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

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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 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.

1. 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 (Ritchie and Roser, 2018). Smoking, in particular, is a major health risk that profoundly affects individual health and the economy at large (Qin et al., 2016).

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 (Miura, 2019). Furthermore, these preferences and behaviors can be transmitted across generations, from parents to children (Brown and van der Pol, 2015). 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. The mechanisms behind the intergenerational transfer of smoking behavior are multifaceted, involving both genetic and environmental factors. Concurrently, cultural factors such as values, beliefs, and attitudes significantly contribute to the intergenerational transmission of behaviors (Brown and van der Pol, 2015). Parents have the ability to influence these cultural factors, which in turn influence socio-emotional development (Zeng and Xie, 2014).

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). These investments can directly or indirectly benefit their grandchildren, serving as vital factors in human capital endowment. Danielsbacka et al. (2015) suggest that natural selection tends to reward actions or behaviors that have a basis in genetics and improve overall inclusive fitness. 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' secondhand smoke are more likely to smoke themselves (Gottfredson et al., 2017), grandparents' role in influencing their grandchildren's behavior

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and thus contributing to the transmission of smoking across generations becomes an interesting area of exploration.

Many studies show that maternal grandmothers provide more care and resources than paternal grandmothers, and numerous studies have demonstrated this matrilateral bias in contact, childcare, and emotional intimacy (see, e.g., Coall and Hertwig (2010); Lehti (2020); Sadruddin et al. (2019); Tu et al. (2021)). This asymmetry is the matrilateral bias in grandmaternal investment. Coall and 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 (Coall and 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. 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). Drawing from previous research, we expect that the impact of maternal grandparents on their grandchildren will be more pronounced compared to paternal grandparents. Specifically, we anticipate a negative association between the influence of maternal grandparents and the probability of smoking, reflecting a pattern of matrilateral bias.

The trend of increased life expectancy has amplified grandparents' role in their grandchildren's lives, offering more opportunities for interaction and influence (Sari, 2023; 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; 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. 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 and 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 (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.

Combining these theoretical perspectives, our study utilizes a unique three-generational dataset from Tromsø, Northern Norway. Our empirical methodology leverages 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 examine this transmission thoroughly 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 possibility of matrilateral bias in health behavior transmission, which may alter the conventional perspective of familial influence and individual health choices. Our finding of a negative direct effect of maternal grandparents makes a valuable contribution by highlighting a novel aspect in the complex interplay between family influence and individual health behavior. Specifically, our findings suggest that grandchildren are less likely to engage in smoking behavior due to the influence of their maternal grandparents. 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.

2. Data

2.1. The Tromsø Study

Tromsø is the largest city in Northern Norway and has about 77,000 inhabitants. The Tromsø Study 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. The study spans the years 1974–2016 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). The study has approval from the Regional Committees for Medical and Health Research Ethics. All participants provided informed consent before being accepted into the study. For more information, you can visit the official website at https://uit.no/research/tromsostudy.

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 intergenerational transmission in risky health behaviors. As presented in Fig. 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.

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.

2.2. Dependent variable: smoking behavior of offspring

We constructed the 'smoking' variable differently for each generation. For G3, we used responses from multiple smoking-related questions across the Tromsø Studies to determine whether they have ever smoked occasionally or regularly. Some of these questions are '*Do you smoke daily at present?*', '*Do you/did you smoke daily?*' and '*Do you smoke, or have you smoked sometimes, but not daily?*' (For more details, see Appendix A.1 Table A.1). Based on these responses, we determined whether G3 was a regular or occasional smoker in the past or at the time of the survey. Later, we categorized the offspring based on their smoking habits: those who smoked regularly or occasionally, either currently or in the past, were classified as smokers and as non-smokers otherwise.

Unlike previous studies on intergenerational transmission of smoking behavior for three generations, we focus on adulthood smoking behavior in the last generation rather than adolescent ages (see, e.g., Duarte et al. (2016); El-Amin et al. (2015); Vandewater et al. (2014)).

