS*
Variables	(1)	(2)	(3)	(4)	(5)	(6)
Maternal G1 Smoking			0.072***	0.077***	-0.053***	0.001
			(0.011)	(0.012)	(0.020)	(0.013)
G2 Smoking					0.118***	0.126***
					(0.015)	(0.015)
G2 household economic conditions during their childhood			-0.003	-0.004		
			(0.012)	(0.011)		
G2 Year born			0.002***	-0.0001		
			(0.001)	(0.001)		
G3 household economic conditions during their childhood					-0.033**	-0.032**
					(0.016)	(0.015)
G3 Year born					-0.004***	-0.006***
					(0.001)	(0.001)
Female					-0.059***	-0.062***
					(0.013)	(0.013)
IMR-1			0.072***		-0.062**	
			(0.025)		(0.029)	
IMR-2					0.134***	
					(0.038)	
IV-2: Tobacco legislation in Norway: 1964			-0.091***			
			(0.015)			
IV-1: CPI at G2's birth year	0.136***	0.136***				
	(0.015)	(0.017)				
Observations	5,725	5,725	5,725	5,725	5,725	5,725
R^2		0.000		0.008		0.026
Akaike Inf. Crit.	7,379.157		5,920.359		7,270.700	
$Cov(\hat{w}, Z_1)^{\dagger}$					0.011	
$Cov(\hat{v}, Z_2)$					-0.005	
$Cov(\hat{w}, \hat{v})$					0.019	
$Cov(Z_1, Z_2)$					0.064	
$Cov(\hat{u}, \hat{w})$					0.002	
$Cov(\hat{u}, \hat{v})$					0.032	
F-test of excluded instrument in first-stage					68.053***	
F-test of excluded instrument and IMR-1 in second-stage					21.387***	
Anderson-Rubin test statistic for endogeneity					6.335***	
Wooldridge test statistic					643.920***	
Placebo test result (p-value)					0.948	
Likelihood ratio test (p-value)					0.001	
Indirect effect of G1 on G3					0.007***	
Total effect					-0.045**	

Note: We calculate the covariance between residuals (third-stage residual – $\hat{u}$, second-stage residual – $\hat{v}$, first-stage residual – $\hat{w}$) and two instrumental variables (CPI for beverages and tobacco at G2's birth year (Z_1) and the first official statement on smoking and health in Norway in 1964 (Z_2)). All covariances are close to zero, indicating that the instrumental variables are exogenous and not correlated. ‡ The results of the F-test show the instruments are not weak. Anderson-Rubin test confirms G1 and G2 smoking behaviors are endogenous. Additionally, based on the results of Wooldridge's regression-based test, it is appropriate to use an instrumental variable to address the endogeneity issue in our analysis. The placebo test provides a check on the strength and validity of our original instruments and shows that the third-stage CF is less likely to be biased by endogeneity. The likelihood ratio test comparing the third-stage CF and without IMR-1 and -2 included version resulted in a significant p-value, which indicates that there is strong evidence to suggest that the third-stage CF provides a significantly better fit to the data with the inclusion of IMR-1 and -2. Based on the AIC and BIC values, third-stage CF has a lower AIC (7272.684) and BIC (7332.557) compared to without IMR-1 and -2 included version (AIC: 7279.987, BIC: 7319.902). This suggests that the third-stage CF provides a better fit to the data regarding model complexity and goodness of fit. Robust standard errors are shown in parentheses. *** Significant at the 1% level. ** Significant at the 5% level. * Significant at the 10% level.

PATERNAL PERSPECTIVE, TABLE A.4

From the paternal perspective, a significant effect is found between G2 Smoking and Paternal G1 Smoking (0.108 and 0.109 in columns (3) and (4), respectively). Unlike the maternal perspective, the impact on G3 Smoking is not negative but rather insignificant in column 5 and positive in column (6) (0.039). The household economic conditions during G2's childhood show a significant negative effect on G2 smoking (-0.035 and -0.034 in columns (3) and (4), respectively), indicating that better economic conditions are associated with a lower likelihood of G2 smoking. However, these conditions do not significantly impact G3 smoking behaviors (columns (5) and (6)). The G2 year born variable does not exhibit a significant impact on G2 smoking in the CF model (column (3)), but it shows a slightly significant negative effect in the naïve OLS model (column (4)). Similarly, the G3 year born variable shows a significant negative effect on G3 smoking, suggesting that younger generations are less likely to smoke. As in the maternal perspective, the female variable shows a significant negative effect on G3 smoking (-0.058 in columns (5) and -0.059 in columns (6)), indicating that females in G3 are less likely to smoke than males.

These detailed results broaden our comprehension of the intergenerational transmission of smoking behaviors. The control variables, particularly economic conditions during childhood and gender, significantly modulate the inherited smoking behaviors across generations. Furthermore, these findings underscore the importance of considering socioeconomic and demographic factors in devising interventions to disrupt the transmission of harmful behaviors such as smoking.

TABLE A.4—DETAILED RESULTS FROM THE INVESTIGATION OF THE INFLUENCE OF GRANDPARENTAL SMOKING ON SUBSEQUENT GENERATIONS: A PATERNAL PERSPECTIVE.

	First-step: Paternal G1 Smoking		Second-step: G2 Smoking		Third-step: G3 Smoking	
Variables	*Marginal effects (CF)* (1)	Naïve *OLS* (2)	*Marginal effects (CF)* (3)	Naïve *OLS* (4)	*Marginal effects (CF)* (5)	Naïve *OLS* (6)
Paternal G1 Smoking			0.108***	0.109***	-0.017	0.039**
			(0.015)	(0.015)	(0.033)	(0.017)
G2 Smoking					0.118***	0.123***
					(0.017)	(0.018)
G2 household economic conditions during their childhood			-0.035***	-0.034**		
			(0.013)	(0.013)		
G2 Year born			0.001	-0.001*		
			(0.001)	(0.001)		
G3 household economic conditions during their childhood					-0.024	-0.024
					(0.020)	(0.020)
G3 Year born					-0.005***	-0.007***
					(0.002)	(0.001)
Female					-0.058***	-0.059***
					(0.015)	(0.015)
IMR-1			0.113*		-0.138**	
			(0.059)		(0.066)	
IMR-2					0.102***	
					(0.052)	
IV-2: Tobacco legislation in Norway: 1964			-0.074***			
			(0.017)			
IV-1: CPI at G2's birth year	0.065***					
	(0.018)					
Observations	4,057	4,057	4,057	4,057	4,057	4,057
R^2		0.000		0.008		0.026
Akaike Inf. Crit.	4,973.214		4,350.763		5,195.554	
$Cov(\hat{w}, Z_1)^{\dagger}$					0.005	
$Cov(\hat{v}, Z_2)$					-0.006	
$Cov(\hat{w}, \hat{v})$					0.027	
$Cov(Z_1, Z_2)$					0.054	
$Cov(\hat{u}, \hat{w})$					0.035	
$Cov(\hat{u}, \hat{v})$					0.014	
F-test of excluded instrument in first-stage					13.130***	
F-test of excluded instrument and IMR-1 in second-stage					19.484***	
Anderson-Rubin test statistic for endogeneity					4.934***	
Wooldridge test statistic					596.082***	
Placebo test result (p-value)					0.900	
Likelihood ratio test (p-value)					0.042	
Indirect effect of G1 on G3					0.015***	
Total effect					-0.002	

