

Intergenerational Transmission of Health and Behaviors

Module 1: Theory

Lecture 4

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Senior Researcher
Health & Social Sciences



08.11.2024



Presentation Outline

CHAPTER I

- 1 Introduction
- 2 Theoretical Foundations
- 3 Historical Case Studies

Q&A Session I

CHAPTER II

- 4 Multigenerational Transmission
- 5 Conceptual Framework – *Example from Norway*
- 6 Discussion and Future Direction

Q&A Session II

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Intergenerational Transmission

- Increasing body of research highlights the importance of the intergenerational transmission of health and health behaviors.
- Early life is significant in determining one's health in later years (Almond et al., 2018; Currie, 2009).
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Why Does Intergenerational Health Matter Today?

- **Understanding Health Beyond the Individual**

Health is not just about individual choices; it's shaped by what previous generations experienced.

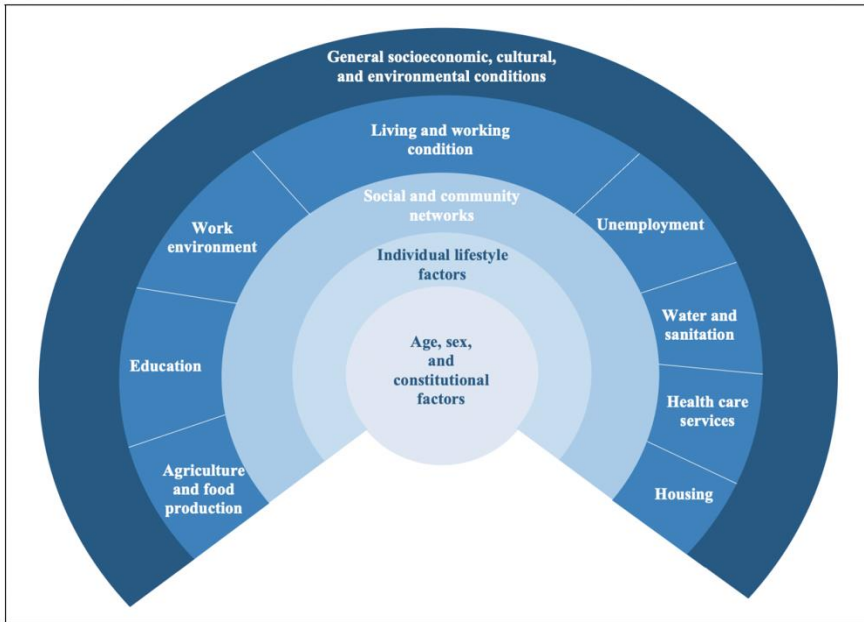
- **Relevance in Today's World**

Recent events like **COVID-19** and economic crises have highlighted how health risks and resources are unequally distributed across generations.

These challenges remind us that today's conditions can have long-term effects on future generations' health.

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Figure 1.1 "The main determinants of health"

Source: Acheson (1998).

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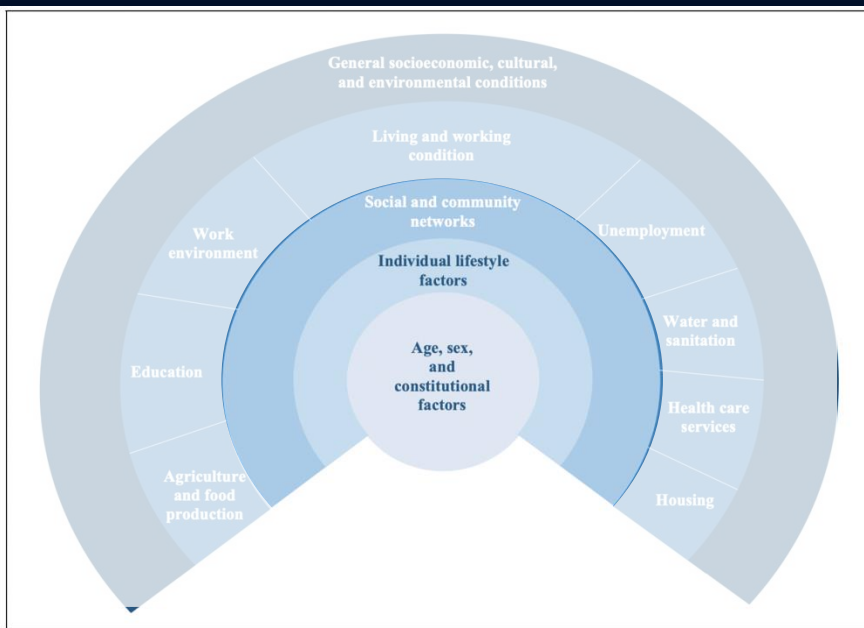


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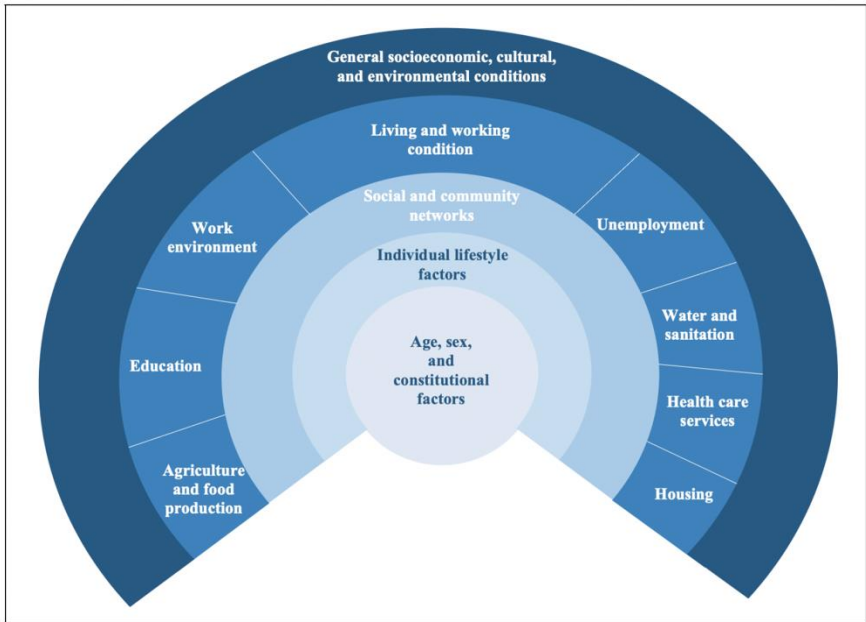
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Multigenerational Health Perspectives: The Role of Grandparents' Influence in Grandchildren's Wellbeing

Emre Sari^{1,2*}

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Keywords: grandparental investment, intergenerational health, public health policy, multigenerational transmission, demographic

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Why Study Intergenerational Transmission of Health?



Why Study Intergenerational Transmission of Health?

- **Long-Term Health Inequalities**

Health isn't just personal; it's shaped by experiences across generations.

- **Impact of Major Events**

Events like pandemics and economic crises affect not only those directly exposed but also their children and grandchildren.

- **Biological and Social Pathways**

Both genetic and social factors play roles in passing down health patterns.

- **Guiding Better Health Policies**

By understanding these patterns, we can create policies that benefit multiple generations.

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Nature vs. Nurture: What Shapes Health Across Generations?

Nature: Genetic Inheritance

Health traits, such as susceptibility to certain diseases, are inherited through genes. Genes set a foundation, but they interact with the environment in complex ways.

Nurture: Environmental and Social Influence

Family lifestyle, socioeconomic status, and early-life experiences shape health behaviors and risks.

Environmental factors like diet, stress, and access to resources can alter health trajectories.

Epigenetics: Where Nature Meets Nurture

Environmental factors can modify gene expression through mechanisms like DNA methylation, influencing health across generations without altering the genetic code. Epigenetics offers evidence of how “nurture” impacts “nature” in a way that can be inherited.

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the human genome

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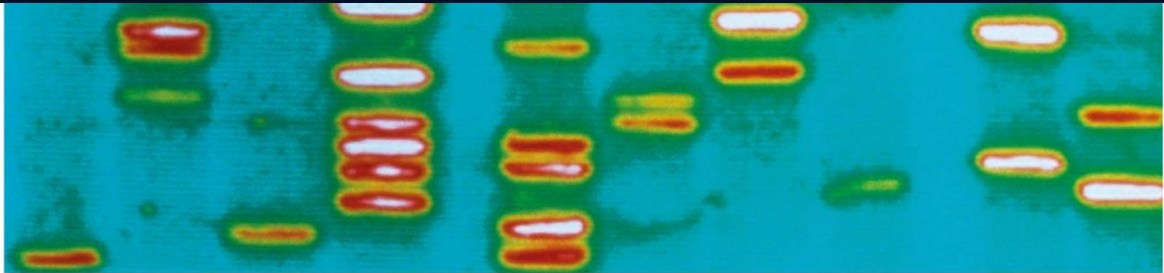
Vol. 291 No. 5507
Pages 1145-1434 \$9

THE HUMAN GENOME



AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE





PETER MENZEL/SPL

DNA sequencing has become routine, but the roles of individual genes can be hard to be pin.

Genes are not the blueprint for life

The view of biology often presented to the public is oversimplified and out of date. **By Denis Noble**

For too long, scientists have been content in espousing the lazy metaphor of living systems operating simply like machines, says science writer Philip Ball in *How Life Works*. Yet, it's important to be open about the complexity of biology – including what we don't know – because public understanding affects policy, health care and trust in science. “So long as we insist that cells are computers and genes are their code,” writes Ball, life might as well be “sprinkled

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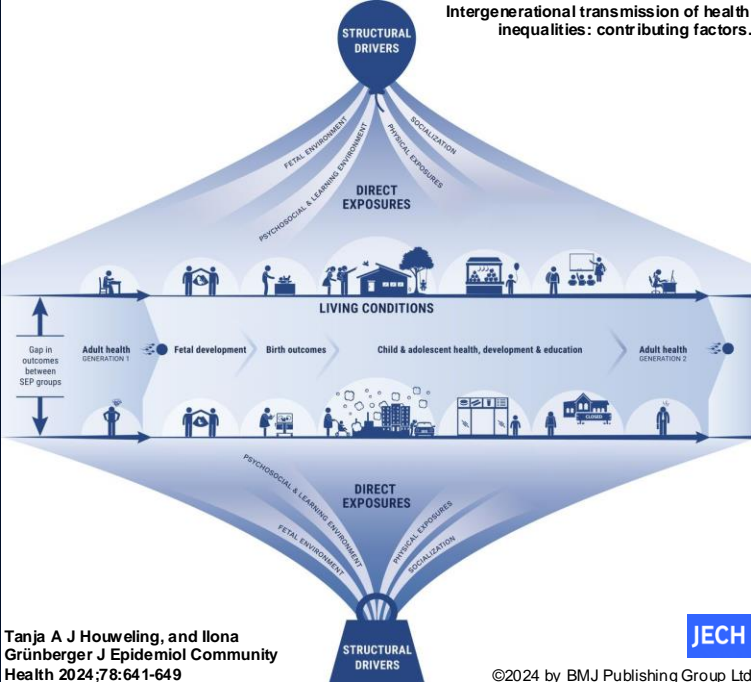
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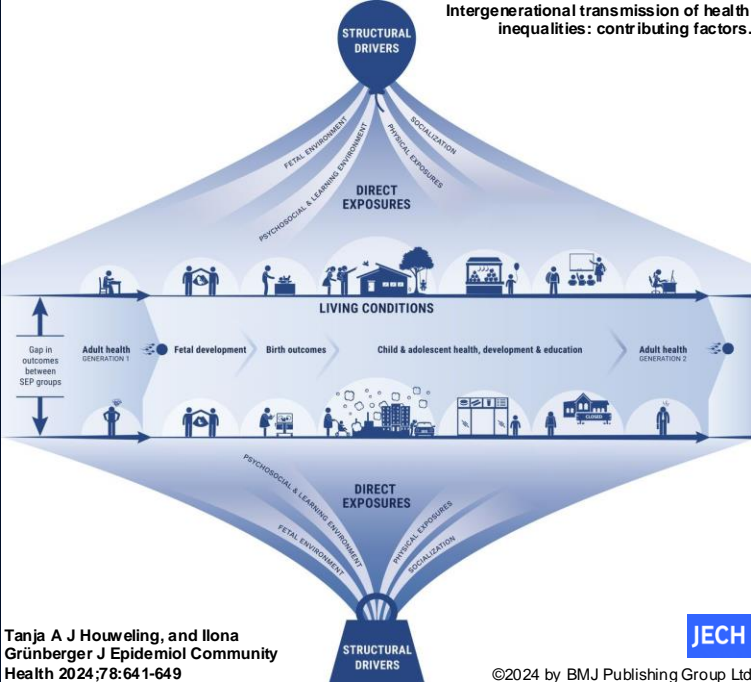
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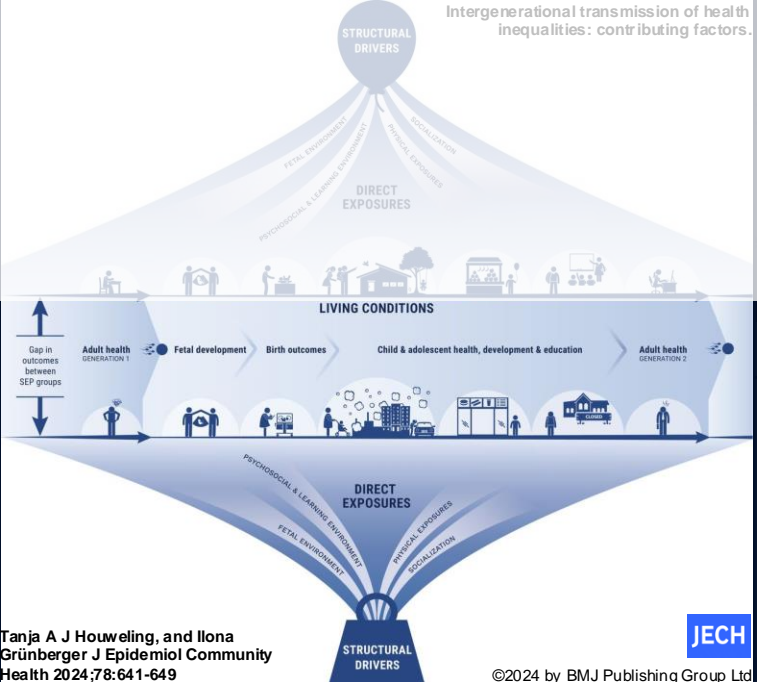
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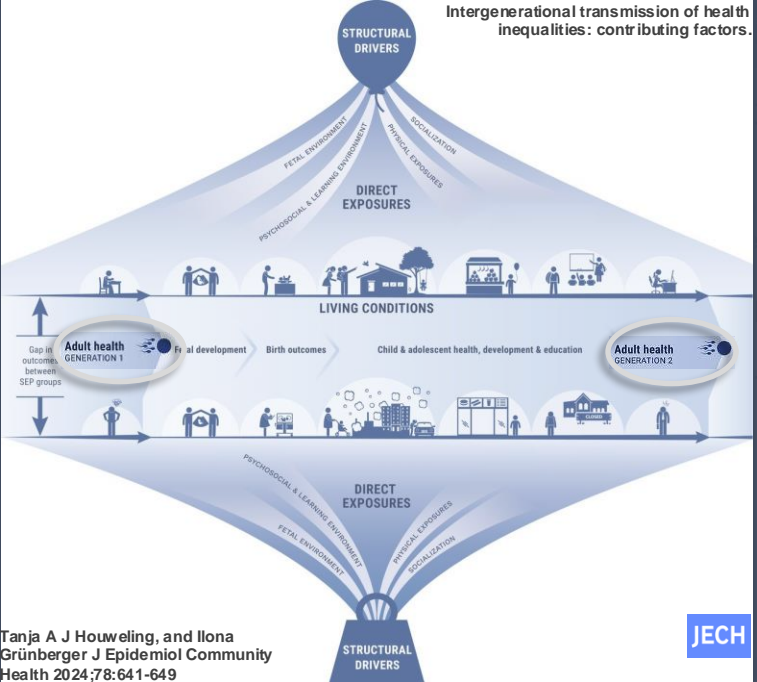
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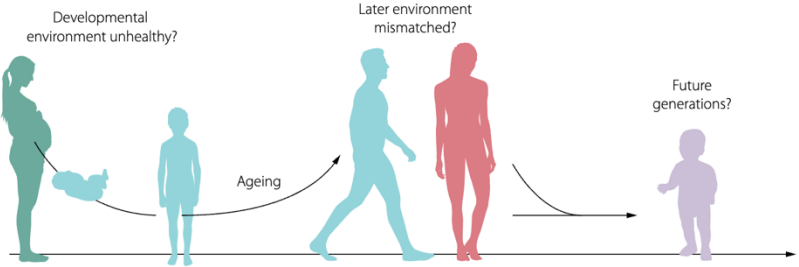
Developmental Epigenetic Processes and Transgenerational Passage of CVD Risk