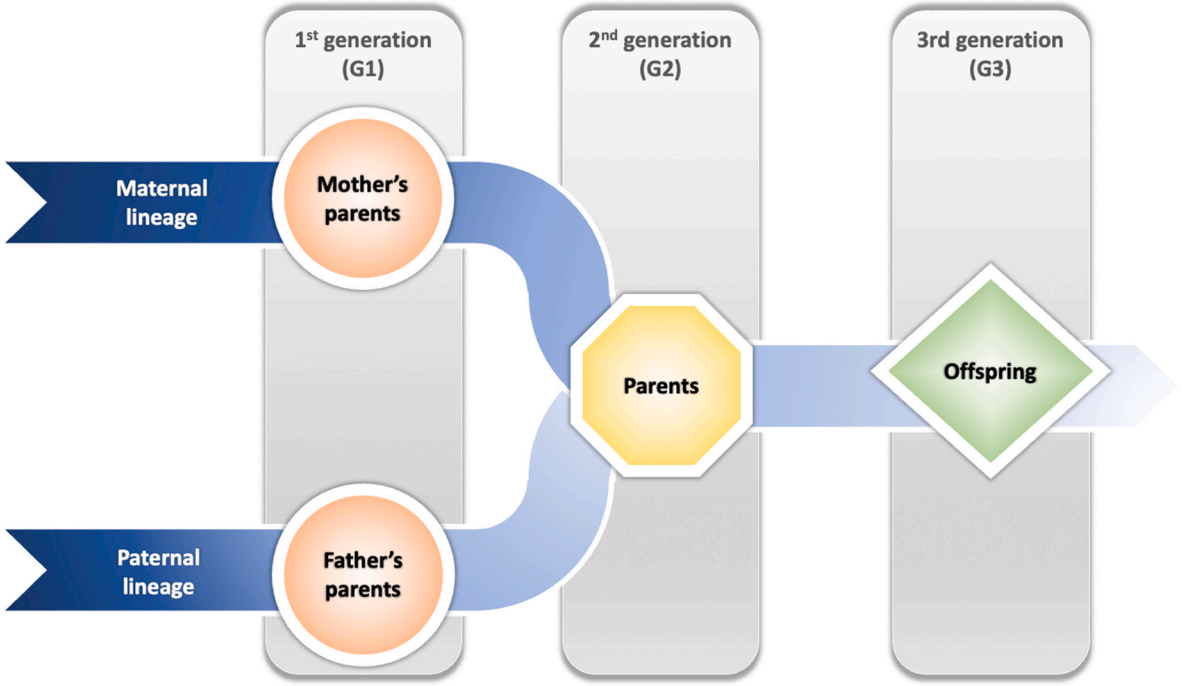

Fig. 1. Diagram and definition of generations.
Note: The generation order begins with the grandparent's generation, labeled as G1. Maternal G1 indicates the mother's parents, maternal lineage, while paternal G1 indicates the father's parents, paternal lineage. The second generation, G2, represents the offspring's (G3) parents, who participated in the Tromsø Study.

Table 1
Descriptive statistics for three generations.

Variable names	Description	Maternal lineage -Mean (SD)	Paternal lineage -Mean (SD)
	Third generation (G3) – Offspring		
G3 Smoking	*Whether G3 smokes as an adult*	0.65 (0.48)	0.64 (0.48)
G3 Year born	*Year G3 was born*	1960 (7.51)	1962 (6.94)
G3 Female	*Gender of G3 (Female = 1)*	0.52 (0.5)	0.52 (0.5)
G3 Childhood economic conditions	*Household economic conditions during G3's childhood*	0.79 (0.41)	0.83 (0.37)
	Second generation (G2) - Parents		
G2 Smoking	*Whether G2 smoked during the G3's upbringing*	0.78 (0.41)	0.77 (0.42)
G2 Recent smoking	*Whether G2 was smoking at the time of the survey.*	0.50 (0.50)	0.67 (0.47)
G2 Year born	*Year G2 was born*	1932 (9.53)	1931 (9)
G2 Childhood economic conditions	*Household economic conditions during G2's childhood*	0.66 (0.47)	0.58 (0.49)
	First generation (G1) - Grandparents		
G1 Smoking	*Whether G1 smoked during the G2's upbringing*	0.65 (0.48)	0.70 (0.46)
N	*Number of observations*	*5725*	*4057*

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' tothe father and his parents. 'Household economic conditions during childhood' refers to the perceived financial situation during childhood, categorized into two groups: lower economic conditions (used as the reference category) and higher economic conditions.

Adult behavior, as opposed to child behavior, is a more informative indicator of how widespread smoking has been among generations (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.