Note: We calculate the covariance between residuals (third-stage residual – $\hat{u}$, second-stage residual – $\hat{v}$, first-stage residual – $\hat{w}$) and two instrumental variables (CPI for beverages and tobacco at G2's birth year (Z_1) and the first official statement on smoking and health in Norway in 1964 (Z_2)). All covariances are close to zero, indicating that the instrumental variables are exogenous and not correlated. ‡ The results of the F-test show the instruments are not weak. Anderson-Rubin test confirms G1 and G2 smoking behaviors are endogenous. Additionally, based on the results of Wooldridge's regression-based test, it is appropriate to use an instrumental variable to address the endogeneity issue in our analysis. The placebo test provides a check on the strength and validity of our original instruments and shows that the third-stage CF is less likely to be biased by endogeneity. The likelihood ratio test comparing the third-stage CF and without $IMR-1$ and -2 included version resulted in a significant p-value, which indicates that there is strong evidence to suggest that the third-stage CF provides a significantly better fit to the data with the inclusion of $IMR-1$ and -2. Based on the AIC and BIC values, third-stage CF has a lower AIC (5195.554) and BIC (5246.02) compared to without IMR-1 and -2 included version (AIC: 5197.891, BIC: 5235.741). This suggests that the third-stage CF provides a better fit to the data regarding model complexity and goodness of fit. Robust standard errors are shown in parentheses. *** Significant at the 1% level. ** Significant at the 5% level. * Significant at the 10% level.

A.5. The Impact of Grandparental and Parental Smoking on G3 Alcohol Use Disorders Identification Test (AUDIT) Score

The Alcohol Use Disorders Identification Test (AUDIT) score is a widely accepted measure for assessing alcohol consumption and related problems (Babor et al., 2001). By exploring the influence of parental and grandparental smoking on G3's AUDIT score, we extend our analysis to the intergenerational transmission of other risky health behaviors. We use this continuous measure to gauge the risk of problematic drinking, intending to explore whether the observed intergenerational transmission of risky health behaviors, specifically smoking, also extends to other risky behaviors, such as binge drinking, which has been shown to be related to familial factors (Yu et al., 2023). This change in the dependent variable allows us to probe whether the intergenerational transmission of smoking behavior, as observed in our main findings, extends to other risky behaviors like alcohol misuse.

In the maternal lineage model (see Table A.5, column (1)), G2 smoking shows a positive and significant relationship at the 10% level with the G3 AUDIT score. This suggests that if G2 smoked, there's an associated increase in the AUDIT score of G3, implying a higher risk of alcohol misuse. This finding aligns with previous research showing a correlation between parental smoking and adolescent drinking behaviors (Yu et al., 2023). However, maternal smoking in G1 presents a negative, albeit insignificant, coefficient, suggesting that it may be associated with a lower AUDIT score in G3, in line with our expectations.

TABLE A.5—INTERGENERATIONAL TRANSMISSION OF RISKY HEALTH BEHAVIORS: THE IMPACT OF PARENTAL AND GRANDPARENTAL SMOKING ON G3'S ALCOHOL USE DISORDERS IDENTIFICATION TEST (AUDIT) SCORE.

Third-step: G3 AUDIT Score	Maternal grandparents CF (*OLS*) (1)	Paternal grandparents (2)
Maternal G1 Smoking	-0.171	
	(0.173)	
Paternal G1 Smoking		0.102
		(0.221)
G2 Smoking	0.221*	0.087
	(0.113)	(0.140)
Control variables for G3	Yes	Yes
$IMR-1$	Yes	Yes
$IMR-2$	Yes	Yes
Observations	3,914	2,750
Adjusted R^2	0.115	0.105

Note: The robustness checks for this analysis were conducted in the same manner as for the overall sample, using control function (CF) methods and including the first- and second-stage residuals, as well as other control variables for G3. The only difference in the analysis is that OLS regression is used instead of probit regression for the variable AUDIT score, as it is a continuous variable. First- and second-stage $IMR-1$ and -2 are used in control function (CF) method in the third-step (columns (1) and (2)). Robust standard errors are shown in parentheses.

*** Significant at the 1% level.

** Significant at the 5% level.

* Significant at the 10% level.

Paper III

Long-term effects of grandparental child neglect on grandchildren's mental health: *A Three-generation study*

Long-term Effects of Grandparental Child Neglect on Grandchildren's Mental Health: A Three-Generation Study

By EMRE SARI, MIKKO MOILANEN, AND MAARTEN LINDEBOOM*

Child neglect is a significant social problem with severe consequences for individuals and society. This study explores how intergenerational transmission of grandparental child neglect affects grandchildren's mental health in adulthood. Using a linear probability model, we analyze the nationally representative three-generation individual data set from the Tromsø Study, examining the role of maternal and paternal grandparents and highlighting the multigenerational long-term effects of child neglect. The results suggest that neglectful parenting behavior during a child's upbringing can lead to an increased risk of depression in adulthood. Moreover, our findings show that only maternal grandparents' neglectful parenting is associated with an increased probability of depression in their grandchildren, conditional on whether their parents neglected them. We contribute to understanding intergenerational transmission by examining the cumulative risk underlying the continuity of child maltreatment across generations.

JEL: D64, I14, I18, J12

Keywords: Child neglect, Adverse childhood experiences, Childhood trauma, Mental health, Depression

I. Introduction

Child neglect is a form of child maltreatment, a significant global problem that violates children's right to a healthy and violence-free life and affects their mental, emotional, and physical well-being in adulthood, as well as the well-being of society as a whole (World Health Organization, 2006). The long-term impact of child neglect can manifest in mental health problems in adulthood, such as depression and anxiety, and have far-reaching personal and societal consequences. The prevalence of these two mental health problems is rapidly increasing worldwide (Institute for Health Metrics and Evaluation, 2020), and they are also the most frequently observed mental health disorders in primary healthcare services in Norway (Norwegian Institute of Public Health, 2018). Through

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their widespread impact on individuals, depression and anxiety disorders impose a significant economic burden on healthcare systems and society (Morrissey & Kinderman, 2020). Therefore, it is crucial to understand the underlying mechanisms of these conditions to develop effective preventive measures and cost-effective policy designs (Persson & Rossin-Slater, 2018).

From the perspective of human capital theory, child neglect can affect an individual's human capital development in several ways. First, neglect can result in developmental delays, including cognitive, social, and emotional delays, which can have long-term effects on an individual's ability to learn and perform in school and the workforce (Vasileva & Petermann, 2018). Second, neglect can lead to physical health problems, such as malnutrition and untreated illness, which can also affect an individual's ability to learn and work (Vasileva & Petermann, 2018). Third, neglect can lead to psychological problems, such as depression and anxiety, affecting an individual's ability to function in society and the workplace (Dubowitz et al., 2022). Such mental health conditions can also hinder the accumulation of human capital, thereby reducing opportunities for employment and lowering adult earnings (Currie & Stabile, 2006; Mousteri et al., 2019). Thus, childhood neglect can, in addition to inter- and multigenerational effects, have profound and lasting effects on an individual's mental health and, ultimately, economic opportunities, making it an important area of study for economists seeking to address inequality and social mobility issues.