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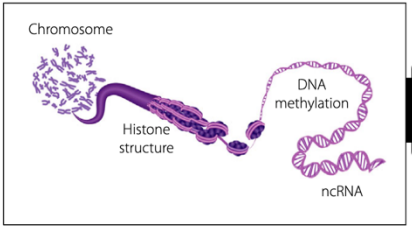
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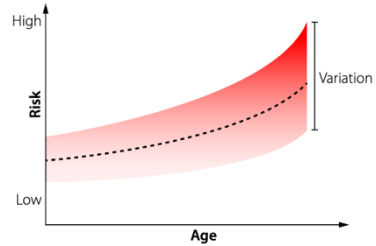
Hanson, M. (2019), The inheritance of cardiovascular disease risk. *Acta Paediatr*, 108: 1747-1756. <https://doi.org/10.1111/apa.14813>



Changes in gene expression affect phenotype

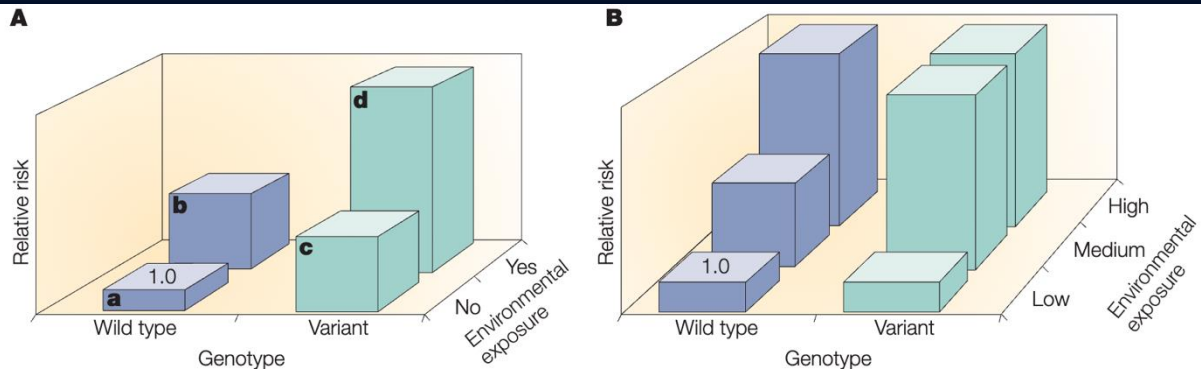


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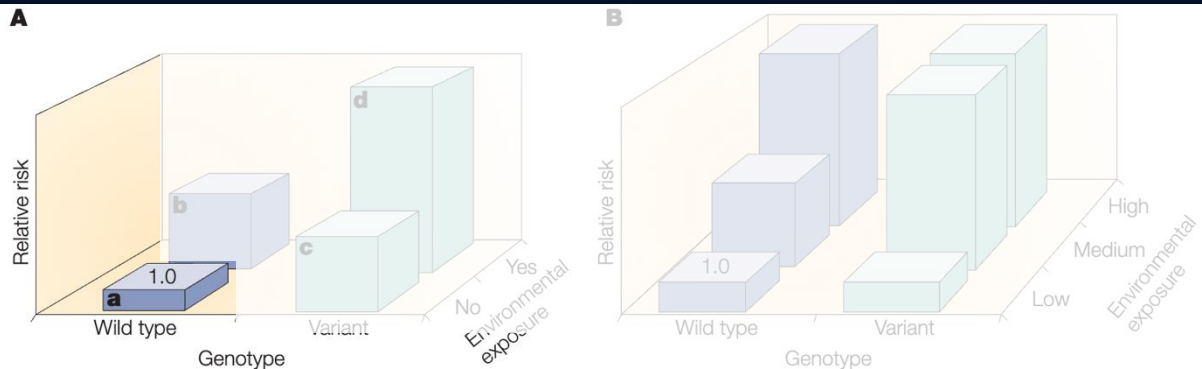
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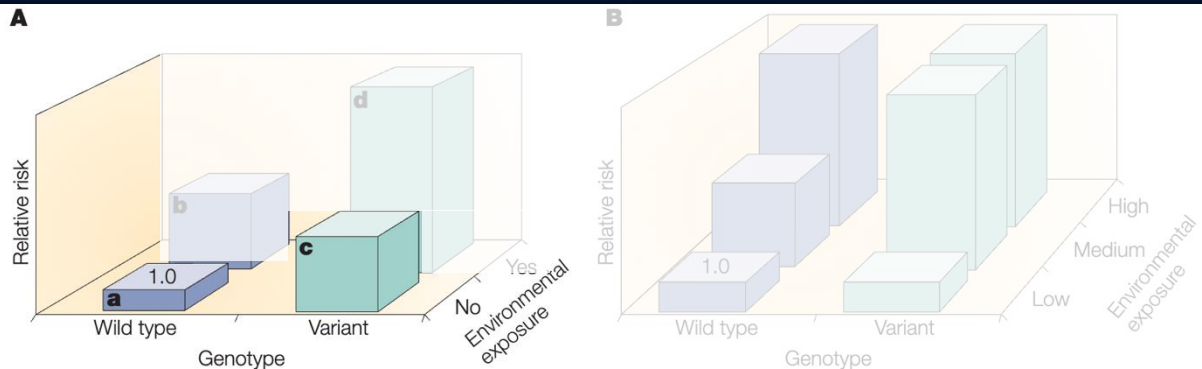
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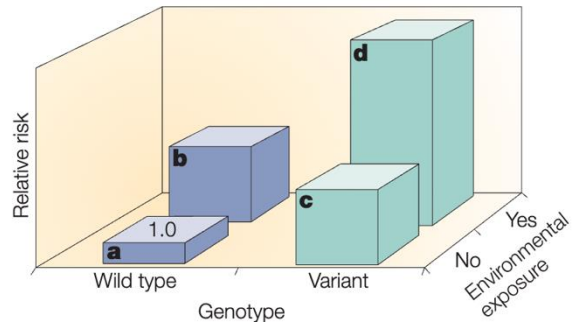


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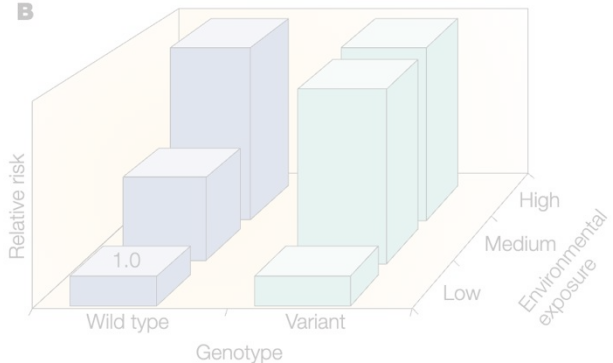
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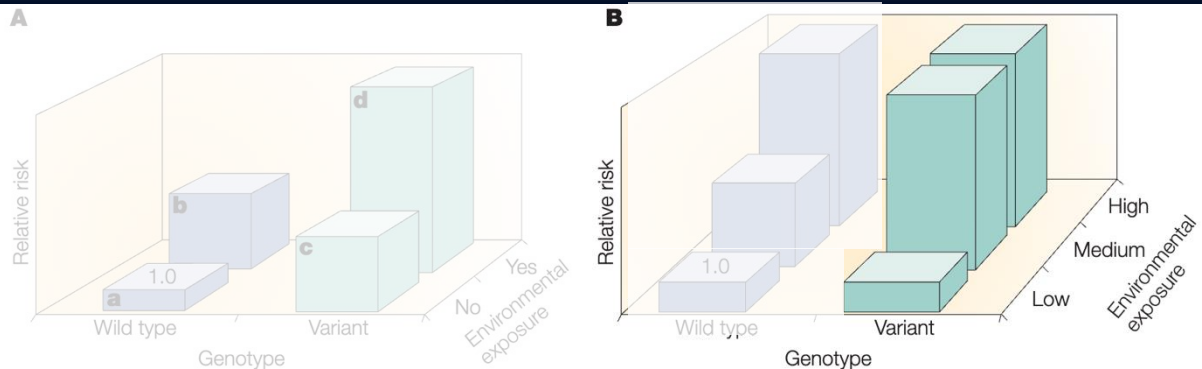
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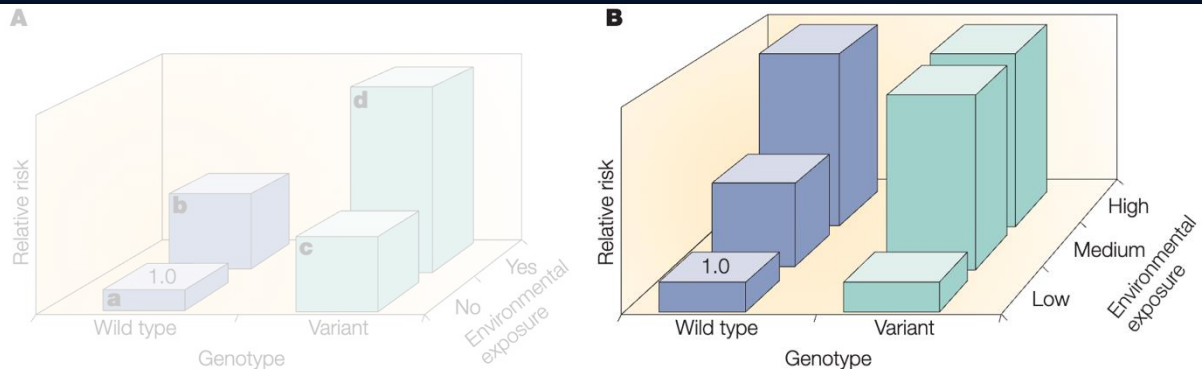
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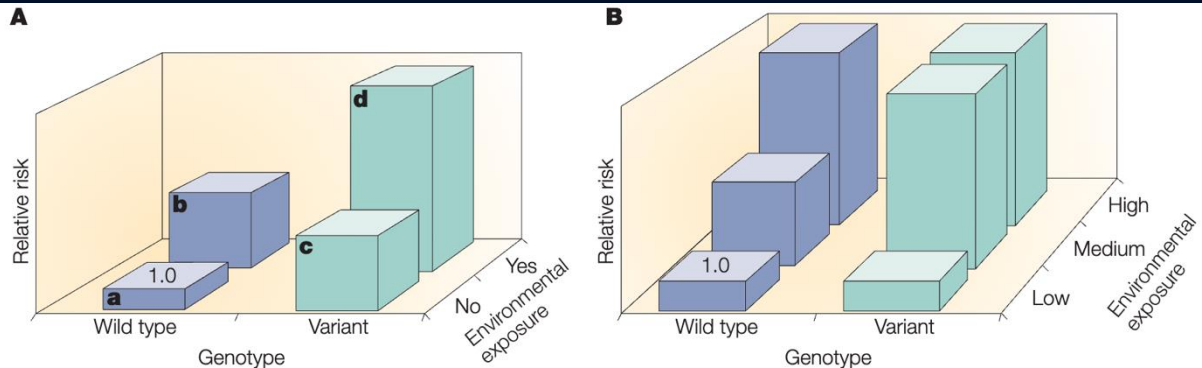
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Theoretical Foundations of Intergenerational Health Transmission



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1. Social and Cultural Transmission - Social Learning Theory

Behaviors, lifestyle choices, and cultural norms related to health are learned and passed down within families and communities.

Bandura, A. (1977). *Social Learning Theory*. Prentice Hall.

Example: Smoking habits or dietary preferences within families.



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Original article

The Role of Parental Engagement in the Intergenerational Transmission of Smoking Behavior and Identity



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^a Department of Health Behavior, Gillings School of Global Public Health, UNC-Chapel Hill, Chapel Hill, North Carolina

^b Center for Developmental Science, Chapel Hill, North Carolina

^c Department of Psychology and Neuroscience, UNC-Chapel Hill, Chapel Hill, North Carolina

Article history: Received May 17, 2016; Accepted November 7, 2016

Keywords: Smoking identity; Parental engagement; Social learning theory; Adolescent smoking

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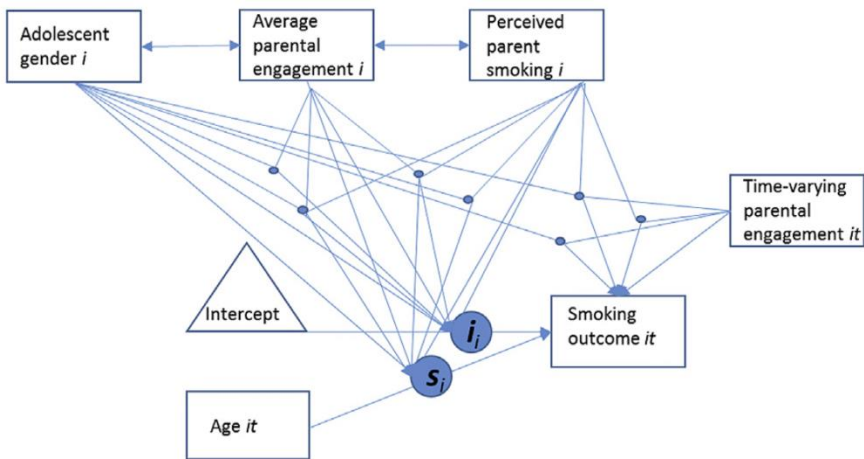


Figure 1. Path diagram of multilevel structural equation model estimated for full model (model 3) for one smoking outcome follows structure laid out by Curran and Bauer [31]. Time-invariant variables are denoted with i subscript and time-varying variables are denoted with it subscript. Large circles represent the random intercept and random effect of age. Dots connecting lines represent interaction effects. Covariate effects are not shown.