For the G2 and G1 generations, we used the recollections of the subsequent generations to determine their past smoking behaviors. Specifically, for G2 smoking and G1 smoking, we used responses from G3 for G2 and G2 for G1 to questions like, *'Did any of the adults smoke at home while you were growing up?'* We adopted this approach since direct data on the smoking behavior of G1 and G2 were not available for the relevant periods. Using the subsequent generation's memories as a proxy is based on the assumption that the household environment creates a lasting impression, particularly during formative years. Sheikh (2017) employed a similar question from the Tromsø Study and defined it as exposure to passive smoke during childhood to create a childhood adversity score and emphasized the good reliability of this variable. Moreover, his study shows a significant relationship between reported childhood adversities and daily smoking in adulthood. Thus, this question effectively captures the perceived smoking environment during G3 and G2's upbringing, which can considerably influence the attitudes and behaviors of the subsequent generation toward smoking.

Additionally, we introduced another variable for the G2 generation, G2 recent smoking, which indicates whether G2 was smoking at the time of their participation in the Tromsø Study. This variable was constructed using the same procedure G3 smoking variable, ensuring a consistent and directly comparable intergenerational analysis. It provides an up-to-date snapshot of G2's recent smoking status and complements the retrospective data captured in G2 smoking. The inclusion of this contemporary measure allows for a better understanding of the smoking behavior dynamics within and across generations, aligning with the literature on smoking behavior over the life course (see, e.g., El-Amin et al. (2015); Gilman et al. (2009)). Importantly, G2 recent smoking serves as a timely counterpart to G3 smoking, enabling a more robust and comprehensive intergenerational comparison. Due to data limitations, we could not include a 'G1 recent smoking' variable, as we did not have sufficient data to capture the most recent smoking status of the G1 generation.

2.3. Control variables

In our models, we controlled for demographic and socioeconomic characteristics. We collected data on childhood economic conditions 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 (0 for lower economic conditions, 1 for higher economic conditions). 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. 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. To achieve this, we include the variable 'childhood economic conditions,' representing the household's economic status during an individual's formative years. This variable serves as a proxy for the socioeconomic status (SES) during both G2's and G3's developmental periods. The significance of this variable cannot be overstated, given the compelling evidence linking childhood SES to health behaviors, including smoking (Gilman et al., 2003; Tian et al., 2019).

We also adjusted for the gender of G3, recognizing its potentially significant influence on smoking behavior (Rodríguez-Planas & Sanz-de-Galdeano, 2019). While we were unable to account for the genders of G1 and G2, it's essential to note that the overall household smoking environment—where gender-related influences are inherently embedded—could still partially reflect the gender dynamics of these generations during their 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 Fig. 2.

3. Empirical methodology

We use a control function approach (CF) (Breen, 2018; Wooldridge, 2015) within a structural equation framework, employing a comprehensive set of individual equations. 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. We use probit regressions with average marginal effects in our empirical analysis. The system of equations is defined as follows.

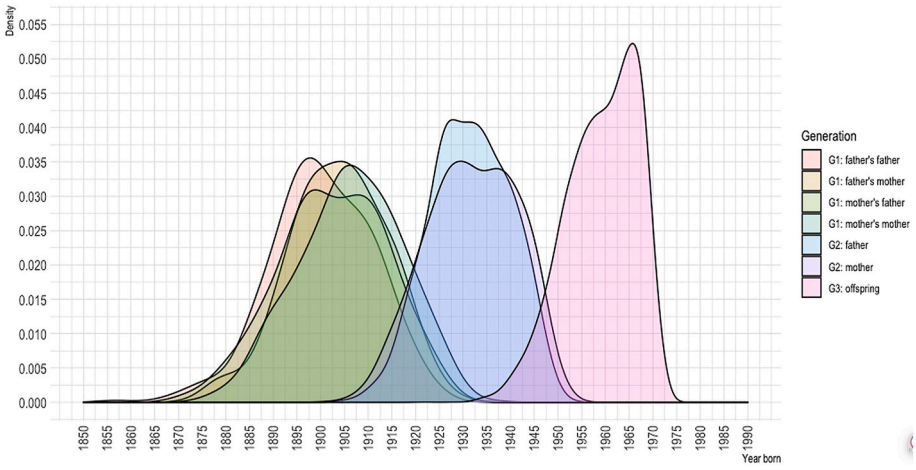

Fig. 2. Year-born density graph.
Note: The density graph illustrates the individuals' birth years in the study by generation. The first grandparent was born in 1825, the parent in 1900, and the offspring in 1922.

3.1. First-stage regression

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

$$P(S_1 = 1|Z_1) = \Phi(Z_1\alpha_1 + w) \quad (1)$$

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, 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 and 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 and Groman (2022)). 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. Given that G1 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 smoking behavior. For robustness, a sensitivity analysis was conducted using the continuous form of the CPI. A more comprehensive discussion of Z_1 can be found in Appendix A.2.