In this study, we examine the intergenerational transmission of child neglect and the resulting risk of depression, one of the most common mental health problems. Our research focuses on the relationship between grandparental neglect and grandchildren's mental health in adulthood. Two recent studies have addressed similar issues. Islam et al. (2023) examine the effects of breaking the cycle of child maltreatment across generations, and Su et al. (2022) review the intergenerational effects of maternal child maltreatment on offspring psychopathology through a meta-analysis of twelve studies. Our research goes beyond previous studies and makes valuable contributions. It evaluates the impact of grandparental child neglect on their grandchildren's risk of developing a mental health problem, shedding light on the underlying mechanism of the intergenerational transmission of child neglect. Specifically, we differentiate between the roles of maternal and paternal grandparents regarding the consequences for grandchildren's mental health in adulthood.

This study takes a more comprehensive approach than Islam et al. (2023) by examining the cumulative risk underlying the continuity of child neglect across generations. Our three-generation study also differs from previous research that relied mainly on the relationship between parents' adverse childhood experiences (see, e.g., Cooke et al. (2021, 2019); Lowthian et al. (2021); Seteanu & Giosan (2022)) and their children's mental health or the continuity of maltreatment across two generations (see, e.g., Armfield et al. (2021); Capaldi et al. (2019); Widom et al. (2015)). Our approach focuses on the long-lasting effects of child neglect by studying whether these effects differ between neglectful maternal and parental grandparents. Thus, while the literature focuses primarily on the intergenerational transmission between parents and their maternal grandparents (see,

e.g., Johnston et al. (2013); Lotto et al. (2021); Yang et al. (2018)), we extend these models to include paternal grandparents.

Why is Norway an important country to study the effects of childhood trauma across multiple generations? Despite its reputation as a welfare state with good population health, significant health inequalities persist in Norway, as argued by Mackenbach (2017), with differences among the largest in Europe. Most previous research on health in Norway has focused on the life course of one generation and its mental health outcomes (see, e.g., Broekhof et al. (2022); Haugland et al. (2021); Rueness et al. (2020)). Limited research has considered intergenerational perspectives (Grönqvist et al., 2017; Myhre et al., 2014). These studies suggest that early life experiences are crucial in shaping long-term health outcomes and that significant health inequalities may persist even in developed countries with strong social welfare systems. Our study is the first to examine the effects of childhood trauma across three generations in Norway, adding to the important body of research on health inequalities in the country.

Our main results show that childhood neglect increases the probability of mental health problems in grandchildren. Additionally, the impact of neglectful parenting on grandchildren is amplified when the maternal grandparents are also neglectful. Overall, our study adds to the literature on the intergenerational transmission of child neglect and highlights the importance of addressing this issue to improve the well-being of future generations.

II. Child Neglect

Neglect is the most prevalent form of child maltreatment and is characterized by the failure of a caregiver to meet a child's basic needs for food, shelter, clothing, medical care, and supervision (Fallon et al., 2020; Yang et al., 2018). Poverty and parenting characteristics are often associated with child neglect, which has serious long-term consequences for children's physical and mental health (Slack et al., 2004). It can be intentional or unintentional and have serious long-term consequences for children's physical and mental health.

Child neglect in Norway is defined as parents or other caregivers not meeting the basic physical needs of a child in daily life or ensuring their emotional well-being, sense of safety, and educational development (Stoltenborgh et al., 2013). In Norwegian, the term for child neglect is *omsorgssvikt*, which has a heavy connotation. Furthermore, under Norwegian law,[1] the childcare service is responsible for instituting early measures to prevent serious neglect cases. If a child is found to be neglected, the child's family faces serious legal consequences. Therefore, we must emphasize the gravity of the situation since the issue of child neglect is addressed head-on as *omsorgssvikt* rather than only indirectly, as in the Tromsø Study.

[1]Child Protection Services Act (*barnevernloven*) of 17 July 1992 No. 100. The Child Protection Act ensures that children and youth who live in conditions that may harm their health and development receive the necessary help and care at the right time. It also ensures safe growing conditions for children and youth.

III. Intergenerational Transmission of Child Neglect

Neglectful behavior of parents toward their children can impact the mental health of individuals across generations. This phenomenon has been observed in the literature and can be explained by various theories. However, we still do not comprehend its underlying mechanisms, which is of particular concern. Alink et al. (2019) noted that a better understanding of the mechanisms behind the intergenerational transmission of child neglect is urgently needed to develop effective interventions to prevent maltreatment in subsequent generations.

The intergenerational transmission of child neglect has been attributed to the intergenerational cycle of violence hypothesis (Abramovaite et al., 2015). This hypothesis suggests that childhood maltreatment can lead to aggression through increased mental health symptoms or emotional dysregulation (Brennan et al., 2021; Newton, 2019), resulting in a cycle of abuse and neglect that can be perpetuated without any genetic link (Abramovaite et al., 2015). A study by Langevin et al. (2023) found that maternal emotional dysregulation and mother-to-child attachment are factors in the intergenerational continuity of child maltreatment. Widom (2017) study also showed that childhood neglect leads to the intergenerational transmission of abuse and neglect through the parent–child relationship. These findings suggest that grandparents' neglect of their own children can lead to the intergenerational transmission of child maltreatment, leading to mental health outcomes such as depression in their adult grandchildren.

Child neglect has far-reaching implications, and one of these outcomes is the transmission of adversity and risk to subsequent generations, meaning that children who experience neglect are more likely to repeat the cycle of neglect as adults, either as neglectful parents themselves or as victims of neglectful relationships (Bartlett et al., 2017; Berlin et al., 2011; Islam et al., 2023; Lamela & Figueiredo, 2018; Madigan et al., 2019). This cycle of neglect can have significant societal and economic costs, including increased healthcare utilization, social service involvement, and criminal justice involvement (World Health Organization, 2020).

Moreover, studies have shown that a parent's experience of child maltreatment increases the probability of engaging in abusive behavior toward their child (Armfield et al., 2021; Capaldi et al., 2019; Widom et al., 2015; Yang et al., 2018). Some earlier empirical studies indicate a relationship between grandparental investment and mental health problems in their grandchildren (Flouri et al., 2010; Jappens & Van Bavel, 2020; Sadruddin et al., 2019), while Tanskanen & Danielsbacka (2018) argue that the relationship is not necessarily causal (Helle et al., 2022). In this context, several researchers have proposed conceptual mechanisms to explain the impact that grandparents exert on their grandchildren and investigate the continuation and discontinuation of the intergenerational transmission of child maltreatment, such as Dixon et al. (2009); Egeland et al. (1988); Islam et al. (2023); Jaffee et al. (2013); Mckenzie et al. (2021). The most recent study, Islam et al. (2023), compares the severity of offspring psychopathology in families with no history of maltreatment to those in which parents, offspring, or both experienced childhood maltreatment. Mainly, they examine the impact of the cumulative risk of grandparental and parental childhood maltreatment on a grandchild's mental

health, test the so-called *additive risk hypothesis*, and find no evidence to support it.

Understanding how grandparental neglect can affect the mental health of their grandchildren requires a theoretical explanation of how maltreatment can be transmitted across generations. **Social learning theory** is fundamental for understanding the transmission of maltreatment, particularly physical maltreatment and harsh parenting (Bandura, 1973). According to this theory, parents are alleged to repeat the parenting practices of their own parents due to their misperceived "positive effects" (Alink et al., 2019). They normalize the use of physical maltreatment as a form of discipline by modeling the physical neglect perpetrated by their own parents (Erten & Keskin, 2020). According to research (Badenes-Ribera et al., 2020; Yang et al., 2018), physical abuse increases the risk of subsequent harsh parenting or the physical abuse of one's own children. Another important channel to consider is the impact of grandparental neglect on the attachment styles of their children, who are the parents of the grandchildren. **Attachment theory** examines how parent–child relationships develop and shape children's character (Bowlby, 1978). Maltreated children have insecure or disorganized attachments compared to those of nonmaltreated children, and insecure or unresolved adult attachments are related to subsequent parenting problems and maltreatment behaviors (Cyr et al., 2010; Marshall et al., 2022; Reijman et al., 2017).