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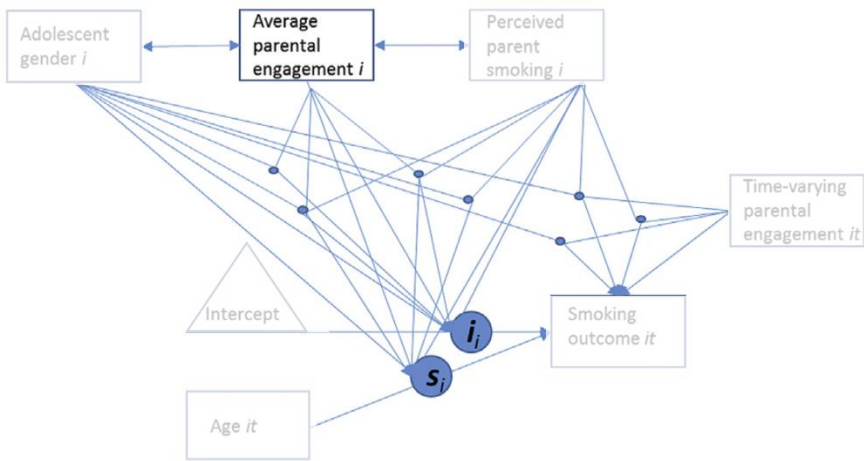


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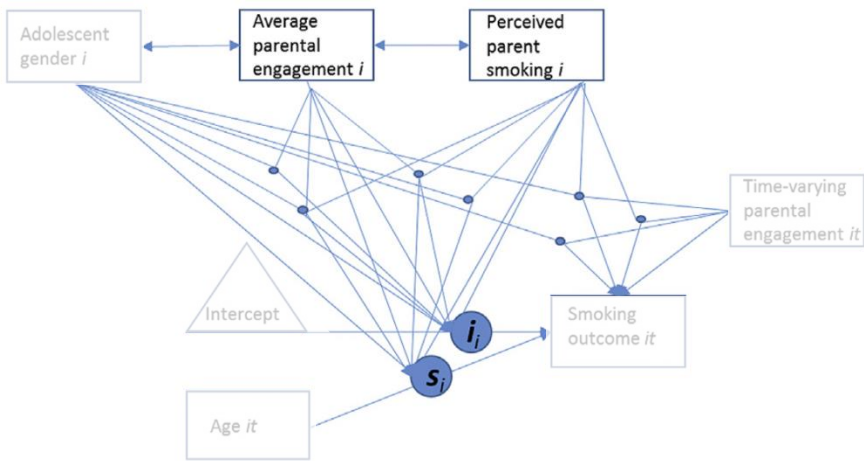


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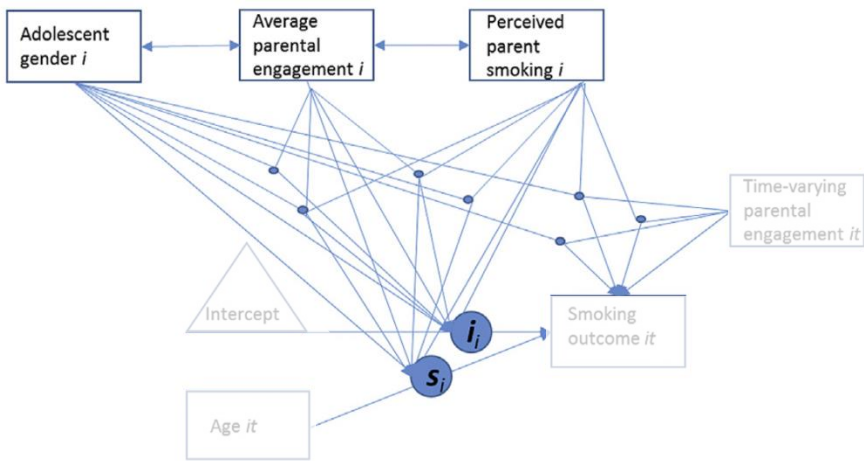


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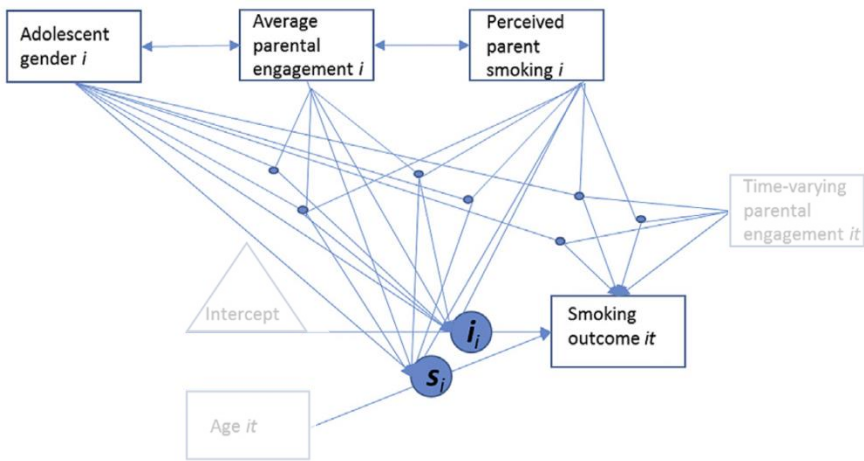


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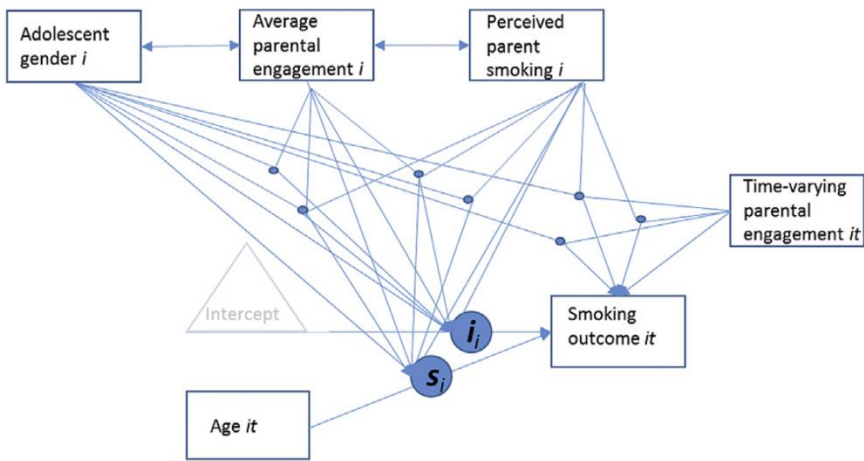


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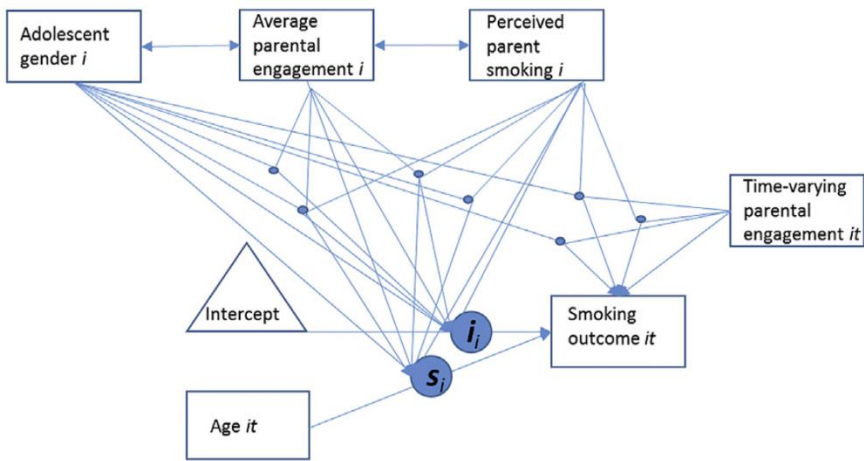


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Table 3

Standardized parameter estimates from models predicting adolescent smoking outcomes from paternal behavior

| Predictors | Past 3-month smoking | | | Smoking identity | | |
|-------------------------------------|----------------------|---------------|---------------|------------------|---------------|---------------|
| | Model 1 | Model 2 | Model 3 | Model 1 | Model 2 | Model 3 |
| Threshold | .67*** (.09) | .67*** (.09) | .68*** (.09) | .58*** (.07) | .64*** (.06) | .64*** (.06) |
| Age | .49*** (.06) | .48*** (.06) | .49*** (.06) | .54*** (.09) | .44*** (.05) | .44*** (.05) |
| Age ² | -.30*** (.03) | -.30*** (.03) | -.30*** (.03) | -.22*** (.02) | -.21*** (.02) | -.21*** (.02) |
| Single parent household | .08*** (.02) | .08*** (.02) | .08*** (.02) | .07*** (.02) | .07*** (.02) | .07*** (.02) |
| Parent education | -.05** (.02) | -.05** (.02) | -.05** (.02) | -.08*** (.02) | -.08*** (.02) | -.08*** (.02) |
| Black | -.14*** (.02) | -.14*** (.02) | -.14*** (.02) | -.09*** (.02) | -.09*** (.02) | -.09*** (.02) |
| Male | -.01 (.02) | -.01 (.02) | -.02 (.03) | .06*** (.02) | .05*** (.02) | .21*** (.02) |
| Parent smoke | .25*** (.02) | .25*** (.02) | .25*** (.02) | .20*** (.02) | .21*** (.02) | .21*** (.02) |
| TIC parent engagement | -.23*** (.02) | -.27*** (.03) | -.26*** (.03) | -.26*** (.02) | -.30*** (.03) | -.30*** (.03) |
| TVC parent engagement | -.10*** (.01) | -.08*** (.02) | -.08*** (.02) | -.08*** (.01) | -.09*** (.02) | -.09*** (.02) |
| Age × male | -.02 (.03) | -.02 (.03) | -.05 (.05) | .06 (.03) | .06 (.03) | .03 (.05) |
| Age × par smoke | .06 (.03) | .07* (.03) | .07* (.03) | .08** (.03) | .10*** (.03) | .10** (.03) |
| Age × TIC engage | .02 (.03) | -.06 (.05) | -.06 (.05) | .08* (.03) | .00 (.05) | .01 (.05) |
| TIC engage × par smoke | | .05 (.03) | .05 (.03) | | .05* (.02) | .06* (.02) |
| TVC engage × par smoke | | -.02 (.02) | -.02 (.02) | | .02 (.02) | .02 (.02) |
| Age × par smoke × TIC engage | | .09 (.05) | .08 (.05) | | .09 (.05) | .09 (.05) |
| Male × par smoke | | | .01 (.02) | | | -.03 (.02) |
| Male × TIC engage | | | .06* (.03) | | | .03 (.03) |
| Male × TVC engage | | | .00 (.02) | | | -.01 (.01) |
| Male × par smoke × TIC engage | | | .00 (.03) | | | -.02 (.02) |
| Male × par smoke × TVC engage | | | -.03 (.02) | | | .02 (.02) |
| Age × male × par smoke | | | .04 (.05) | | | .05 (.04) |
| Age × male × TIC engage | | | -.08 (.05) | | | -.05 (.05) |
| Age × male × par smoke × TIC engage | | | .04 (.05) | | | .14 (.08) |
| Intercept res variance | .82*** (.01) | .81*** (.01) | .81*** (.01) | .84*** (.01) | .84*** (.01) | .84*** (.01) |
| Age res variance | .96*** (.01) | .96*** (.01) | .95*** (.02) | .97*** (.01) | .97*** (.01) | .96*** (.01) |
| Residual correlation | .19** (.07) | .19** (.07) | .20** (.07) | .12 (.07) | .11 (.07) | .11 (.07) |

* $p < .01$; ** $p < .01$; *** $p < .001$.

TIC = time invariant covariate; TVC = time-varying covariate.

Table 3

Standardized parameter estimates from models predicting adolescent smoking outcomes from paternal behavior

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|-------------------------------------|----------------------|---------------|---------------|------------------|---------------|---------------|
| | Model 1 | Model 2 | Model 3 | Model 1 | Model 2 | Model 3 |
| Threshold | .67*** (.09) | .67*** (.09) | .68*** (.09) | .58*** (.07) | .64*** (.06) | .64*** (.06) |
| Age | .49*** (.06) | .48*** (.06) | .49*** (.06) | .54*** (.09) | .44*** (.05) | .44*** (.05) |
| Age ² | -.30*** (.03) | -.30*** (.03) | -.30*** (.03) | -.22*** (.02) | -.21*** (.02) | -.21*** (.02) |
| Single parent household | .08*** (.02) | .08*** (.02) | .08*** (.02) | .07*** (.02) | .07*** (.02) | .07*** (.02) |
| Parent education | -.05** (.02) | -.05** (.02) | -.05** (.02) | -.08*** (.02) | -.08*** (.02) | -.08*** (.02) |
| Black | -.14*** (.02) | -.14*** (.02) | -.14*** (.02) | -.09*** (.02) | -.09*** (.02) | -.09*** (.02) |
| Male | -.01 (.02) | -.01 (.02) | -.02 (.03) | .06*** (.02) | .05*** (.02) | .21*** (.02) |
| Parent smoke | .25*** (.02) | .25*** (.02) | .25*** (.02) | .20*** (.02) | .21*** (.02) | .21*** (.02) |
| TIC parent engagement | -.23*** (.02) | -.27*** (.03) | -.26*** (.03) | -.26*** (.02) | -.30*** (.03) | -.30*** (.03) |
| TVC parent engagement | -.10*** (.01) | -.08*** (.02) | -.08*** (.02) | -.08*** (.01) | -.09*** (.02) | -.09*** (.02) |
| Age × male | -.02 (.03) | -.02 (.03) | -.05 (.05) | .06 (.03) | .06 (.03) | .03 (.05) |
| Age × par smoke | .06 (.03) | .07* (.03) | .07* (.03) | .08** (.03) | .10*** (.03) | .10** (.03) |
| Age × TIC engage | .02 (.03) | -.06 (.05) | -.06 (.05) | .08* (.03) | .00 (.05) | .01 (.05) |
| TIC engage × par smoke | | .05 (.03) | .05 (.03) | | .05* (.02) | .06* (.02) |
| TVC engage × par smoke | | -.02 (.02) | -.02 (.02) | | .02 (.02) | .02 (.02) |
| Age × par smoke × TIC engage | | .09 (.05) | .08 (.05) | | .09 (.05) | .09 (.05) |
| Male × par smoke | | | .01 (.02) | | | -.03 (.02) |
| Male × TIC engage | | | .06* (.03) | | | .03 (.03) |
| Male × TVC engage | | | .00 (.02) | | | -.01 (.01) |
| Male × par smoke × TIC engage | | | .00 (.03) | | | -.02 (.02) |
| Male × par smoke × TVC engage | | | -.03 (.02) | | | .02 (.02) |
| Age × male × par smoke | | | .04 (.05) | | | .05 (.04) |
| Age × male × TIC engage | | | -.08 (.05) | | | -.05 (.05) |
| Age × male × par smoke × TIC engage | | | .04 (.05) | | | .14 (.08) |
| Intercept res variance | .82*** (.01) | .81*** (.01) | .81*** (.01) | .84*** (.01) | .84*** (.01) | .84*** (.01) |
| Age res variance | .96*** (.01) | .96*** (.01) | .95*** (.02) | .97*** (.01) | .97*** (.01) | .96*** (.01) |
| Residual correlation | .19** (.07) | .19** (.07) | .20** (.07) | .12 (.07) | .11 (.07) | .11 (.07) |

* $p < .01$; ** $p < .01$; *** $p < .001$.

TIC = time invariant covariate; TVC = time-varying covariate.

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| Age | .49*** (.06) | .48*** (.06) | .49*** (.06) | .54*** (.09) | .44*** (.05) | .44*** (.05) |
| Age ² | -.30*** (.03) | -.30*** (.03) | -.30*** (.03) | -.22*** (.02) | -.21*** (.02) | -.21*** (.02) |
| Single parent household | .08*** (.02) | .08*** (.02) | .08*** (.02) | .07*** (.02) | .07*** (.02) | .07*** (.02) |
| Parent education | -.05** (.02) | -.05** (.02) | -.05** (.02) | -.08*** (.02) | -.08*** (.02) | -.08*** (.02) |
| Black | -.14*** (.02) | -.14*** (.02) | -.14*** (.02) | -.09*** (.02) | -.09*** (.02) | -.09*** (.02) |
| Male | -.01 (.02) | -.01 (.02) | -.02 (.03) | .06*** (.02) | .05*** (.02) | .21*** (.02) |
| Parent smoke | .25*** (.02) | .25*** (.02) | .25*** (.02) | .20*** (.02) | .21*** (.02) | .21*** (.02) |
| TIC parent engagement | -.23*** (.02) | -.27*** (.03) | -.26*** (.03) | -.26*** (.02) | -.30*** (.03) | -.30*** (.03) |
| TVC parent engagement | -.10*** (.01) | -.08*** (.02) | -.08*** (.02) | -.08*** (.01) | -.09*** (.02) | -.09*** (.02) |
| Age × male | -.02 (.03) | -.02 (.03) | -.05 (.05) | .06 (.03) | .06 (.03) | .03 (.05) |
| Age × par smoke | .06 (.03) | .07* (.03) | .07* (.03) | .08** (.03) | .10*** (.03) | .10** (.03) |
| Age × TIC engage | .02 (.03) | -.06 (.05) | -.06 (.05) | .08* (.03) | .00 (.05) | .01 (.05) |
| TIC engage × par smoke | | .05 (.03) | .05 (.03) | | .05* (.02) | .06* (.02) |
| TVC engage × par smoke | | -.02 (.02) | -.02 (.02) | | .02 (.02) | .02 (.02) |
| Age × par smoke × TIC engage | | .09 (.05) | .08 (.05) | | .09 (.05) | .09 (.05) |
| Male × par smoke | | | .01 (.02) | | | -.03 (.02) |
| Male × TIC engage | | | .06* (.03) | | | .03 (.03) |
| Male × TVC engage | | | .00 (.02) | | | -.01 (.01) |
| Male × par smoke × TIC engage | | | .00 (.03) | | | -.02 (.02) |
| Male × par smoke × TVC engage | | | -.03 (.02) | | | .02 (.02) |
| Age × male × par smoke | | | .04 (.05) | | | .05 (.04) |
| Age × male × TIC engage | | | -.08 (.05) | | | -.05 (.05) |
| Age × male × par smoke × TIC engage | | | .04 (.05) | | | .14 (.08) |
| Intercept res variance | .82*** (.01) | .81*** (.01) | .81*** (.01) | .84*** (.01) | .84*** (.01) | .84*** (.01) |
| Age res variance | .96*** (.01) | .96*** (.01) | .95*** (.02) | .97*** (.01) | .97*** (.01) | .96*** (.01) |
| Residual correlation | .19** (.07) | .19** (.07) | .20** (.07) | .12 (.07) | .11 (.07) | .11 (.07) |

* $p < .01$; ** $p < .01$; *** $p < .001$.

TIC = time invariant covariate; TVC = time-varying covariate.

Table 3

Standardized parameter estimates from models predicting adolescent smoking outcomes from paternal behavior

| Predictors | Past 3-month smoking | | | Smoking identity | | |
|-------------------------------------|----------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| | Model 1 | Model 2 | Model 3 | Model 1 | Model 2 | Model 3 |
| Threshold | .67*** (.09) | .67*** (.09) | .68*** (.09) | .58*** (.07) | .64*** (.06) | .64*** (.06) |
| Age | .49*** (.06) | .48*** (.06) | .49*** (.06) | .54*** (.09) | .44*** (.05) | .44*** (.05) |
| Age ² | -.30*** (.03) | -.30*** (.03) | -.30*** (.03) | -.22*** (.02) | -.21*** (.02) | -.21*** (.02) |
| Single parent household | .08*** (.02) | .08*** (.02) | .08*** (.02) | .07*** (.02) | .07*** (.02) | .07*** (.02) |
| Parent education | -.05** (.02) | -.05** (.02) | -.05** (.02) | -.08*** (.02) | -.08*** (.02) | -.08*** (.02) |
| Black | -.14*** (.02) | -.14*** (.02) | -.14*** (.02) | -.09*** (.02) | -.09*** (.02) | -.09*** (.02) |
| Male | -.01 (.02) | -.01 (.02) | -.02 (.03) | .06*** (.02) | .05*** (.02) | .21*** (.02) |
| Parent smoke | .25*** (.02) | .25*** (.02) | .25*** (.02) | .20*** (.02) | .21*** (.02) | .21*** (.02) |
| TIC parent engagement | -.23*** (.02) | -.27*** (.03) | -.26*** (.03) | -.26*** (.02) | -.30*** (.03) | -.30*** (.03) |
| TVC parent engagement | -.10*** (.01) | -.08*** (.02) | -.08*** (.02) | -.08*** (.01) | -.09*** (.02) | -.09*** (.02) |
| Age × male | -.02 (.03) | -.02 (.03) | -.05 (.05) | .06 (.03) | .06 (.03) | .03 (.05) |
| Age × par smoke | .06 (.03) | .07* (.03) | .07* (.03) | .08** (.03) | .10*** (.03) | .10** (.03) |
| Age × TIC engage | .02 (.03) | -.06 (.05) | -.06 (.05) | .08* (.03) | .00 (.05) | .01 (.05) |
| TIC engage × par smoke | | .05 (.03) | .05 (.03) | | .05* (.02) | .06* (.02) |
| TVC engage × par smoke | | -.02 (.02) | -.02 (.02) | | .02 (.02) | .02 (.02) |
| Age × par smoke × TIC engage | | .09 (.05) | .08 (.05) | | .09 (.05) | .09 (.05) |
| Male × par smoke | | | .01 (.02) | | | -.03 (.02) |
| Male × TIC engage | | | .06* (.03) | | | .03 (.03) |
| Male × TVC engage | | | .00 (.02) | | | -.01 (.01) |
| Male × par smoke × TIC engage | | | .00 (.03) | | | -.02 (.02) |
| Male × par smoke × TVC engage | | | -.03 (.02) | | | .02 (.02) |
| Age × male × par smoke | | | .04 (.05) | | | .05 (.04) |
| Age × male × TIC engage | | | -.08 (.05) | | | -.05 (.05) |
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| Intercept res variance | .82*** (.01) | .81*** (.01) | .81*** (.01) | .84*** (.01) | .84*** (.01) | .84*** (.01) |
| Age res variance | .96*** (.01) | .96*** (.01) | .95*** (.02) | .97*** (.01) | .97*** (.01) | .96*** (.01) |
| Residual correlation | .19** (.07) | .19** (.07) | .20** (.07) | .12 (.07) | .11 (.07) | .11 (.07) |

* $p < .01$; ** $p < .01$; *** $p < .001$.

TIC = time invariant covariate; TVC = time-varying covariate.

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| Age ² | -.30*** (.03) | -.30*** (.03) | -.30*** (.03) | -.22*** (.02) | -.21*** (.02) | -.21*** (.02) |
| Single parent household | .08*** (.02) | .08*** (.02) | .08*** (.02) | .07*** (.02) | .07*** (.02) | .07*** (.02) |
| Parent education | -.05** (.02) | -.05** (.02) | -.05** (.02) | -.08*** (.02) | -.08*** (.02) | -.08*** (.02) |
| Black | -.14*** (.02) | -.14*** (.02) | -.14*** (.02) | -.09*** (.02) | -.09*** (.02) | -.09*** (.02) |
| Male | -.01 (.02) | -.01 (.02) | -.02 (.03) | .06*** (.02) | .05*** (.02) | .21*** (.02) |
| Parent smoke | .25*** (.02) | .25*** (.02) | .25*** (.02) | .20*** (.02) | .21*** (.02) | .21*** (.02) |
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| Age × male | -.02 (.03) | -.02 (.03) | -.05 (.05) | .06 (.03) | .06 (.03) | .03 (.05) |
| Age × par smoke | .06 (.03) | .07* (.03) | .07* (.03) | .08** (.03) | .10*** (.03) | .10** (.03) |
| Age × TIC engage | .02 (.03) | -.06 (.05) | -.06 (.05) | .08* (.03) | .00 (.05) | .01 (.05) |
| TIC engage × par smoke | | .05 (.03) | .05 (.03) | | .05* (.02) | .06* (.02) |
| TVC engage × par smoke | | -.02 (.02) | -.02 (.02) | | .02 (.02) | .02 (.02) |
| Age × par smoke × TIC engage | | .09 (.05) | .08 (.05) | | .09 (.05) | .09 (.05) |
| Male × par smoke | | | .01 (.02) | | | -.03 (.02) |
| Male × TIC engage | | | .06* (.03) | | | .03 (.03) |
| Male × TVC engage | | | .00 (.02) | | | -.01 (.01) |
| Male × par smoke × TIC engage | | | .00 (.03) | | | -.02 (.02) |
| Male × par smoke × TVC engage | | | -.03 (.02) | | | .02 (.02) |
| Age × male × par smoke | | | .04 (.05) | | | .05 (.04) |
| Age × male × TIC engage | | | -.08 (.05) | | | -.05 (.05) |
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| Intercept res variance | .82*** (.01) | .81*** (.01) | .81*** (.01) | .84*** (.01) | .84*** (.01) | .84*** (.01) |
| Age res variance | .96*** (.01) | .96*** (.01) | .95*** (.02) | .97*** (.01) | .97*** (.01) | .96*** (.01) |
| Residual correlation | .19** (.07) | .19** (.07) | .20** (.07) | .12 (.07) | .11 (.07) | .11 (.07) |

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| Age ² | -.30*** (.03) | -.30*** (.03) | -.30*** (.03) | -.22*** (.02) | -.21*** (.02) | -.21*** (.02) |
| Single parent household | .08*** (.02) | .08*** (.02) | .08*** (.02) | .07*** (.02) | .07*** (.02) | .07*** (.02) |
| Parent education | -.05** (.02) | -.05** (.02) | -.05** (.02) | -.08*** (.02) | -.08*** (.02) | -.08*** (.02) |
| Black | -.14*** (.02) | -.14*** (.02) | -.14*** (.02) | -.09*** (.02) | -.09*** (.02) | -.09*** (.02) |
| Male | -.01 (.02) | -.01 (.02) | -.02 (.03) | .06*** (.02) | .05*** (.02) | .21*** (.02) |
| Parent smoke | .25*** (.02) | .25*** (.02) | .25*** (.02) | .20*** (.02) | .21*** (.02) | .21*** (.02) |
| TIC parent engagement | -.23*** (.02) | -.27*** (.03) | -.26*** (.03) | -.26*** (.02) | -.30*** (.03) | -.30*** (.03) |
| TVC parent engagement | -.10*** (.01) | -.08*** (.02) | -.08*** (.02) | -.08*** (.01) | -.09*** (.02) | -.09*** (.02) |
| Age × male | -.02 (.03) | -.02 (.03) | -.05 (.05) | .06 (.03) | .06 (.03) | .03 (.05) |
| Age × par smoke | .06 (.03) | .07* (.03) | .07* (.03) | .08** (.03) | .10*** (.03) | .10** (.03) |
| Age × TIC engage | .02 (.03) | -.06 (.05) | -.06 (.05) | .08* (.03) | .00 (.05) | .01 (.05) |
| TIC engage × par smoke | | .05 (.03) | .05 (.03) | | .05* (.02) | .06* (.02) |
| TVC engage × par smoke | | -.02 (.02) | -.02 (.02) | | .02 (.02) | .02 (.02) |
| Age × par smoke × TIC engage | | .09 (.05) | .08 (.05) | | .09 (.05) | .09 (.05) |
| Male × par smoke | | | .01 (.02) | | | -.03 (.02) |
| Male × TIC engage | | | .06* (.03) | | | .03 (.03) |
| Male × TVC engage | | | .00 (.02) | | | -.01 (.01) |
| Male × par smoke × TIC engage | | | .00 (.03) | | | -.02 (.02) |
| Male × par smoke × TVC engage | | | -.03 (.02) | | | .02 (.02) |
| Age × male × par smoke | | | .04 (.05) | | | .05 (.04) |
| Age × male × TIC engage | | | -.08 (.05) | | | -.05 (.05) |
| Age × male × par smoke × TIC engage | | | .04 (.05) | | | .14 (.08) |
| Intercept res variance | .82*** (.01) | .81*** (.01) | .81*** (.01) | .84*** (.01) | .84*** (.01) | .84*** (.01) |
| Age res variance | .96*** (.01) | .96*** (.01) | .95*** (.02) | .97*** (.01) | .97*** (.01) | .96*** (.01) |
| Residual correlation | .19** (.07) | .19** (.07) | .20** (.07) | .12 (.07) | .11 (.07) | .11 (.07) |

* $p < .01$; ** $p < .01$; *** $p < .001$.

TIC = time invariant covariate; TVC = time-varying covariate.

Theoretical Foundations of Intergenerational Health Transmission

2. Health Belief Model

Concept: Beliefs about health risks and benefits influence behaviors; perceptions are shaped by family and community.

Example: Family beliefs about preventive health can influence vaccination and health check-up behaviors.

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JOURNAL ARTICLE

Social Learning Theory and the Health Belief Model

Irwin M. Rosenstock, Victor J. Strecher and Marshall H. Becker

Health Education Quarterly

Vol. 15, No. 2 (Summer 1988), pp. 175-183 (9 pages)

Published By: Sage Publications, Inc.



Theoretical Foundations of Intergenerational Health Transmission

3. Attachment Theory

Concept: Early attachment relationships influence emotional regulation, stress response, and later health behaviors.

Example: Secure attachments in childhood are linked to better coping strategies and health outcomes in adulthood.

Bowlby, J. (1969). *Attachment and Loss*.

Theoretical Foundations of Intergenerational Health Transmission

4. Life Course Theory

Early-life experiences and exposures, such as childhood nutrition, stress, or socioeconomic conditions, influence health outcomes across the lifespan and can affect future generations.

Example: The Barker Hypothesis, which suggests that poor fetal and infant health can lead to increased risk of chronic diseases in adulthood.

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Review

A life course approach to reproductive health: Theory and methods

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ABSTRACT

Taking a life course approach to the study of reproductive health involves the investigation of factors across life and, also **across generations**, that influence the timing of menarche, fertility, pregnancy outcomes, gynaecological disorders, and age at menopause. It also recognises the important influence of reproductive health on chronic disease risk in later life. Published literature supports the use of an integrated life course approach to study reproductive health, which examines the whole life course, considers the continuity of reproductive health and the interrelationship between the different markers of this. This is in contrast to more traditional approaches that tend to focus only on contemporary risk factors and which consider each marker of reproductive health separately. For instance, we found evidence linking early life factors such as growth, socioeconomic conditions, and parental divorce with ages at menarche and menopause, although the nature of the relationship differs. We discuss the different theoretical mod-

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Theoretical Foundations of Intergenerational Health Transmission

The Barker Hypothesis – Developmental Origins of Health and Disease (DOHaD) – Fetal Origins Hypothesis

The fetal and infant origins of adult disease

The womb may be more important than the home

A hundred years ago, when tuberculosis and rheumatic heart disease were common, the proposition that the childhood environment affects adult health would have been self evident. This proposition may still hold, even though infective disease has given place to degenerative disease.

Studies in Norway, Finland, Britain, and the United States have shown that death rates from cardiovascular disease are inversely related to adult height, and geographical differences in cardiovascular mortality are related to past differences in infant mortality.^{1,2} These findings have been interpreted as evidence that adverse living conditions during childhood, such as poor housing and diet, increase the risk of ischaemic heart disease.³ Case-control studies have generally supported this^{4,5}; patients with myocardial infarction have higher infant death rates among their siblings,^{6,7} are more likely to come from larger families, and are more likely to have fathers who were unemployed.⁸ Now studies in Finland show that men with ischaemic heart disease had worse socioeconomic conditions in childhood (p 1121)⁹—an observation also made in Britain.¹⁰

The completeness of infant mortality records in England and Wales from 1911 onwards has allowed detailed geographical comparisons of the relation between infant mortality 70 years ago and mortality from cardiovascular disease today. Differences in the death rates from cardiovascular disease among the 212 local authority areas of England and Wales are closely related to past differences in neonatal mortality.¹¹ Most neonatal deaths were associated with low birth weight, and rates were high in areas where mothers had poor health and high death rates during childbirth.^{12,13} These findings suggested that research should be redirected towards the intrauterine environment rather than the environment in later childhood—housing, family income, diet, and other influences. The Medical Research Council employed a historian to search for old records of birth

rate programming is beginning to emerge.¹⁴ A recent symposium heard evidence that diseases other than cardiovascular disease may also be determined by the maternal environment.¹⁵ Schizophrenia and obstructive lung disease are two examples.

The old model of adult degenerative disease was based on the interaction between genes and an adverse environment in adult life. The new model that is developing will include programming by the environment in fetal and infant life.

D J P BARKER

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Killing Me Softly: The Fetal Origins Hypothesis

Douglas Almond and Janet Currie

Historical Context of Intergenerational Health Transmission



Historical Context of Intergenerational Health Transmission

Dutch Famine of 1944-45 (The "Hunger Winter")

Impact: Individuals exposed to famine in utero showed higher risks of obesity, diabetes, and cardiovascular disease in adulthood.

Intergenerational Findings: Children and even grandchildren of those affected showed increased health risks, suggesting lasting epigenetic changes.



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Long-run effects on longevity of a nutritional shock early in life: The Dutch Potato famine of 1846–1847

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^a VU University Amsterdam, HEB, Tinbergen Institute, IZA, Netspar, The Netherlands

^b VU University Amsterdam, Tinbergen Institute, The Netherlands

^c University of Mannheim, VU University Amsterdam, IFAU-Uppsala, CEPR, IZA, IFS, Netspar, The Netherlands

2. Overview of the Dutch mortality trends and agricultural sector in the 19th century

At the beginning of the 19th century, the Netherlands witnessed relatively high infant mortality compared to the rest of Europe. The rates only began to decrease after the 1870s, with a sharp fall until the Second World War. The conditions of the water and the breastfeeding practices explain to a large extent the high (infant) mortality ([Wintle, 2000](#)). We return to that below. The drop in mortality rates after 1870 is mainly related to the increase in the availability of better food, the improved medicine and health care, and the improved public health environment ([Wintle, 2000](#)). All these developments were linked to a more sustained growth after 1870. See for instance [Van Zanden and van Riel \(2004\)](#) for a detailed discussion of the Dutch agricultural sector in the 19th century.

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Reference: He with prenatal ex

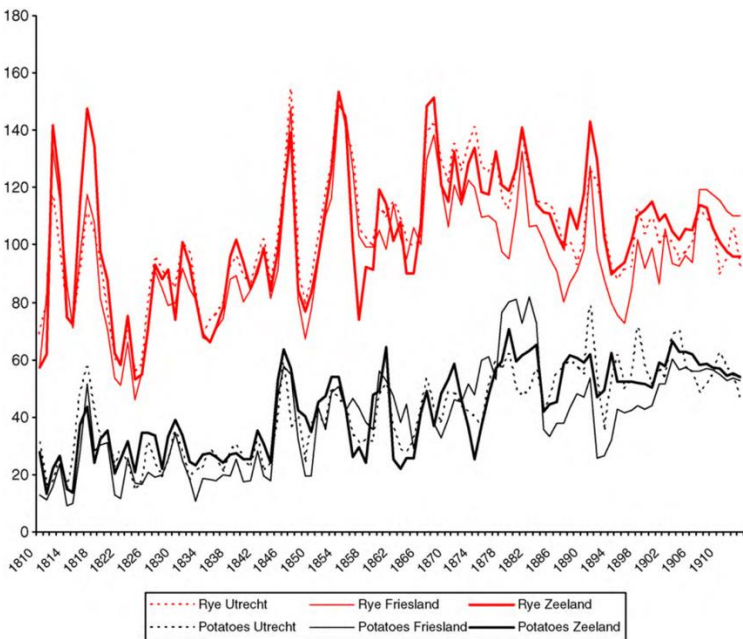


Fig. 1. Yearly real market prices per province.