When considering the intergenerational transmission of child neglect through the lens of attachment theory, it is important to recognize that the effects may differ between maternal and paternal grandparents (Crocetto, 2019; Yehuda & Lehrner, 2018). Studies have suggested that maternal grandparents may have a greater influence on their grandchildren than paternal grandparents, possibly due to the more frequent and involved caregiving role of mothers in traditional family structures (Chan & Elder, 2000; Coall & Hertwig, 2010). However, this is a complex and multidimensional phenomenon that requires further investigation, emphasizing the importance of distinguishing between maternal and paternal grandparents.

By examining the impact of maternal and paternal grandparents separately, we aim to expand upon the current literature and investigate the following research questions: *To what extent does grandparental child neglect in the first generation predict the probability of mental health problems in the third generation, and whether neglectful maternal and paternal grandparents have differential effects on their grandchildren?*

IV. Data

Our study uses individual-level data from the Tromsø Study,[2] a cohort study involving the residents of Tromsø, the largest city in Northern Norway with approximately 77,000 inhabitants. From 1974 to 2016, the Tromsø Study, also known as Tromsø 1-7, has been conducted in seven waves, with participation rates ranging from 64.7% to 78.5% (Jacobsen et al., 2012; Sari et al., 2021). The survey data include health-related information on the adult population residing in Tromsø and is representative of the overall adult population in Norway, as indicated by previous studies (Jacobsen et al., 2012; Olsen et al.,

[2]Regional Committees for Medical and Health Research Ethics approved the study, and informed consent was obtained from each participant prior to their enrollment. For more details, https://uit.no/research/tromsostudy

2020).[3]

Specifically, we used data from the Tromsø7 study conducted between 2015 and 2016. The reason for selecting this particular study was the presence of the child neglect variable in the data set. Using family ID numbers provided by the Norwegian Tax Administration, we were able to establish family connections between participants in the Tromsø Study for the first time within the context of this study. Our sample has 1,258 observations, including participants from two generations, grandchildren (G3) and their parents (G2). Information regarding the maternal and parental grandparents (G1) was gathered through responses from the G2 participants. Figure 1 presents the definitions of generations and demonstrates the linkages between family members across generations.

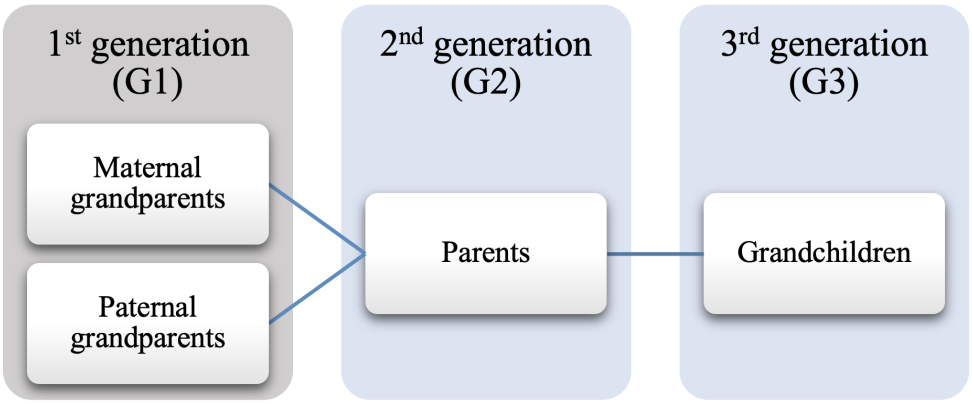

FIGURE 1. DEFINITION AND PRESENTATION OF GENERATIONS.

Note: The order of generations begins with the generation of the grandparents, labeled G1. G1 indicates the maternal and paternal grandparents, while G2 indicates the parents of the grandchildren. G2 and G3 are participants in the Tromsø Study, as indicated by the blue boxes. The gray box indicates that G1 is not a participant in the Tromsø Study. G2 responds to their experience of child neglect by G1, while G3 responds to their experience of child neglect by G2.

A. Measures

Dependent variable: depression. — We assessed the mental health status of the G3 using self-report measures of depression as a proxy. To trace the mental health of G3, we consider the respondents' answers to the question, "Have you felt depressed or sad during the last week?". To facilitate data analysis, we dichotomized responses, following previous research (Byrow et al., 2022; Kim et al., 2021). Responses indicating "no complaint" were used as a reference point, while those indicating "little," "pretty much," or "very much" complaints were considered indicative of depression in G3. With this classification, depression was reported by 29.7% of participants in our study sample (Table

[3]Individuals with a university degree were slightly overrepresented in the study sample; however, overall, the population is regarded as typical of the Norwegian adult population as a whole (Olsen et al., 2020).

1).

Covariates. — Child neglect in this context refers to parents/caregivers who did not provide their children with the basic necessities of food, clothing, shelter, and care/love. The child neglect variable for grandparents referred to the failure of G1 parents to provide adequate care for their children (G2); the same applied to the failure of G2 parents to provide adequate care for their children (G3). Among the G2s in our sample, 4.8% neglected their G3 child (Table 1). Among G1s, 9.2% of maternal G1s and 5.9% of paternal G1s were neglectful.

We also control for several individual-level demographic and socioeconomic status variables for G3, G2, and G1, as shown in Table 1. Household economic status is used as a proxy for socioeconomic status, and also controls for the gender, year of birth, and marital status of G3 individuals are included (Berlin et al., 2011; Haugland et al., 2021; Kong et al., 2021; Langevin et al., 2023). Furthermore, we also include total taxable household income[4] for G3 individuals in the year prior to their participation in the study (Lamela & Figueiredo, 2018). The responses were divided into two groups, which is consistent with Statistics Norway's household income and wealth statistics (Statistics Norway, 2022). Those who earned 551,000-750,000 Norwegian kroner or more were classified as the higher income group, while the rest were classified as the lower income group, which serves as a reference. The household economic status of G2 and G1 individuals during upbringing is also included (Maniar et al., 2019) and grouped into two categories: lower and higher, with the lower as the reference group.

[4]Total taxable income includes income from work, social benefits, and the like.

TABLE 1—STUDY SAMPLE CHARACTERISTICS.

Variable names	*N*	% (Median)[a]	Standard deviation	Min	Max
Third generation (G3) – *Grandchildren*					
Mental health status					
Not depressed (ref)	957	70.3			
Depressed	404	29.7			
Gender					
Female (ref)	685	50.3			
Male	676	49.7			
Marital status[b]					
Single (ref)	642	47.2			
Married	719	52.8			
Year of birth	1,361	1969	4.77	1951	1975
Household income					
Lower (ref)	247	18.1			
Higher	1,114	81.9			
Second generation (G2) – *Parents*					
Child-neglecting parents					
No (ref)	1,295	95.2			
Yes	66	4.8			
Household economic status[c]					
Lower (ref)	165	12.1			
Higher	1,196	87.9			
First generation (G1) - *Grandparents*					
Maternal child-neglecting grandparents[*]					
No (ref)	1,209	90.8			
Yes	123	9.2			
Paternal child-neglecting grandparents[*]					
No (ref)	1,214	94.1			
Yes	76	5.9			
Household economic status[c]					
Lower (ref)	655	48.1			
Higher	706	51.9			

Note: [a] The median of the year of birth variables for individuals in G3 and G2 is reported. [b] Marital status is divided into two categories: "single", which includes persons who are single, widowed, divorced, or separated, and "married", which includes persons in registered partnerships. [c] To classify the household economic status of G2 raising G3 and G1 raising G2, two categories were created based on responses to the question, "How was your family's financial situation during childhood?" Responses indicating "difficult" and "very difficult" were grouped under lower household economic status, while responses indicating "good" and "very good" were grouped under higher household economic status. [d] Child-neglecting grandparents are at least maternal or paternal grandparents who neglected their child (G2) while raising them. Below are the shares of child-neglecting grandparents, presented separately for maternal and paternal grandparents (G1). [*] These variables in our sample have some missing observations, but the proportion of missing data is trivially low. To address this issue, we used listwise deletion method (Conde & Poston, 2020; Hoover & Perez, 2004).

V. Empirical Strategy

We use a linear probability model with ordinary least squares regression to estimate the associations between G1 and G2 child neglect and the mental health status of their G3. In support of our econometric approach, previous research conducted by Hellevik (2009) demonstrates the utility of linear regression in modeling binary dependent variables. Our approach provides a significant advantage by producing coefficients and proportional differences that can be interpreted as the change in probability of a specific dependent variable value while keeping all other independent variables constant (Wooldridge, 2010). While the interpretation of linear regression results is both meaningful and straightforward, the same cannot be said for loglinear association measures, which lack a comparable level of clarity (Hellevik, 2009). However, as highlighted by Hellevik (2009) and Mood (2010), heteroscedasticity can present a challenge for linear probability models. To account for this issue, we employ heteroscedasticity-robust standard errors.

Our empirical model aims to estimate the probability of a relationship between G3 mental health status, specifically depression, and both G1 and G2 child neglect, defined as the failure of parents/caregivers to provide adequate care for their children. The equation used for this estimation is as follows:

$$Y_{G3} = \beta_0 + \beta_1 S_{G1} + \beta_2 S_{G2} + \beta_3 S_{G1} * S_{G2} + \beta_4 C + \varepsilon, \tag{1}$$

where Y_{G3} is a binary variable that equals one if G3 reported a mental health problem and zero otherwise. As mentioned, G3 represents the adult grandchild generation, G2 represents the parent generation, and G1 represents the grandparent generation. S_{G2} is a binary variable that equals to one if at least one of the G2 parents/caregivers neglected G3 during their upbringing, and zero otherwise. Similarly, S_{G1} is a binary variable that equals to one if at least one of the G1 parents/caregivers neglected G2 during their upbringing, and zero otherwise. C is the vector of the control variables. β_0 is the intercept; β_1 and β_2 are the coefficients on the main explanatory variables, G1 child neglect and G2 child neglect, respectively. The estimated coefficient β_2 can be interpreted directly as the change in the probability of a grandchild having poorer mental health due to the parents' child neglect. The interaction term $S_{G1} * S_{G2}$ in our model captures the joint effect of G1 and G2 child neglect on G3 mental health problems, testing the additive risk hypothesis. The coefficient β_3 measures the change in the probability of G3 mental health problems associated with the interaction between G1 and G2 child neglect. A positive and statistically significant β_3 would indicate that the effect of grandparental neglect on G3 mental health is greater when parental neglect is also present, possibly indicating that parental neglect amplifies the adverse effects of grandparental neglect on G3 mental health. In addition, we include the separate variables for maternal and paternal G1 child neglect in our model to explore whether the effect on G3 mental health differs based on the parental side. β_4 is a vector of coefficients on the control variables, and ε is the error term.

To validate the reliability of our findings, we also conduct probit regressions using

the same set of variables as our main model to estimate the average marginal effect (Wooldridge, 2010). Our analysis show that the average marginal effects of the probit model closely matched the OLS coefficients, indicating that the results are robust to changes in the regression method used. It is worth noting that we use the term "effect," we are referring to a statistical association and not insinuating any causal link between variables.

VI. Results

Our analysis begins by examining whether our results are consistent with those of Islam et al. (2023) concerning the testing of the additive risk hypothesis. Table 2 presents the results of estimations that investigate the intergenerational transmission of child neglect[5] and its effect on the mental health of the grandchildren generation (G3). Columns (1) and (2) report the OLS regression, and columns (3) and (4) report the probit marginal effects for G3 mental health status.

TABLE 2—RESULTS OF THE EFFECT OF NEGLECT FROM GRANDPARENTS AND PARENTS ON GRANDCHILDREN'S MENTAL HEALTH.

	Dependent variable: *Mental health status of G3*			
	OLS		Probit (Marginal effects)	
Variables	(1)	(2)	(3)	(4)
G2 Child-neglect	0.202***	0.200***	0.204***	0.202***
	(0.064)	(0.083)	(0.065)	(0.084)
G1 Child-neglect	-0.008	-0.008	-0.011	-0.011
	(0.036)	(0.037)	(0.037)	(0.039)
G1 Child-neglect x G2 Child-neglect		0.004		0.005
		(0.128)		(0.115)
Control variables	√	√	√	√
Observations	1,361	1,361	1,361	1,361
R-squared	0.027	0.027		
AIC			1,638.6	1,640.6

Note: Columns (1) and (2) present coefficients from OLS regressions, while columns (3) and (4) present marginal effects from probit regressions. The interaction between G1 child neglect and G2 child neglect is reported in columns (2) and (4). We have adjusted all estimates for G3's gender, year of birth, marital status, household income, and the economic status of both G2 and G3 households during their children's upbringing. The results for these control variables are presented in Appendix Table A.2 for OLS results, and Appendix Table A.3 for probit regressions. We assessed G3's mental health status using self-reported measures of depression. Heteroskedasticity-robust standard errors are shown in parentheses for OLS models, while delta method standard errors are shown in parentheses for probit models. AIC is Akaike Information Criterion.

*** Significant at the 1% level.

** Significant at the 5% level.

* Significant at the 10% level.

[5]Our additional results show that the coefficient for G1 child neglect on G2 child neglect is highly significant in both OLS and probit models at the 1% level (Appendix Table A.1). This result indicates a strong and positive correlation between grandparents (G1) neglecting their children and the likelihood of their offspring (G2) neglecting their own children, which is consistent with previous research in the literature (Yang et al., 2018).

The results in Table 2 show that there is a significant positive relationship between parents (G2) who neglect the children (G3) and the mental health status of their children, which holds in both OLS and probit models. The estimated coefficients for G2 child neglect in the OLS and the average marginal effect in the probit model are 0.202 (Table 2, column (1)) and 0.204 (Table 2, column (3)), respectively. These results indicate that child neglect among G2 is associated with an increased probability of poor mental health in G3. However, there is no significant relationship between having a child-neglecting G1 and G3 mental health status. This suggests that having a child-neglecting G1 may not have a direct effect on the mental health of grandchildren.

We further look at the interaction between G1 child neglect and G2 child neglect to examine whether the presence of G2 child neglect amplifies the effect of G1 child neglect on G3 poorer mental health (Table 2, columns (2) and (4)). As in Islam et al. (2023), this interaction term is not statistically significant, suggesting that the effect of G1 child neglect on G3 mental health is not moderated by the presence or absence of G2 child neglect. It is important to note, however, that the absence of a significant interaction effect does not necessarily mean that G1 child neglect has no effect on G3 mental health status. Therefore, to further examine the potential impact of G1 child neglect on G3 mental health status, we differentiate maternal and paternal grandparent child neglect as separate variables in our model, which is presented in Table 3.