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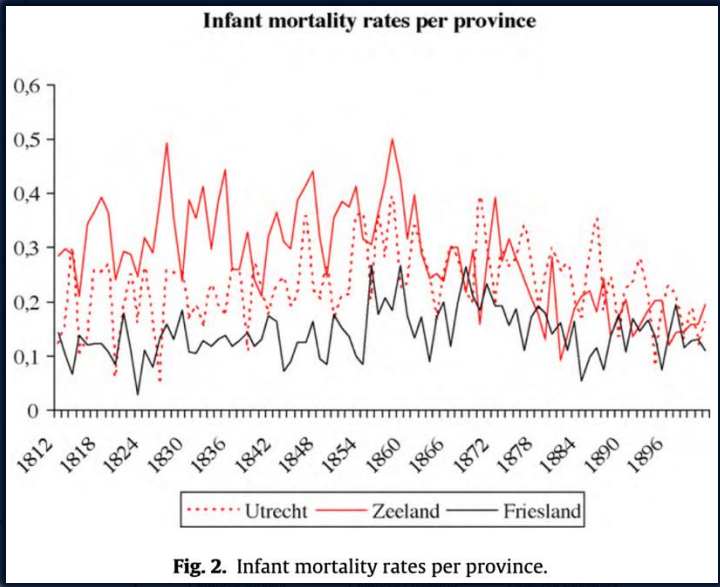


Fig. 2. Infant mortality rates per province.

Fig. 1. Yearly real market prices per province.

Table 2
(Residual) life expectancies of cohorts exposed or not exposed to the famine^a.

| Date of birth | Total life expectancy (#) | | Residual life expectancy | | | |
|---|---------------------------|-------------|--------------------------|------------------------|------------------------|------------------------|
| | | | At age 1 (#) | At age 20 (#) | At age 40 (#) | At age 50 (#) |
| <i>Males</i> | | | | | | |
| 1/9/1846–1/12/1847 (born or in gestation for at least 6 months during famine) | 29.3 (47) | | 38.1 (35) | 37.4 (22) | 26.3 (17) | 17.4 (16) |
| <i>Controls</i> | | | | | | |
| 1/9/1848–1/9/1855 (born after famine) | 30.9 (316) | 0.37 | 42.3 (225) 0.23 | 45.0 (142) 0.03 | 30.5 (124) 0.08 | 20.5 (114) 0.02 |
| 1/9/1837–1/9/1844 (born before famine) | 26.9 (306) | 0.68 | 38.5 (208) 0.48 | 38.3 (133) 0.42 | 28.8 (100) 0.24 | 18.8 (87) 0.04 |
| <i>Females</i> | | | | | | |
| 1/9/1846–1/12/1847 (born or in gestation for at least 6 months during famine) | 32.5 (41) | | 46.4 (28) | 44.3 (20) | 29.5 (17) | 20.7 (16) |
| <i>Controls</i> | | | | | | |
| 1/9/1848–1/9/1855 (born after famine) | 31.0 (270) | 0.60 | 43.4 (188) 0.67 | 46.1 (121) 0.34 | 32.5 (102) 0.15 | 22.5 (97) 0.10 |
| 1/9/1837–1/9/1844 (born before famine) | 34.2 (275) | 0.22 | 41.4 (221) 0.78 | 41.9 (145) 0.69 | 30.5 (114) 0.38 | 20.5 (104) 0.20 |

Figures into brackets are numbers of individuals per group and figures in bold are *p*-values of statistical tests for difference in means between individuals exposed to the Potato famine and controls.

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Historical Context of Intergenerational Health Transmission

1918 Influenza Pandemic

Impact: Children born to mothers infected during the pandemic had higher rates of physical and cognitive impairments.

Intergenerational Findings: Increased risk of heart disease and mental health issues observed in later generations, highlighting the role of prenatal stress.



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Does *in utero* exposure to illness matter? The 1918 influenza epidemic in Taiwan as a natural experiment[☆]



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^b University of Houston, United States

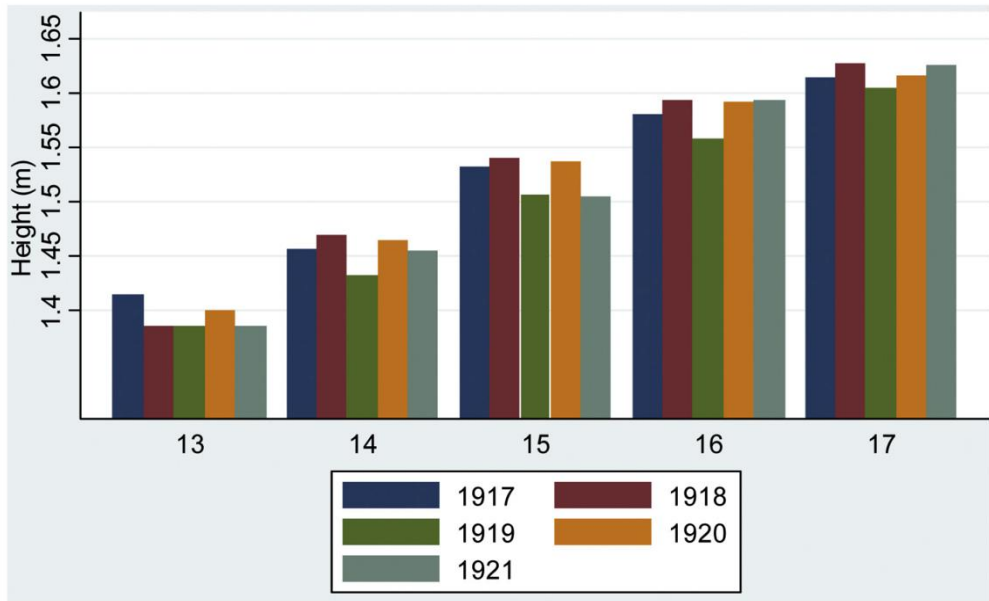


Fig. 4. Average height of male students. Age 13–17, born in 1917–1921.

Data Source: Taipei County's Statistical Books 1929–1938.

Historical Context of Intergenerational Health Transmission

Table 2
Effect of maternal mortality rate on height in the 1927 height report of school children.

| | Mean (cm) | Dependent variable | | | |
|--------|-----------|----------------------------------|-------------------------|--|--------------------------------------|
| | | (1) Height (cm) | (2) Height (z-score) | (3) Stunting (height \leq 5th percentile) | (4) (height \geq 75 percentile) |
| All | 119.5 | -4.029 [*] [0.072] | -1.524 [0.116] | 0.219 [0.212] | -0.390 ^{**} [0.039] |
| Male | 120.8 | -4.410 ^{***} [0.000] | -1.509 [0.144] | 0.398 ^{**} [0.024] | -0.458 ^{**} [0.029] |
| Female | 118.0 | -3.810 [0.424] | -1.347 [0.112] | -0.286 [*] [0.080] | -0.330 [0.121] |

Note: Wild bootstrap p -values with 500 repetitions are in brackets. Stunting is a dummy variable it equals 1 if the height is lower than 5 percentile for given age-gender group. The dependent variable in Column 4 indicates whether one's height is above 75 percentile for a given age-gender group. Each coefficient is from a separate regression. There are a total of 83,211 male students and 31,039 female students ranging from age 7 to age 10. Maternal mortality rate (ranging from 0 to 100) is imputed as an average of region-specific maternal mortality rate from the year (1927-age) and the year prior to that. Age is included in regressions for Column 1, gender dummies are included in regressions for all population (row 1), and infant mortality rate, region dummies, and region-specific time trends are included in all regressions.

^{*} Significant at 10% level.

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^{*} Significant at 10% level.

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Historical Context of Intergenerational Health Transmission

Chinese Great Famine (1959-61)

Impact: Survivors of the famine exhibited higher rates of metabolic disorders and mental health issues later in life.

Intergenerational Findings: Children of those exposed in utero had increased risks of obesity and schizophrenia, indicating multigenerational health impacts.



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Malnutrition in early life and adult mental health: Evidence from a natural experiment

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Historical

Chinese Grandchildren
Impact: Sur
health issue
Intergenerational
obesity and

Table 4

Estimated odds ratios of risk of mental illness predicted by famine exposure, based on ordered logit regression with difference-in-difference estimator.

| | Women | | Men | |
|------|-------|---------------|------|--------------|
| | OR | 95% CI | OR | 95% CI |
| 1956 | 1.95 | (0.80, 4.76) | 0.81 | (0.25, 2.60) |
| 1957 | 0.86 | (0.26, 2.84) | 0.68 | (0.23, 2.02) |
| 1958 | 1.48 | (0.52, 4.22) | 0.54 | (0.18, 1.57) |
| 1959 | 4.99 | (1.68, 14.84) | 0.69 | (0.19, 2.53) |
| 1960 | 2.24 | (0.71, 7.05) | 0.55 | (0.22, 1.37) |
| 1961 | 1.82 | (0.54, 6.13) | 0.65 | (0.18, 2.32) |
| 1962 | 2.34 | (0.98, 5.59) | 0.34 | (0.14, 0.80) |

Reference group is the 1963 birth cohort. Method of computing the estimated effect of famine exposure on the expanded three-level measure of risk of mental disorders is described in the [Methods Section](#).

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Historical Context of Intergenerational Health Transmission

The Biafran Famine (1967-1970)

Impact: This famine during the Nigerian Civil War led to high mortality and long-term health consequences in survivors, including increased susceptibility to infections and stunted growth.

Intergenerational Findings: Research indicates increased health risks in children of survivors, including low birth weight and developmental delays, potentially due to both physiological and psychological factors passed down.

First- and Second-Generation Impacts of the Biafran War

 Richard Akresh,  Sonia Bhalotra,  Marinella Leone and  Una Osili

Journal of Human Resources, March 2023, 58 (2) 488-531; DOI: <https://doi.org/10.3368/jhr.58.4.0118-9272R1>

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ABSTRACT

We analyze long-term impacts of the 1967–1970 Nigerian Civil War, providing the first evidence of intergenerational impacts. War exposure among women results in reduced adult stature, an increased likelihood of being overweight, earlier age at first birth, and lower educational attainment. War exposure of mothers has adverse impacts on next-generation child survival, growth, and education. Impacts vary with age of exposure. For the mother and child health outcomes, the largest impacts stem from adolescent exposure. Exposure to a primary education program mitigates impacts of war exposure. War exposure leads to men marrying later and having fewer children.

Q&A



Chapter II

Generational Transmission of Health

Key Question: How do health outcomes pass from parents to children and to their grandchildren?

- Beyond just biology, deeply intertwined with socioeconomic factors.
- Research insights:
 - Extensive studies on intergenerational transmission of health outcomes, from parents to children.
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Generational Transmission of Health

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Generational Transmission of Health

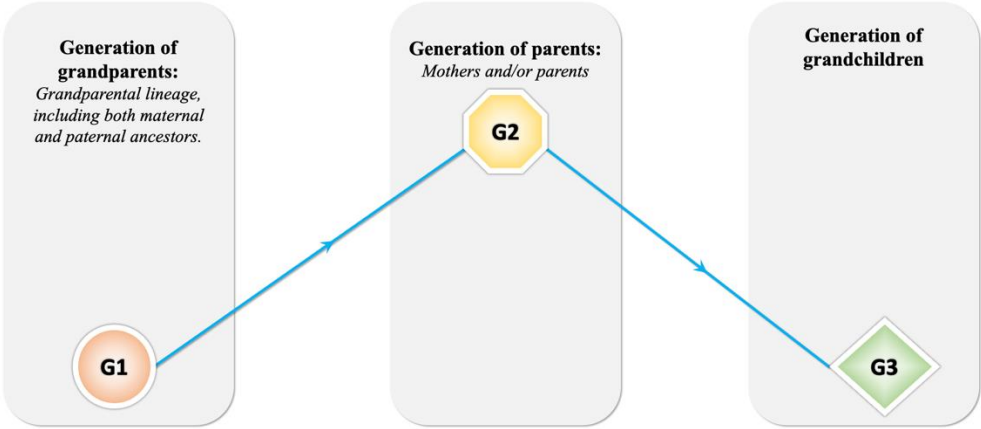
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Generational Transmission of Health: *Key Differences*

- Health is transmitted across generations through two primary mechanisms.
 - **Intergenerational:** Direct effects from one generation to the next adjacent generation.
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- **Multigenerational transmission** refers to effects that span more than two generations without necessarily skipping any.

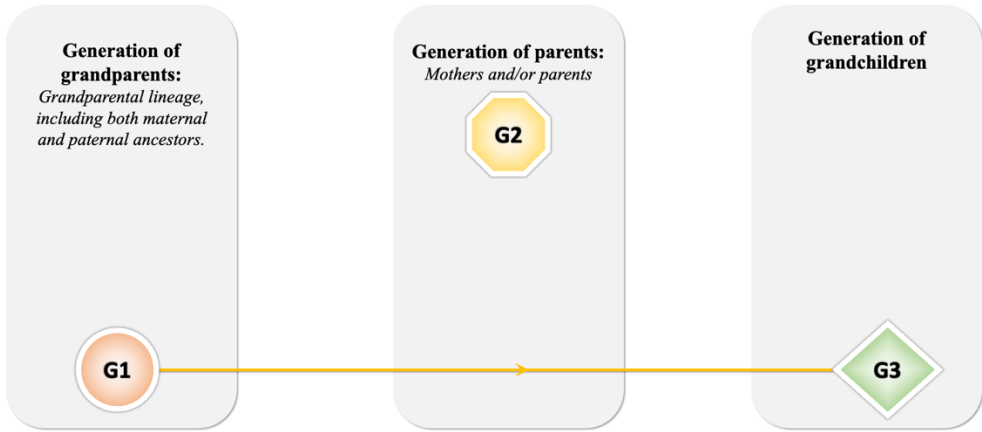
Generational Transmission of Health: *Intergenerational (Indirect Effects)*



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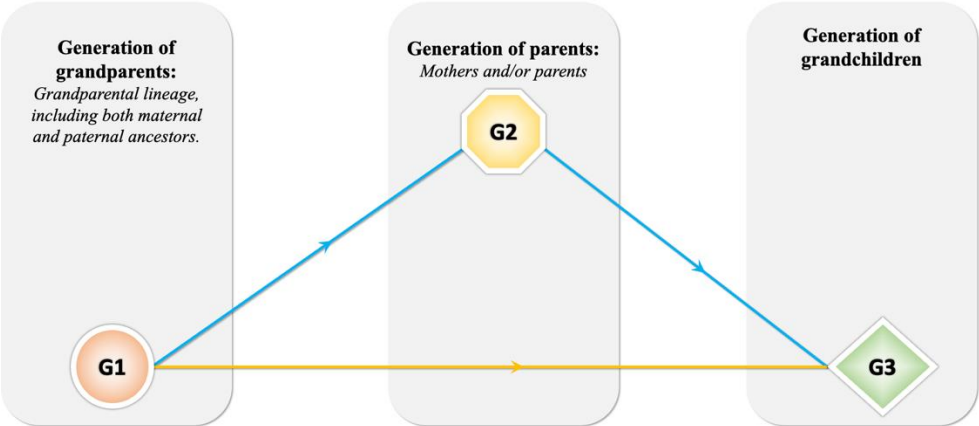
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Generational Transmission of Health: *Key Differences*

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Generational Transmission of Health: *Multigenerational transmission*





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



Now: My grandmother, Hatice San.
Photo by Fatma Geylan.