Table 3 presents findings on the intergenerational transmission of child neglect and its impact on the risk of mental health problems, examining the effect of neglect from maternal and paternal grandparents on their grandchildren. The results show that similar to the results in Table 2, G2 child neglect still has a positive and statistically significant association with poorer offspring (G3) mental health in all specifications. Maternal G1 child neglect and paternal G1 child neglect do not show a statistically significant direct relationship with G3 mental health problems.

However, the interaction variables between maternal and paternal G1 child neglect and G2 child neglect produce interesting results (Table 3). The positive coefficient value for the interaction between maternal G1 child neglect and G2 child neglect indicates that the adverse effect of maternal G1 child neglect on G3 mental health is stronger when combined with child neglect experienced by G2. Thus, the effect of childhood maltreatment on G3, together with neglectful maternal G1, is worse on offspring's mental problems than the effect of childhood maltreatment by parents (G2) alone. We do not find this effect for parental grandparents. In summary, the results show that grandparents' effects on their grandchildren's mental health differ depending on whether they are maternal or paternal G1s.

TABLE 3—RESULTS OF THE EFFECT OF CHILD NEGLECT FROM MATERNAL AND PATERNAL GRANDPARENTS AND PARENTS ON GRANDCHILDREN'S MENTAL HEALTH.

	Dependent variable: *Mental health status of G3*			
	OLS		Probit (Marginal effects)	
Variables	(1)	(2)	(3)	(4)
G2 Child-neglect	0.249***	0.199***	0.251***	0.201***
	(0.075)	(0.083)	(0.076)	(0.084)
Maternal G1 Child-neglect	-0.004	-0.052	-0.009	-0.057
	(0.062)	(0.065)	(0.067)	(0.068)
Paternal G1 Child-neglect	0.039	0.042	0.037	0.040
	(0.070)	(0.072)	(0.073)	(0.075)
Maternal G1 Child-neglect x G2 Child-neglect		0.379**		0.426*
		(0.180)		(0.222)
Paternal G1 Child-neglect x G2 Child-neglect		-0.059		-0.053
		(0.324)		(0.293)
Control variables	√	√	√	√
Observations	1,258	1,258	1,258	1,258
R-squared	0.024	0.027		
AIC			1,517.6	1,518.2

Note: Columns (1) and (2) present coefficients from OLS regressions, while columns (3) and (4) present marginal effects from probit regressions. The interaction between maternal and paternal G1s child neglect and G2 child neglect are reported in columns (2) and (4). We have adjusted all estimates for G3's gender, year of birth, marital status, household income, and the economic status of both G2 and G3 households during their children's upbringing. The results for these control variables are presented in Appendix Table A.4. We assessed G3's mental health status using self-reported measures of depression. Heteroskedasticity-robust standard errors are shown in parentheses for OLS models, while delta method standard errors are shown in parentheses for probit models. AIC is Akaike Information Criterion.

*** Significant at the 1% level.

** Significant at the 5% level.

* Significant at the 10% level.

VII. Discussion

Child neglect is a pervasive problem with severe implications for the well-being of individuals and society as a whole. Our study aims to examine the intergenerational transmission of child neglect and its impact on adult mental health. Our results suggest that the cumulative effect of childhood maltreatment by both neglectful maternal grandparents and parents is worse for the mental health of the grandchildren than the effect of childhood maltreatment by parents alone.

Our study contributes significantly to the literature on the intergenerational transmission of child neglect by taking a comprehensive approach to examine the submechanisms perpetuating child maltreatment across generations. We focus on the role of grandparents in shaping the mental health outcomes of future generations and extend previous research by including grandparents from both sides in the model of intergenerational transmission between parents and children. We provide evidence for the additive risk hypothesis, which suggests that exposure to child neglect adversely affects future generations. The negative effects of child neglect are amplified across multiple generations by

the repetition of child neglect in each generation. This finding contradicts the findings of Islam et al. (2023).

Enlow et al. (2018); Langevin et al. (2023); Noll et al. (2009) discuss this intergenerational continuity of cumulative child maltreatment and the potential negative impact on offspring mental health. Langevin et al. (2023) present that maternal emotional dysregulation and mother-to-child attachment plays a key role in this continuity. They highlight the need for additional support for families affected by multiple forms of violence. Enlow et al. (2018) also discusses the physiological and neurocognitive vulnerabilities that can result from exposure to negative environmental influences such as offspring maltreatment, leading to a trajectory toward poor mental health. Noll et al. (2009) emphasize the burden that children born into adversity are required to bear and compare the magnitude of this burden across demographically similar groups of individuals who differ in the presence of maternal childhood sexual abuse.

Our finding of the intergenerational transmission of child neglect between grandparents and parents is consistent with previous research on the intergenerational transmission of child neglect, which has demonstrated that children who experience neglect are more likely to repeat the cycle of neglect as adults (Bartlett et al., 2017; Berlin et al., 2011; Islam et al., 2023; Madigan et al., 2019). These studies mainly suggest that neglectful grandparents may influence the parenting behaviors of their own children, who may in turn transmit the same parenting style to their own children, perpetuating the cycle of neglect across generations. This idea is supported by previous studies that have shown that parenting styles are influenced by parental experiences and that these experiences are passed on to subsequent generations (Capaldi et al., 2019; Seteanu & Giosan, 2022).

Our findings also support the theoretical predictions of intergenerational transmission of child neglect, which suggests that individuals who experience neglectful behavior during childhood are more likely to repeat the cycle of neglect as adults. Social learning theory suggests that children learn from observing their parents and grandparents' behavior, and the experience of neglect can increase the probability of perpetuating neglectful behavior in future generations (Kong et al., 2021). Neglectful parenting can lead to insecure attachment styles in children, which can increase the likelihood of mental health problems later in life (Schore, 2001). Another possible explanation is that child neglect can lead to attachment issues and disruptions in the parent–child relationship, which can increase the risk of depression in children. Attachment theory focuses on the emotional bonds between parents and children. Children who experience neglect may not develop secure attachment relationships with their caregivers, leading to a range of negative outcomes, including poor emotional regulation and difficulties forming healthy relationships in adulthood (Marshall et al., 2022). These negative outcomes may then be passed on to the next generation (Robboy & Anderson, 2011), as the children of neglectful parents may also struggle to form secure attachments with their own children.

Grandparents and grandchildren can also be distinguished in terms of the sex of either the grandchild or the grandparent (Dunifon & Bajracharya, 2012; Yehuda & Lehrner, 2018). Demographic changes and historical family structures have a profound impact

on family dynamics and intergenerational transmission. Although maternal grandparents may have a significant effect on grandchildren, the absence of a similar effect for paternal grandparents does not necessarily mean there is no intergenerational transmission through the paternal side. Considering the traditional gender roles of the family in mid-1900s Norway, we know that mothers were primarily responsible for childcare (Lorentzen, 2013), which may contribute to the observed differences between maternal and paternal grandparents in the transmission of values and behaviors to the next generation. An evolutionary explanation is also at the forefront of theories that consider this phenomenon (Coall & Hertwig, 2010; Yehuda & Lehrner, 2018). In particular, maternal grandmothers are more certain of their biological ties to their grandchildren than other grandparents and make a more significant investment in their grandchildren's lives. Maternal grandparents who neglect their own children may pass on this parenting style and behavior to their children, who in turn may be more likely to neglect their own children, including their grandchildren.

A. Policy implications

To address child neglect and its detrimental effects on mental health, policymakers should prioritize the implementation of support programs that target parents who lack necessary parenting skills. Such programs could take the form of parental education and training, providing parents with the tools and resources necessary to promote healthy attachment and positive parenting behaviors. In addition, policymakers should consider improving access to mental health services, particularly for those who have experienced neglect or have a family history of neglectful parenting. This could include increasing funding for mental health services and community-based programs, as well as incentivizing mental health providers to specialize in treating individuals who have experienced childhood neglect. Overall, implementing such interventions would not only improve the mental health of individuals who have experienced neglect but would also help break the cycle of neglectful parenting, ultimately leading to better outcomes for families and society.

B. Limitations and future directions

While our study contributes to the literature on the intergenerational transmission of child neglect, several limitations should be considered. Our study relies on self-reported data, which can lead to recall bias and social desirability bias. Future studies could incorporate observational data to improve the validity of the results. Additionally, our study is limited to Norway, and the results may not be generalizable to other countries. Therefore, future research should investigate the intergenerational transmission of neglect in diverse populations to examine whether the findings are consistent across different cultural contexts. Additionally, the sample size may limit the statistical power of our analysis, and future studies with larger samples are needed to confirm our findings.

Moreover, our data, like those used by Duarte et al. (2016), do not include information on whether a grandparent lives in the same household as the offspring during their childhood. Additionally, we construct family linkages using register data. Mothers in Norway

are the ones to make the initial declaration of paternity, with the father following suit. Due to our emphasis on the role of environmental factors, such as extended family, rather than on genetic predisposition, we do not expect the possibility of a nongenetic father to skew our findings significantly.

One potential limitation of our current design is the challenge of establishing causality, as it is uncertain whether poorer mental health biases the individual's perception of their parents or if the actual lack of care contributes to the development of depression. As pointed out by Islam et al. (2023), there is a possibility that children who have a predisposition to psychopathology may be more likely to experience negative parenting behaviors, such as abuse and neglect, rather than the other way around. Another limitation is that the measure used to assess parent–child relationships may reflect perceived rather than actual relationships, which the illness of the participants may influence. Nonetheless, Koszycki et al. (2010) note that the perceived characteristics of parents hold significance in the development of psychological disorders and should not be disregarded.

It is important to note that the relationship between maternal grandparents' child neglect and their grandchildren's depression is complex and may be influenced by a range of factors. Therefore, there are several directions for future research that could build on our study. Future studies could investigate the potential genetic mechanisms underlying the intergenerational transmission of child neglect. It would be important to explore the extent to which genetic factors contribute to the continuity of child neglect across generations. Future studies should also explore the potential moderating factors that may influence the intergenerational transmission of child neglect, such as the quality of the grandparent-grandchild relationship, the type and severity of childhood maltreatment experienced by the grandparents, and the cultural and social context in which transmission occurs. Moreover, longitudinal studies are needed to examine the long-term effects of grandparents' neglect on their grandchildren's mental health outcomes and to determine the critical periods for intervention.

Further research is needed to better understand the mechanisms underlying this relationship and to identify potential interventions to break the cycle of intergenerational child neglect and depression.

VIII. Conclusions

Our study contributes to the literature on the intergenerational transmission of child neglect by examining the impact of grandparents on the mental health outcomes of their grandchildren. The results suggest that the probability of depression in grandchildren is amplified when both their maternal grandparents and parents were neglectful in their parenting. Our study provides insight into a potential mechanism underlying this transmission of child neglect and evidence for the additive risk hypothesis. The study also supports the theoretical framework of intergenerational transmission of child neglect and highlights the role of parenting styles and attachment in this transmission. Overall, our study underscores the need for interventions to break the cycle of intergenerational transmission of neglect and promote the mental health of future generations.

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APPENDIX

APPENDIX

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Long-term effects of grandparental child neglect on grandchildren's mental Health:

A three-generation study

by

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The appendix tables provide detailed results on the intergenerational transmission of child neglect and its effects on mental health status. The tables below present results from OLS and probit regressions. These additional tables are not necessary for the main argument of the study, but they provide more detailed information. Therefore, we include them in the appendix to keep the main text focused on the main findings and conclusions. In addition, the step-by-step and detailed presentation of the results with control variables is important to show how the models were developed and to demonstrate the robustness of the results across different modeling approaches. Overall, the tables in the appendix provide additional evidence and insight into the main results presented in the paper.

Table A.1 shows the results of the intergenerational transmission of child neglect, exploring the effect of neglect from grandparents to parents. The table presents results from both OLS regressions (columns (1), (2), and (3)) and probit regressions (columns (4), (5), and (6)). The coefficients in columns (1), (2), and (3) indicate the relationship between G1 Child-neglect and G2 Child-neglect while controlling for G2 and G1 household economic status while raising their children (G2 for G1, and G3 for G2). The results show that G1 Child-neglect has a significant positive effect on G2 Child-neglect and that the higher household economic status of G2 has a significant negative effect on G2 Child-neglect. It is noteworthy that the magnitude of the coefficients is quite similar between the OLS and probit regressions, indicating that the results are robust across different modeling approaches.

TABLE A.1—DETAILED RESULTS OF THE INTERGENERATIONAL TRANSMISSION OF CHILD NEGLECT: EXPLORING THE EFFECT OF NEGLECT FROM GRANDPARENTS TO PARENTS.

	Dependent variable: *G2 Child-neglect*					
	OLS			Probit (Marginal effects)		
Variables	(1)	(2)	(3)	(4)	(5)	(6)
G1 Child-neglect	0.116***	0.096***	0.096***	0.116***	0.083***	0.083***
	(0.026)	(0.025)	(0.025)	(0.026)	(0.023)	(0.024)
G2 household economic status		-0.102***	-0.102***		-0.085***	-0.085***
		(0.028)	(0.028)		(0.025)	(0.026)
G1 household economic status			-0.002			0.000
			(0.012)			(0.010)
Observations	1,361	1,361	1,361	1,361	1,361	1,361
R-squared	0.036	0.059	0.059			
AIC				496.387	478.146	480.146

Note: Columns (1), (2), and (3) present coefficients from OLS regressions, while columns (4), (5) and (6) present marginal effects from probit regressions. Heteroskedasticity-robust standard errors are shown in parentheses for OLS models, while delta method standard errors are shown in parentheses for probit models. AIC is Akaike Information Criterion.
*** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

Table A.2 presents the results of OLS regressions examining the effect of neglect from grandparents on the mental health status of grandchildren, controlling for various demo-

graphic and economic factors. The dependent variable is the mental health status of G3 (grandchildren). The main independent variable of interest is G2 Child-neglect (neglect experienced by the parent of G3). The coefficients for G2 Child-neglect are positive and statistically significant at the 1% level across all eight specifications. The coefficient for G1 child-neglect is not statistically significant in any of the eight specifications. This indicates that there is no evidence for the direct effect of grandparental neglect on the mental health of their grandchildren. Other control variables in the model include G3 gender, G3 year of birth, G3 marital status, G3 household income, G2 household economic status, G1 household economic status, and G1 child-neglect x G2 child-neglect interaction term. Among these, higher household income of G3 is negatively associated with the mental health of grandchildren, while being married for G3 is negatively associated with mental health in some specifications. Overall, the results suggest that the effect of neglectful parents in childhood has a significant impact on the mental health of their children in adulthood, while the direct effect of grandparental neglect on the mental health of their grandchildren is not significant.