For access to the thesis:
<https://hdl.handle.net/10037/31578>

Conceptual Framework

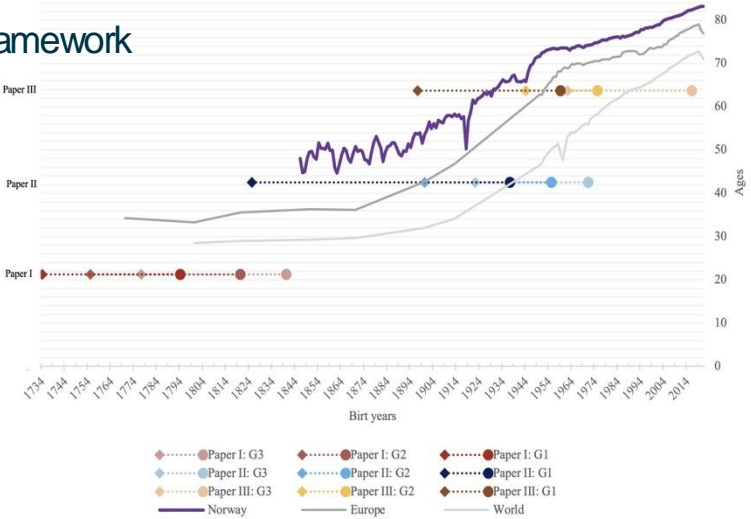


Figure 3.1 *The life expectancy at birth in Norway and the birth years covered in the Papers.*

Conceptual Framework

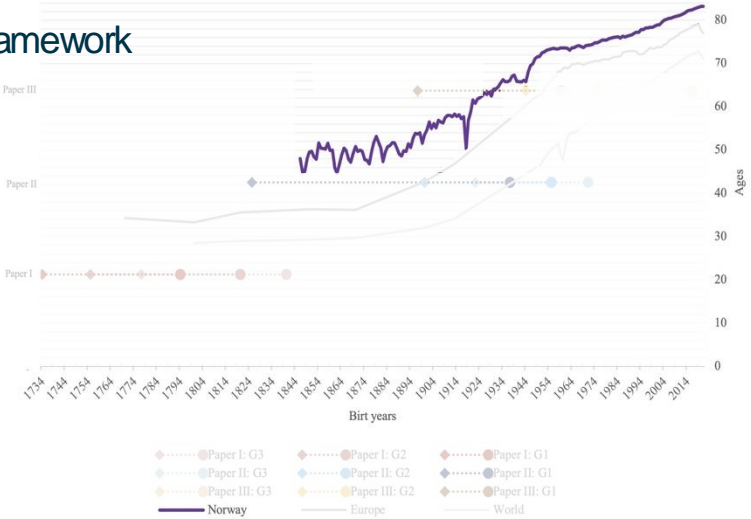


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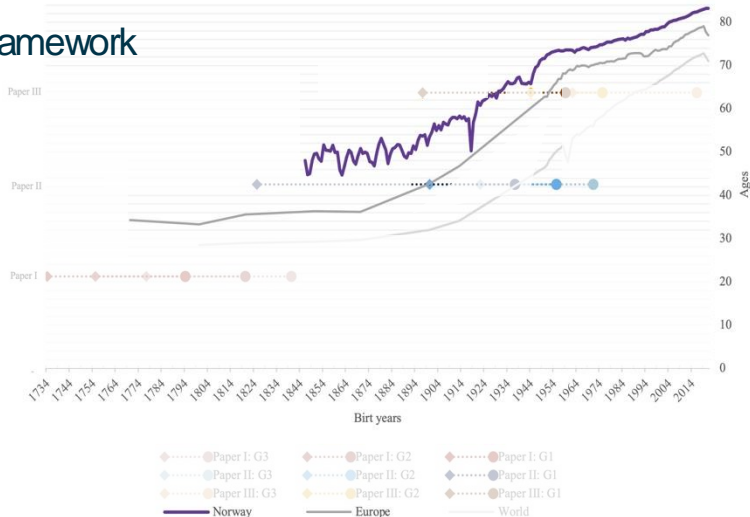


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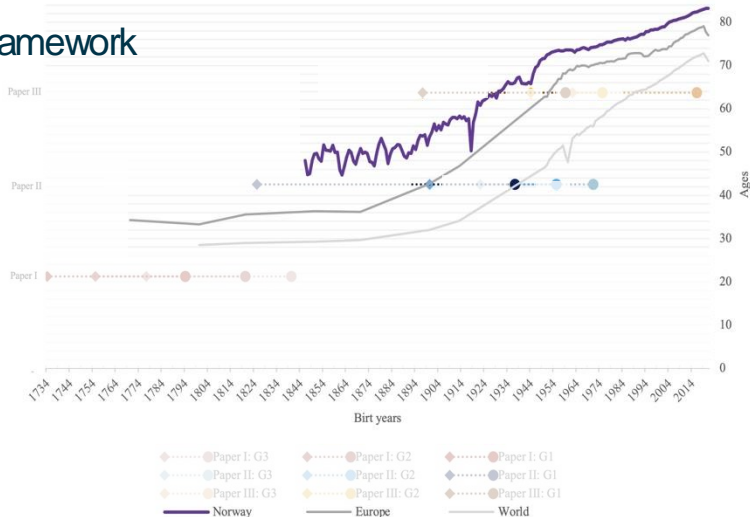


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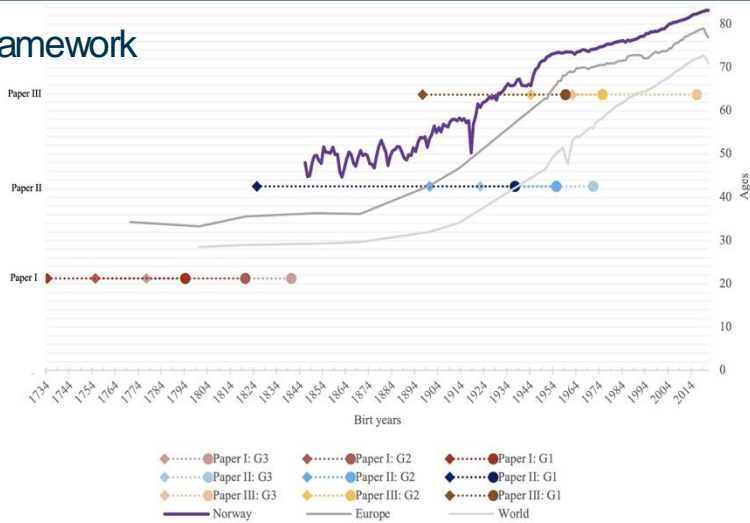


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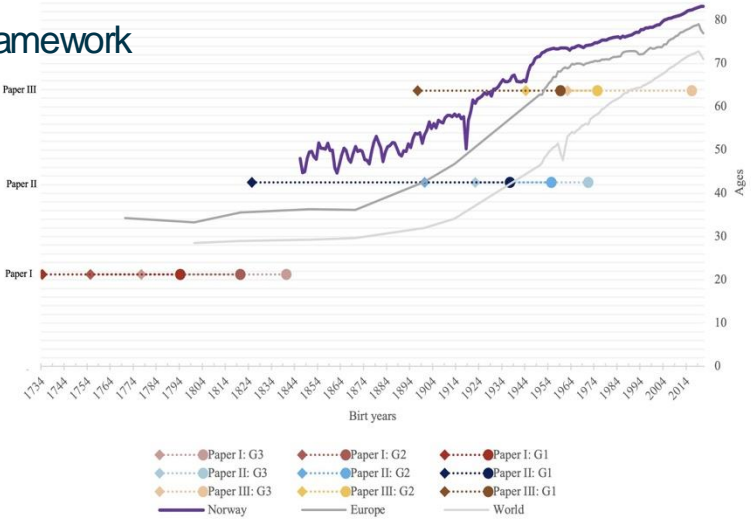


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Contents lists available at ScienceDirect

Economics and Human Biology

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



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

Emre Sari^{a,*}, Mikko Moilanen^a, Hilde Leikny Sommerseth^b

^a School of Business and Economics, Faculty of Biosciences Fisheries and Economics, UiT The Arctic University of Norway, Tromsø, Norway

^b Department of Archaeology, History, Religious Studies and Theology, Faculty of Humanities Social Sciences and Education, UiT The Arctic University of Norway, Tromsø, Norway

ARTICLE INFO

JEL Classification:

I1
I14
I15
J12
J13
J62
N33

Keywords:

Economic hardship

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.

Introduction

What are the underlying mechanisms behind **the transgenerational persistence in health?**

- **Motivation:** Understanding of relationship between economic hardship during pregnancy and grandchildren's health condition.
- **Aim:** To provide a historical overview of health transfer through three consecutive generations, based on the evidence from Rendalen over 1734-1840.
- **Hypothesis:** In this study, we hypothesize two main mechanisms by which economic hardship during the grandmother's pregnancy to her daughter can be associated with a grandchild's life span:
 - Positive culling effect
 - Negative scarring effect through the mother's health condition

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Introduction

- Only a limited number of studies have addressed how *in utero* exposure to external shock can affect subsequent generations:
 - **Lee (2014)** - the Kwangju uprising in South Korea
 - Cook et al. (2019) - the 1918 influenza pandemic
 - Van Den Berg & Pinger (2016) - German famine of 1916–1918

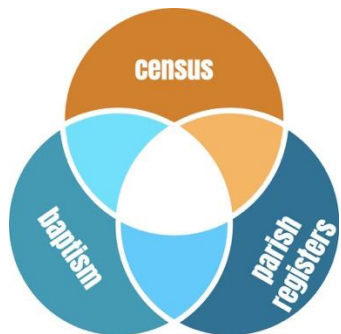
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Data Variable Description and Summary Statistics

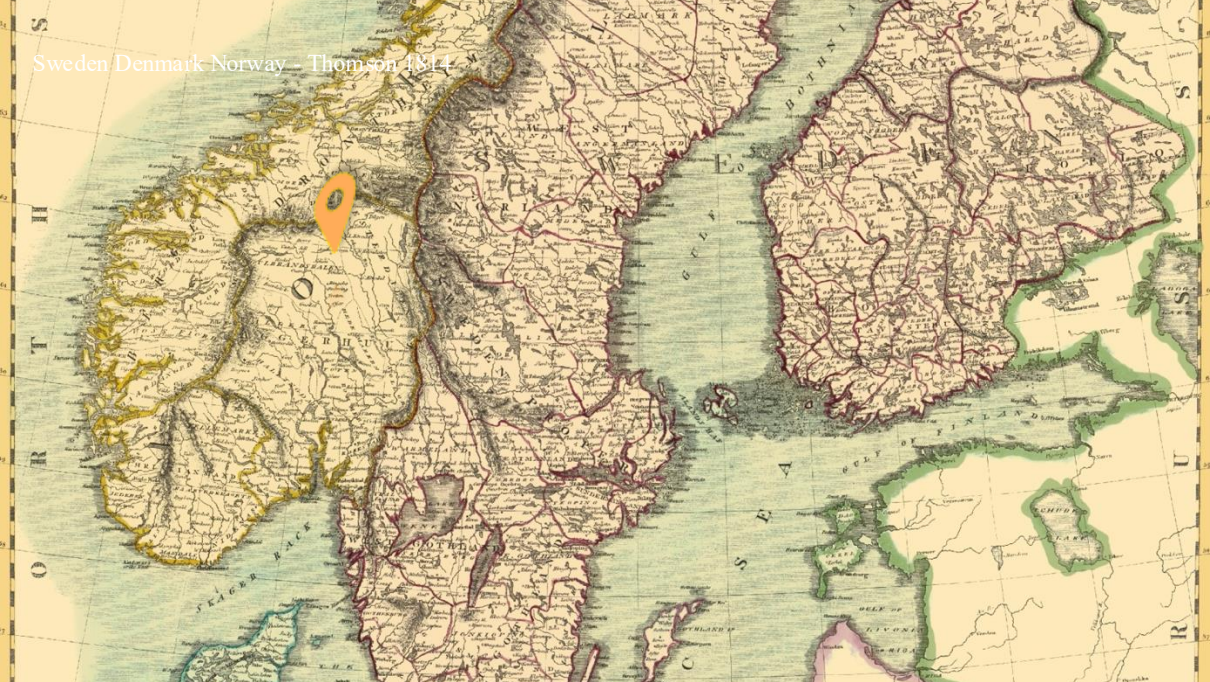


The Norwegian Historical Data Centre (NHDC)

The dataset was created by linking the censuses (1801, 1865, 1875, 1900 and 1910), parish registers, baptism and cadastral records (1733-1925).

Three-generation linked dataset that includes 798 children with parents and grandparents.

Sweden Denmark Norway - Thomson 1814



Data - Economic Hardship

The annual inflation rates (Grytten, 2018) determined in this study help us to define economic hardship over the period under examination.

- Grytten (2018, p. 50) characterizes the period between 1700 and 1820 as “turbulent economy- and inflationary-wise”.
- Our method Qvigstad (2005):
 - No static cutoff point.
 - Used inflation rates beyond interquartile range.
 - An annual inflation above the 3rd quartile (6.9%) or below the 1st quartile (-3.4%)
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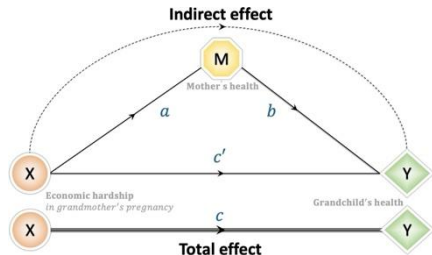
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Mediation Analysis

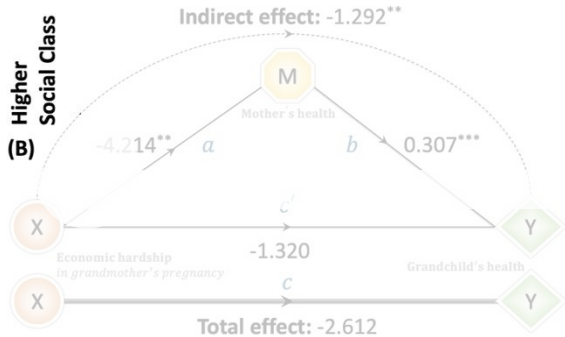
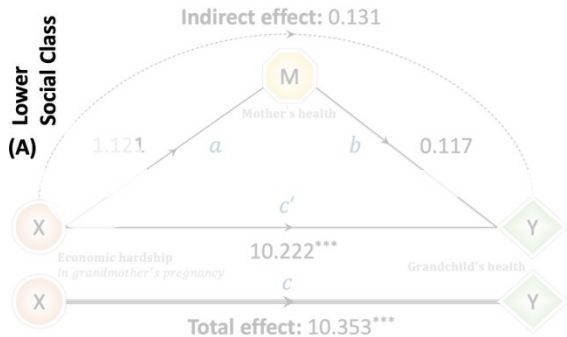
- The product coefficient of ab is a denotation of an average mediation effect. We calculated the total effect by multiplying the **a-path** coefficient by the b-path coefficient and adding the **c'-path** coefficient ($c' + ab$).

$$\text{Indirect effect} = ab$$

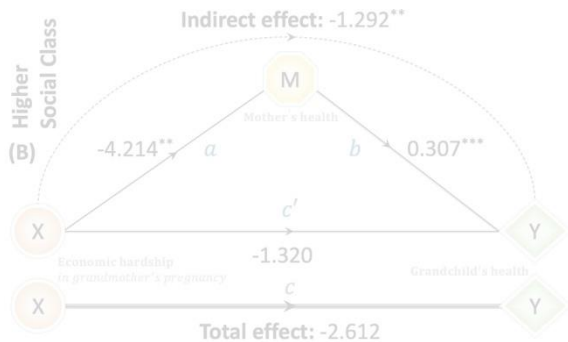
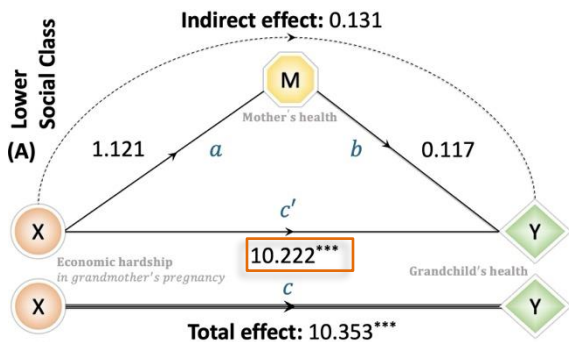
$$\text{Total effect} = c' + ab$$