TABLE A.2—DETAILED RESULTS OF THE EFFECT OF NEGLECT FROM GRANDPARENTS AND PARENTS ON GRANDCHILDREN'S MENTAL HEALTH: OLS REGRESSIONS.

	Dependent variable: *Mental health status of G3*				
	OLS				
Variables	(1)	(2)	(3)	(4)	(5)
G2 Child-neglect	0.210***	0.206***	0.212***	0.202***	0.200**
	(0.064)	(0.063)	(0.063)	(0.064)	(0.083)
G1 Child-neglect	0.010	0.008	0.001	-0.008	-0.008
	(0.036)	(0.036)	(0.035)	(0.036)	(0.037)
G3 Gender		-0.028	-0.020	-0.021	-0.021
		(0.025)	(0.025)	(0.025)	(0.025)
G3 Year of birth		-0.0001	0.001	0.001	0.001
		(0.003)	(0.003)	(0.003)	(0.003)
G3 Marital status		-0.051**	-0.008	-0.008	-0.008
		(0.025)	(0.026)	(0.027)	(0.027)
G3 Household income			-0.141***	-0.140***	-0.140***
			(0.037)	(0.037)	(0.037)
G2 Household economic status				-0.042	-0.042
				(0.040)	(0.040)
G1 Household economic status				0.008	0.008
				(0.025)	(0.025)
G1 Child-neglect x G2 Child-neglect					0.004
					(0.128)
Observations	1,361	1,361	1,361	1,361	1,361
R-squared	0.010	0.014	0.026	0.027	0.027

Note: All columns present coefficients from OLS regressions. Heteroskedasticity-robust standard errors are shown in parentheses for OLS models.
*** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

Table A.3 presents the detailed results on the intergenerational transmission of child neglect and the risk of mental health problems, exploring the effect of neglect from grandparents to grandchildren using probit regression to estimate the average marginal effects. The table presents the marginal effects for the dependent variable G2 Child-neglect, with different independent variables including G1 Child-neglect and G2 household economic status. The results show that G1 Child-neglect has a significant positive effect on G2 Child-neglect and that G2 household economic status has a significant negative effect on G2 Child-neglect.

TABLE A.3—DETAILED RESULTS OF THE EFFECT OF NEGLECT FROM GRANDPARENTS AND PARENTS ON GRANDCHILDREN'S MENTAL HEALTH: PROBIT REGRESSIONS.

	Dependent variable: *Mental health status of G3*				
	Probit (Marginal effects)				
Variables	(1)	(2)	(3)	(4)	(5)
G2 Child-neglect	0.210***	0.206***	0.215***	0.204***	0.202**
	(0.064)	(0.064)	(0.064)	(0.065)	(0.084)
G1 Child-neglect	0.010	0.007	-0.001	-0.011	-0.011
	(0.036)	(0.036)	(0.036)	(0.037)	(0.039)
G3 Gender		-0.028	-0.021	-0.023	-0.023
		(0.025)	(0.025)	(0.025)	(0.025)
G3 Year of birth		0.000	0.001	0.001	0.001
		(0.003)	(0.003)	(0.003)	(0.003)
G3 Marital status		-0.051**	-0.008	-0.008	-0.008
		(0.025)	(0.027)	(0.027)	(0.027)
G3 Household income			-0.142***	-0.140***	-0.140***
			(0.038)	(0.038)	(0.038)
G2 Household economic status				-0.043	-0.043
				(0.041)	(0.041)
G1 Household economic status				0.008	0.008
				(0.025)	(0.025)
G1 Child-neglect x G2 Child-neglect					0.005
					(0.115)
Observations	1,361	1,361	1,361	1,361	1,361
AIC	1,648.763	1,649.229	1,635.892	1,638.569	1,640.567

Note: All columns present marginal effects from probit regressions. Delta method standard errors are shown in parentheses. AIC is Akaike Information Criterion.
*** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

Comparing Table A.2 and Table A.3, we can observe that the coefficients of the independent variables (G2 child neglect, G1 child neglect, G3 gender, etc.) are similar in both models. This indicates that both OLS and probit models provide similar estimates of the effect of these variables on the mental health status of G3. Overall, both OLS and probit models seem to provide similar estimates of the effect of child neglect on the mental health status of grandchildren.

Table A.4 presents the findings of a regression analysis that investigates the effect of child neglect transmitted from maternal and paternal grandparents on the mental health status of grandchildren. The dependent variable is the mental health status of G3, and the independent variables include G2 child neglect and G1 child neglect by maternal and paternal grandparents. The table reports the coefficients for the OLS (columns (1)) and the marginal effects for the probit model (columns (2)). The results show that the child neglect experienced by G2 has a significant positive effect on the mental health problems of G3, regardless of whether the OLS or probit model is used. In contrast, the child neglect experienced by maternal and paternal grandparents does not have a significant effect on the mental health status of G3, as the coefficients are close to zero and not statistically significant. Additionally, the table includes control variables such as gender, year of birth, marital status, household income, and household economic status for G2 and G1. The table also includes interaction terms between the child neglect experienced by G2 and that experienced by G1. The interaction term between maternal G1 child neglect and G2 child neglect has a significant positive effect on the mental health problems of G3, while the interaction term between paternal G1 child neglect and G2 child neglect is not significant. In general, the results of the OLS and probit models are quite similar, with both models indicating that G2 child neglect has a significant impact on the mental health status of G3 children.

TABLE A.4—DETAILED RESULT OF THE INTERGENERATIONAL TRANSMISSION OF CHILD NEGLECT AND THE RISK OF MENTAL HEALTH PROBLEMS: EXPLORING THE EFFECT OF NEGLECT FROM MATERNAL AND PATERNAL GRANDPARENTS ON GRANDCHILDREN.

	Dependent variable: *Mental health status of G3*	
	OLS	Probit (Marginal effects)
Variables	(2)	(4)
G2 Child-neglect	0.199**	0.201**
	(0.083)	(0.084)
Paternal G1 Child-neglect	-0.052	-0.057
	(0.065)	(0.068)
Maternal G1 Child-neglect	0.042	0.040
	(0.072)	(0.075)
G3 Gender	-0.019	-0.021
	(0.026)	(0.026)
G3 Year of birth	-0.00001	0.000
	(0.003)	(0.003)
G3 Marital status	-0.117***	-0.117***
	(0.039)	(0.039)
G3 Household income	-0.009	-0.009
	(0.028)	(0.028)
G2 Household economic status	-0.035	-0.036
	(0.045)	(0.045)
G1 Household economic status	0.007	0.007
	(0.026)	(0.026)
Maternal G1 Child-neglect x G2 Child-neglect	0.379**	0.426*
	(0.180)	(0.222)
Paternal G1 Child-neglect x G2 Child-neglect	-0.059	-0.053
	(0.324)	(0.293)
Observations	1,258	1,258
R^2	0.027	
AIC		1,518.2

Note: Column (1) presents coefficients from OLS regressions, while column (2) presents marginal effects from probit regressions. Heteroskedasticity-robust standard errors are shown in parentheses for OLS models, while delta method standard errors are shown in parentheses for probit models. AIC is Akaike Information Criterion.

*** Significant at the 1% level.

** Significant at the 5% level.

* Significant at the 10% level.