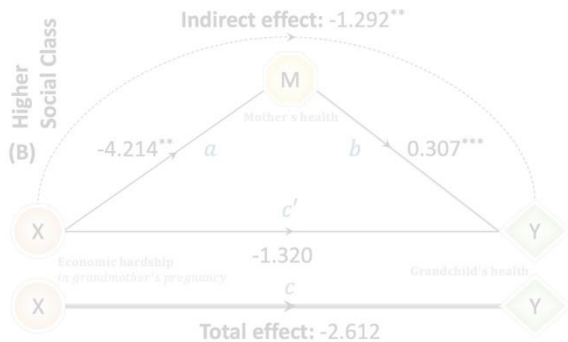
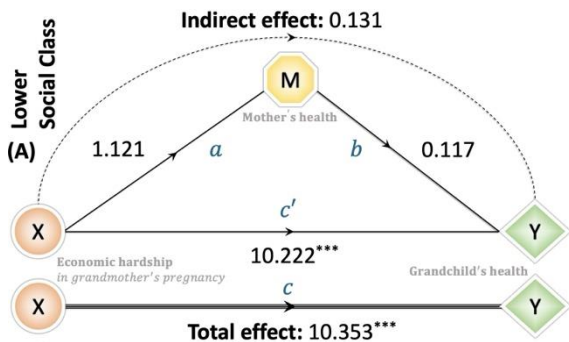
Results



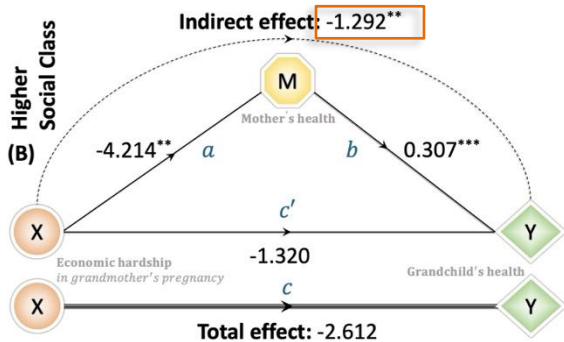
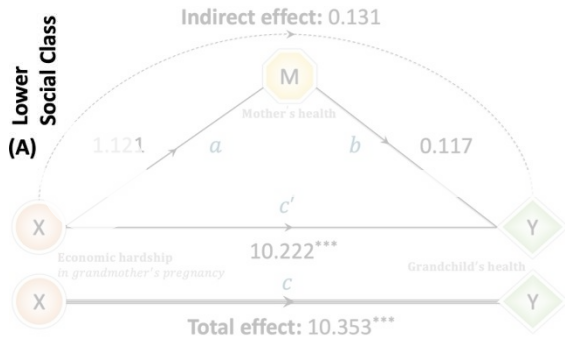
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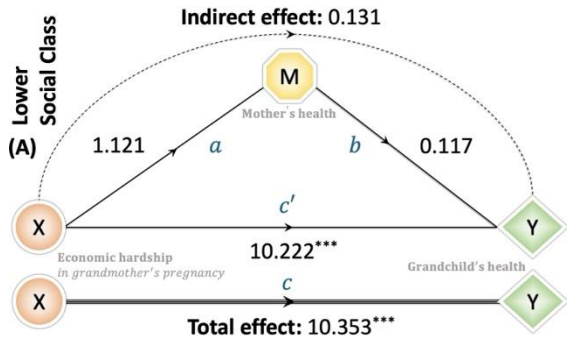
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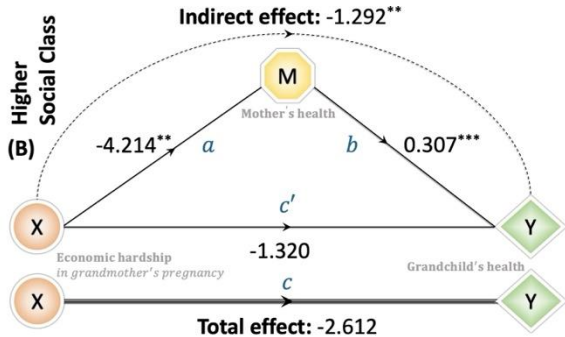
Results



Results



Positive selection/Culling effect



Negative scaring effect



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Social Science & Medicine

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



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

Emre Sari^{a,b,*}, Mikko Moilanen^a, Maarten Lindeboom^c

^a *UIT the Arctic University of Norway, School of Business and Economics, Tromsø, Norway*

^b *NORCE Norwegian Research Centre, Division for Health and Social Sciences, Tromsø, Norway*

^c *Vrije Universiteit Amsterdam, School of Business and Economics, Amsterdam, Netherlands*

ARTICLE INFO

Handling Editor: Social Epidemiology Office

JEL classification:

JEL
I10
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Keywords:

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

ABSTRACT

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. The findings show a matrilineal 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.

Introduction

This study investigates the multigenerational transmission of risky health behaviors, specifically smoking, within the Norwegian context.

- **Motivation:** Literature shows parent-offspring smoking links, but clarity on direct grandparent-offspring connections is missing.
- **Aim:** To investigate whether tobacco smoking is correlated with earlier generations' smoking behavior and, if it is, whether maternal versus paternal grandparents affect grandchildren differently.
- **Hypothesis:** Adult smoking behavior of grandchildren may be directly influenced by their grandparents' past smoking behavior, independent of their parents' smoking behavior.

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- **Vandewater et al. (2014)** — U.S. - *Indirect effect*
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- **Duarte et al. (2015)** — Spain - *Direct & Indirect effect*
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The existing literature mostly focuses on indirect effect and parent-child transmission, with less attention given to the direct influence of grandparents.

- The study is grounded in **Social Learning Theory** and **Health Behavior Models**.
- These theories offer a robust and comprehensive understanding of how observational learning, personal health beliefs, and social norms contribute to the transmission and prevention of smoking behaviors across generations.

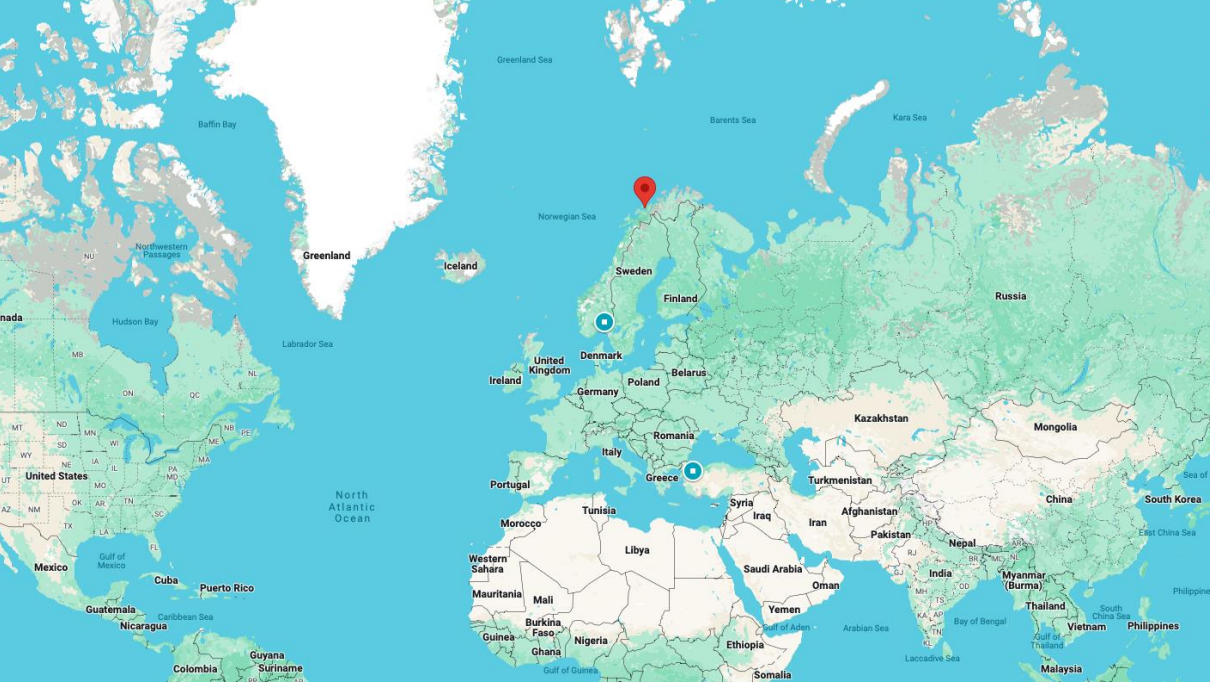
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Data - The Tromsø Study

- Conducted from 1974-2016 in 7 waves.
- Covers health-related data of adults in Tromsø.
- First time establishing family connections.
 - The study database and key family identification numbers were obtained from the Norwegian Tax Administration, ensuring the robustness of the linkages.



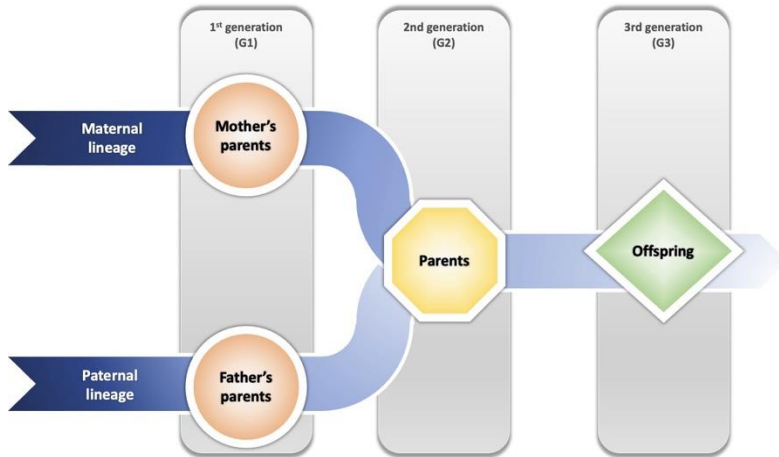
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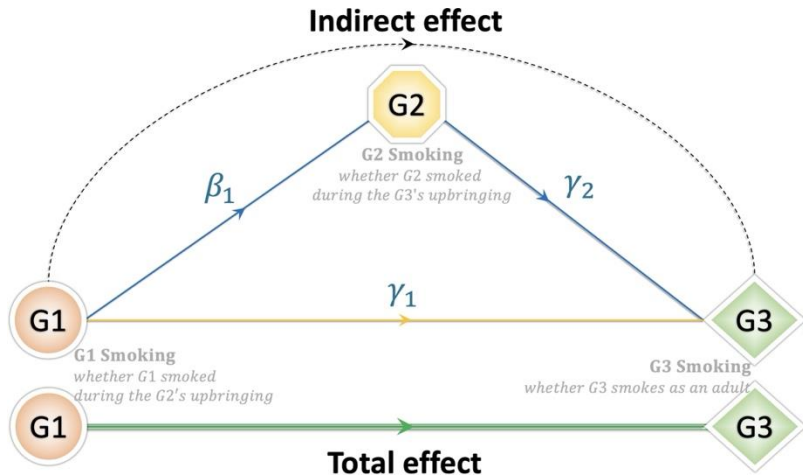
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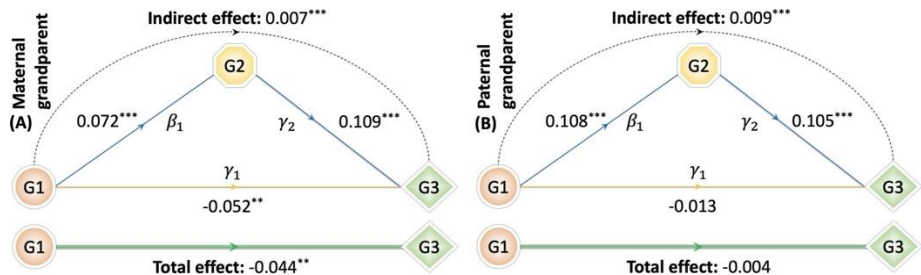
Data - Generations



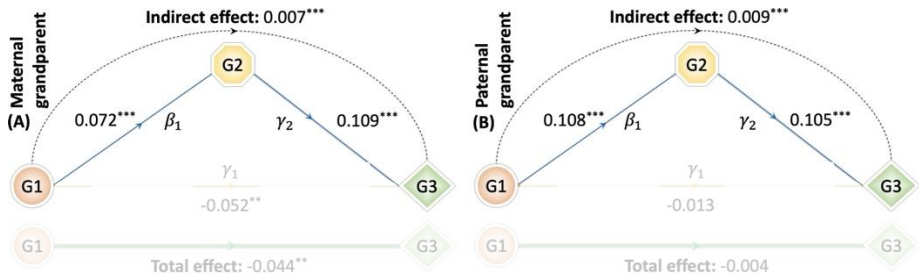
Sobel's Product of Coefficients Approach



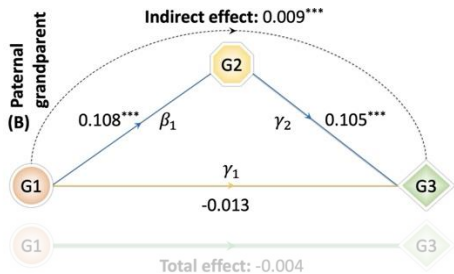
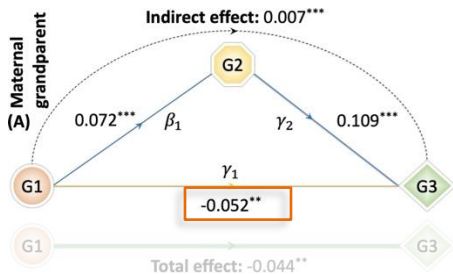
Results



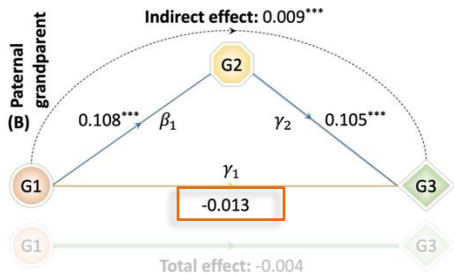
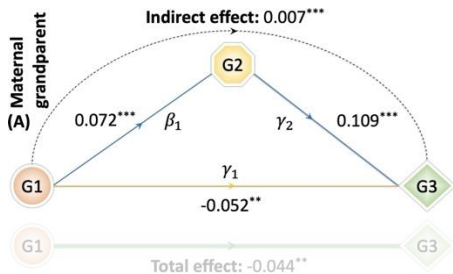
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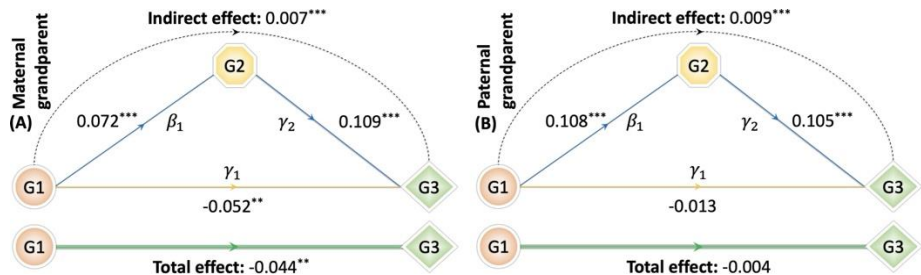
Results



Results



Results





ELSEVIER

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SSM - Population Health

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



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

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^a School of Business and Economics, UiT the Arctic University of Norway, Tromsø, Norway

^b Division for Health and Social Sciences, NORCE Norwegian Research Centre, Oslo, Norway

^c School of Business and Economics, Vrije Universiteit Amsterdam, Amsterdam, Netherlands

^d Centre for Health Economics, Monash University, Melbourne, Australia

ARTICLE INFO

Keywords:

Child neglect
Adverse childhood experiences
Childhood trauma
Mental health
Depression
Grandparental influence

ABSTRACT

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. We utilize a three-generational dataset from the Tromsø Study and estimate a linear probability model to find the distinct roles of both maternal and paternal grandparents. We test the additive risk hypothesis for continuous, intergenerational effects of child neglect in both the maternal and paternal lineages. Furthermore, we use structural equation modeling to test how sequential exposures to neglect across generations ultimately bear on adult mental health outcomes. Our results confirm the additive risk hypothesis but only for maternal grandparents: our findings show that only maternal parents' neglectful parenting is associated with an increased probability of depression in their grandchildren, conditional on whether their parents neglected them. These results contribute to research on intergenerational transmission by the finding that additive risks of child maltreatment flow down generations mainly through maternal lineages.

Introduction - Research Questions

- This study aims to extend upon current research by considering the impact of maternal and paternal grandparents separately.
- We seek to answer the following questions:
 - ① To what extent does grandparental child neglect in the first generation predict the probability of mental health problems in the third generation?
 - ② Do neglectful maternal and paternal grandparents have differential relations to their grandchildren's mental health?

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Introduction - Intergenerational Transmission

- Neglectful behavior impacts generations.
- Intergenerational cycle of violence hypothesis (Alink et al., 2019; Abramovaite et al., 2015).
- Grandparents' neglect may lead to mental issues in grandchildren (Langevin et al., 2023; Widom, 2017).
- Cycle can lead to societal and economic costs (World Health Organization, 2020).

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Data

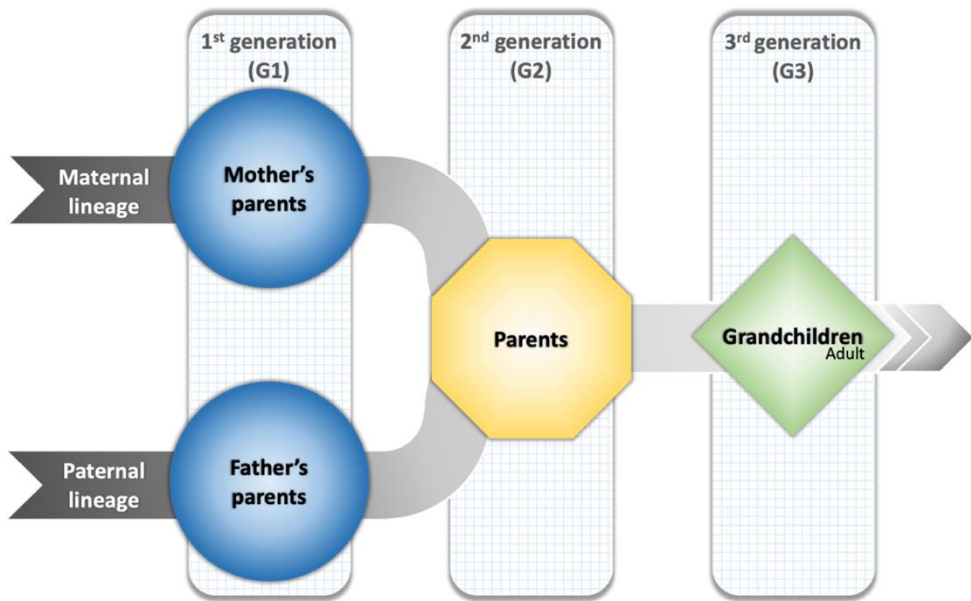
Tromsø7

- First time establishing family connections.
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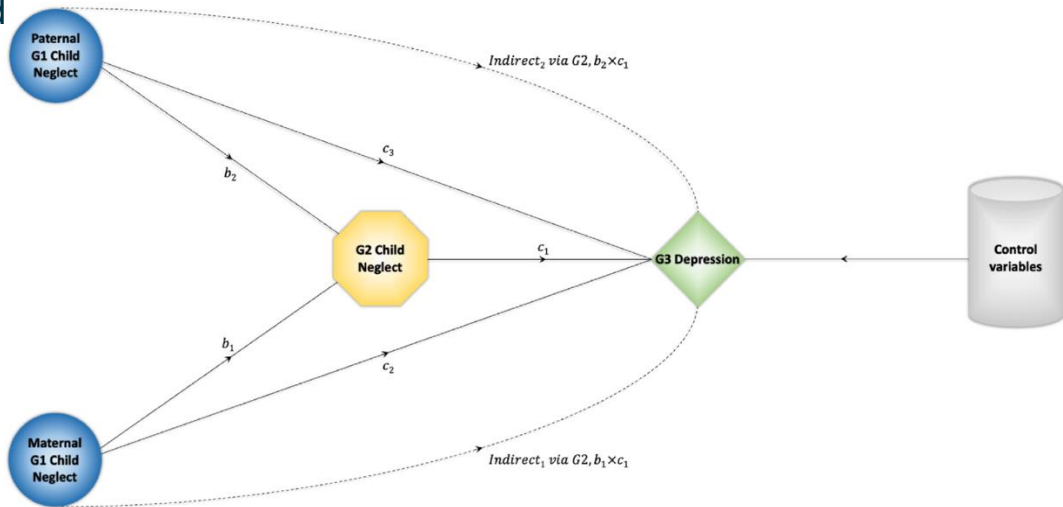


Fig. 2. Conceptual framework of Structural Equation Modeling.

Results

Table 4

Results of the effect of child neglect from maternal and paternal grandparents and parents on grandchildren's mental health.

| Variables | Dependent variable: <i>Mental health status of G3</i> | | | |
|--|---|---------------------|---------------------------|---------------------|
| | OLS | | Probit (Marginal effects) | |
| | (1) | (2) | (3) | (4) |
| G2 Child-neglect | 0.249*** (0.075) | 0.199*** (0.083) | 0.251*** (0.076) | 0.201*** (0.084) |
| Maternal G1 Child-neglect | -0.004 (0.062) | -0.052 (0.065) | -0.009 (0.067) | -0.057 (0.068) |
| Paternal G1 Child-neglect | 0.039 (0.070) | 0.042 (0.072) | 0.037 (0.073) | 0.040 (0.075) |
| Maternal G1 Child-neglect x G2 Child-neglect | | 0.379** (0.180) | | 0.426* (0.222) |
| Paternal G1 Child-neglect x G2 Child-neglect | | -0.059 (0.324) | | -0.053 (0.293) |
| Control variables | ✓ | ✓ | ✓ | ✓ |
| Observations | 1258 | 1258 | 1258 | 1258 |
| <i>R-squared</i> | 0.024 | 0.027 | | |
| <i>AIC</i> | | | 1517.6 | 1518.2 |

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 controlled 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.3](#). 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.

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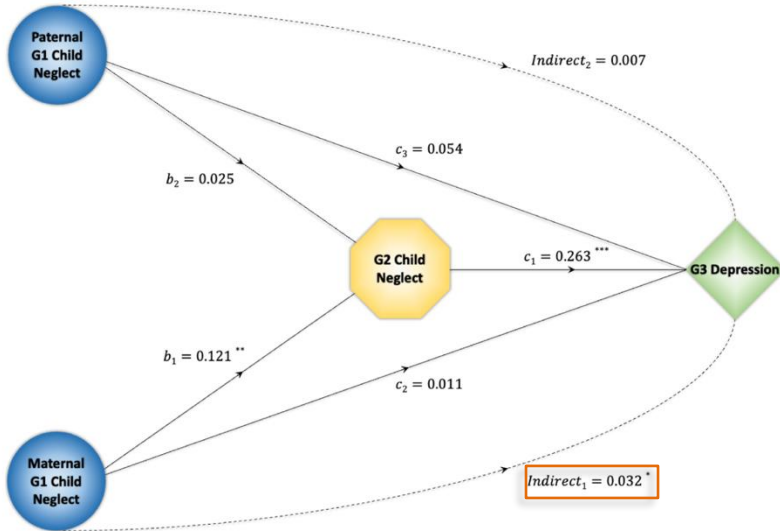
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Discussion and Future Research

Promising Directions:

Explore innovative longitudinal studies on intergenerational health, leveraging extensive historical datasets.

Develop novel methodologies for analyzing health inequalities over time, including cross-country comparisons.

Future Research Areas:

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Grandparental investment: Past, present, and future

David A. Coall

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Ralph Hertwig

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Abstract: What motivates grandparents to their altruism? We review answers from evolutionary theory, sociology, and economics. Sometimes in direct conflict with each other, these accounts of grandparental investment exist side-by-side, with little or no theoretical integration. They all account for some of the data, and none account for all of it. We call for a more comprehensive theoretical framework of grandparental investment that addresses its proximate and ultimate causes, and its variability due to lineage, values, norms, institutions (e.g., inheritance laws), and social welfare regimes. This framework needs to take into account that the demographic shift to low fecundity and mortality in economically developed countries has profoundly altered basic parameters of grandparental investment. We then turn to the possible impact of grandparental acts of altruism, and examine whether benefits of grandparental care in industrialized societies may manifest in terms of less tangible dimensions, such as the grandchildren's cognitive and verbal ability, mental health, and well-being. Although grandparents in industrialized societies continue to invest substantial amounts of time and money in their grandchildren, we find a paucity of studies investigating the influence that this investment has on grandchildren in low-risk family contexts. Under circumstances of duress – for example, teenage pregnancy or maternal depression – there is converging evidence that grandparents can provide support that helps to safeguard their children and grandchildren against adverse risks. We conclude by discussing the role that grandparents could play in what has been referred to as Europe's demographic suicide.

Keywords: child development; demographic transition; empathy; grandmother hypothesis; grandparental investment; grandparental solicitude; intergenerational transfers; kin altruism; maternal depression; reciprocal altruism

Multigenerational Effects of Early-Life Health Shocks

C. Justin Cook¹ · Jason M. Fletcher^{2,3,4,5,6,7} · Angela Forgues^{3,6,7}

Published online: 29 July 2019

© Population Association of America 2019

Abstract

A large literature has documented links between harmful early-life exposures and later-life health and socioeconomic deficits. These studies, however, have typically been unable to examine the possibility that these shocks are transmitted to the next generation. Our study uses representative survey data from the United States to trace the impacts of *in utero* exposure to the 1918 influenza pandemic on the outcomes of the children and grandchildren of those affected. We find evidence of multigenerational effects on educational, economic, and health outcomes.

Keywords 1918 influenza · Multigenerational effects · Wisconsin Longitudinal Study



Some Methodological Problems in the Study of Multigenerational Mobility

Richard Breen*

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Submitted February 2018; revised July 2018; accepted August 2018

Abstract

A number of recent studies by sociologists have sought to discover whether a person's status (typically their social class, education, or socio-economic status) is directly affected by the status of their grandparents, once the effects of parents' status are controlled. The results have been ambiguous, with some studies finding a direct effect of grandparents on their grandchildren, while others find no effect. I use causal graphical methods to demonstrate some of the methodological problems that occur in trying to identify this direct effect, and I offer some suggestions as to how they might be addressed.

Introduction

association between grandparents and their grandchildren,



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Intergenerational health consequences of *in utero* exposure to maternal stress: Evidence from the 1980 Kwangju uprising[☆]



Chulhee Lee

Department of Economics, Seoul National University, Seoul, South Korea

ARTICLE INFO

Article history:

Available online 2 July 2014

Keywords:

Stress in pregnancy
Birth outcomes
Low birth weight
Preterm birth
Intergenerational effect
Kwangju uprising

ABSTRACT

The evidence that demonstrates the negative effects of maternal psychological stress during pregnancy on a wide variety of offspring outcomes is growing. Animal studies suggest that negative influences of maternal stress during pregnancy persist across multiple generations, but the direct evidence to confirm that the effect is present among human populations is scarce. This study draws evidence on the intergenerational influences of maternal stress from the Kwangju uprising (May 18–27, 1980), arguably the bloodiest incident that occurred in South Korea since the end of the Korean War in 1953. The results of difference-in-difference estimations suggest that *in utero* exposure to the Kwangju uprising significantly diminished the offspring birth weight and length of gestation, and increased the risks of low birth weight and preterm birth. Exposure to stress during the second trimester of pregnancy exerted the strongest negative effect on grandchildren's birth outcomes.



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Transgenerational effects of childhood conditions on third generation health and education outcomes



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ABSTRACT

This paper examines the extent to which pre-puberty nutritional conditions in one generation affect productivity-related outcomes in later generations. Recent findings from the biological literature suggest that the so-called slow growth period around age 9 is a sensitive period for male germ cell development. We build on this evidence and investigate whether undernutrition at those ages transmits to children and grandchildren. Our findings indicate that third generation males (females) tend to have higher mental health scores if their paternal grandfather (maternal grandmother) was exposed to a famine during the slow growth period. These effects appear to reflect biological responses

A Multigenerational View of Inequality

Robert D. Mare

Published online: 27 January 2011

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Abstract The study of intergenerational mobility and most population research are governed by a two-generation (parent-to-offspring) view of intergenerational influence, to the neglect of the effects of grandparents and other ancestors and nonresident contemporary kin. While appropriate for some populations in some periods, this perspective may omit important sources of intergenerational continuity of family-based social inequality. Social institutions, which transcend individual lives, help support multigenerational influence, particularly at the extreme top and



Contents lists available at ScienceDirect

Social Science & Medicine

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

Review article

How do grandparents influence child health and development? A systematic review



Aalyia F.A. Sadruddin^a, Liliana A. Ponguta^a, Anna L. Zonderman^b, Kyle S. Wiley^a,
Alyssa Grimshaw^a, Catherine Panter-Brick^{a,*}

^a Yale University, USA^b The University of Chicago, USA

ARTICLE INFO

Keywords:

Generation
Family
Parenting
Child
Health
Development
Research design
Policy

ABSTRACT

Grandparents are often a key source of care provision for their grandchildren, yet they are sidelined in caregiving research and policy decisions. We conducted a global, systematic review of the literature to examine the scope and quality of studies to date (PROSPERO database CRD42019133894). We screened 12,699 abstracts across 7 databases, and identified 206 studies that examined how grandparents influence child health and development. Indicators of grandparent involvement were contact, caregiving behaviors, and financial support. Our review focused on two research questions: how do grandparents influence child health and development outcomes, and what range of child outcomes is reported globally? We examined study design, sample characteristics, key findings, and outcomes pertaining to grandchildren's physical health, socio-emotional and behavioral health, and cognitive and educational development. Our search captured studies featuring grandparent custodial care (n = 35), multigenerational care (n = 154), and both types of care (n = 17). We found substantial heterogeneity in the data provided on co-residence, caregiving roles, resources invested, outcomes, and mechanisms through which "grandparent effects" are manifested. We identified two important issues, related to operationalizing



Grandparenting, health, and well-being: a systematic literature review

Mirkka Danielsbacka¹ · Lenka Křenková² · Antti O. Tanskanen¹

Accepted: 6 December 2021 / Published online: 4 January 2022
© The Author(s) 2022

Abstract

Whether grandparenting is associated with improved health or well-being among older adults is a salient question in present-day aging societies. This systematic review compiles studies that consider the health or well-being outcomes of grandparenting, concerning (1) custodial grandparent families, where grandparents are raising grandchildren without parental presence; (2) three-generation households, where grandparents are living with adult children and grandchildren; and (3) non-coresiding grandparents, who are involved in the lives of their grandchildren. Review was based on literature searches conducted in September 2019 via Web of Science, PubMed, PsycINFO, and Ebsco. We screened 3868 abstracts across four databases, and by following the PRISMA guidelines, we identified 92 relevant articles (117 studies) that were published between 1978 and 2019. In 68% of cases, custodial grandparenting was associated with decreased health or well-being of grandparents. The few studies considering the health or well-being of grandparents living in three-generation households provided mixed findings (39% positive; 39% negative). Finally, in 69% of cases, involvement of non-coresiding grandparents was associated with improved grandparental outcomes; however, there was only limited support for the prediction that involved grandparenting being causally associated with grandparental health or well-being. Despite this, after different robustness checks (counting all nonsignificant results, taking into account the representativeness of the data and causal methodology), the main finding remains the same: the most negative results are found among custodial grandparents and three-generation households and most positive results among non-coresiding grandparents.

Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development

JANET CURRIE*

There are many possible pathways between parental education, income, and health, and between child health and education, but only some of them have been explored in the literature. This essay focuses on links between parental socioeconomic status (as measured by education, income, occupation, or in some cases area of residence) and child health, and between child health and adult education or income. Specifically,

ARTICLE

Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period

G Kaati¹, LO Bygren^{*1} and S Edvinsson²

¹*Department of Community Medicine and Rehabilitation, Social Medicine, Umeå University, Umeå, Sweden;*

²*Demographic Database, Umeå University, Umeå, Sweden*

Overfeeding and overeating in families are traditions that are often transferred from generation to generation. Irrespective of these family traditions, food availability might lead to overfeeding, in its turn leading to metabolic adaptations. Apart from selection, could these adaptations to the social environment have transgenerational effects? This study will attempt to answer the following question: Can overeating during a child's slow growth period (SGP), before their prepubertal peak in growth velocity influence descendants' risk of death from cardiovascular disease and diabetes? Data were collected by following three cohorts born in 1890, 1905 and 1920 in Överkalix parish in northern Sweden up until death or 1995. The parents' or grandparents' access to food during their SGP was determined by referring to historical data on harvests and food prices, records of local community meetings and general historical facts. If food was not readily available during the father's slow growth period, then cardiovascular disease mortality of the proband was low. Diabetes mortality increased if the paternal grandfather was exposed to a surfeit of food during his slow growth period. (Odds Ratio 4.1, 95%

Thank you!

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