After estimating the equation, we predict $\widehat{S}_1$ from S_1 and derive the inverse Mills ratio (λ_1) or *IMR* for use in the subsequent regression stage (see, Appendix A.4). Incorporating the *IMR* into our study corrects non-random sampling of smoking behavior, ensuring our estimates accurately represent population dynamics (Heckman, 1979; Wooldridge, 2014, 2015).

3.2. 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:

$$P(S_2 = 1\ |S_1, X, Z_2, \lambda_1) = \Phi(S_1\beta_1 + X\beta_2 + Z_2\beta_3 + \lambda_1\beta_4 + v) \quad (2)$$

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. From Equation (3), we obtain the predicted probabilities ($\widehat{S}_2$) from S_2 and calculate the $IMR-2$ (λ_2) to introduce it in the third-stage regression (see, Appendix A.4).

In the second stage, we use 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 (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. 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 effects of G2 smoking behavior on the health of their G3 offspring. For a more in-depth discussion on the validity of this instrument, we refer readers to Appendix A.3.

3.3. Third-stage regression

Finally, we regress the adult G3 smoking behavior, S_3, on G1 and G2 smoking, a set of control variables for G3 (W), and the $IMR-1$ and -2 from the first and second stages. Additionally, we control for G2 recent smoking, $S_2^{'}$, which captures the smoking behavior of G2 at the time they participated in the Tromsø Study and serves as a contemporaneous control variable for G3 adult smoking.

$$P(S3 = 1\ |S_1, S_2, W, \lambda_1, \lambda_2) = \Phi\left(S_1\gamma_1 + S_2\gamma_2 + S_2^{'}\gamma_3 + W\gamma_4 + \lambda_1\gamma_5 + \lambda_2\gamma_6 + u\right) \quad (3)$$

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 ($\widehat{\gamma}_1$) and ($\widehat{\gamma}_2$) with robust standard errors and seek to elucidate the potential pathways between the smoking behavior of the G1 and G3. The parameter estimate ($\widehat{\gamma}_1$) represents the socio-emotional influence of G1 smoking behavior on G3 smoking behavior (Zeng and Xie, 2014; Zhang et al., 2021), after controlling for G2's influence and possible selection bias. As Zeng and Xie (2014) also underline, 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. To verify the effectiveness of our instrumental variables, we execute numerous tests and exhibit their results in Table 2.

To address the concerns about the methodological rigor and to provide additional validation of our results, we also employ an instrumental variables (IV) probit model as a robustness check (Angrist and Pischke, 2009; Wooldridge, 2010). This approach serves to validate the findings from our primary control function approach. We use the same instrumental variables, Z_1 for S_1 and Z_2 for S_2, in the IV regression model. The IV approach allows us to account for potential endogeneity and unobserved heterogeneity that might otherwise bias our estimates (Angrist and Krueger, 2001), and presented alongside our main findings to offer a comprehensive view of the relationships under study. This dual-method approach not only strengthens the validity of our findings (Cameron and Trivedi, 2009) but also allows for cross-method verification (Angrist and Pischke, 2009). It is important to note that our investigation into multigenerational transmission of smoking behavior employs causal methods to shed light on the underlying mechanisms. Nonetheless, the potential influence of unobserved characteristics on intergenerational smoking patterns still necessitates a cautious interpretation of results.

The indirect influence of G1 smoking on G3 smoking through G2 smoking is calculated using Sobel's product of coefficients approach (Sobel, 1982). The indirect effect is estimated by 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). This indirect effect, within the scope of this research, explicates the extent to which G1 smoking behavior is transmitted to G3 via G2 smoking. Additionally, we compute the total effect, incorporating both direct and indirect influences of G1 smoking on G3. The total effect equals the direct effect, γ_1, plus the indirect effect. To obtain robust standard errors for the indirect effect, we conduct a Monte Carlo simulation with 10,000 replications.

4. 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. Table 2 provides a comprehensive view of these influences, featuring results derived from control function (CF) and instrumental variable (IV) methods. These results are presented as average marginal effects alongside coefficients obtained from naïve ordinary least squares (OLS) regression to facilitate comparison across maternal and paternal lineages. Naïve OLS, despite their simplifying assumptions, are widely recognized for their interpretability

Table 2
Investigation of the influence of grandparental smoking on subsequent generations.

Third-step: G3 Smoking	Maternal lineage			Paternal lineage		
Variables	*Marginal effects*		Naïve *OLS*	*Marginal effects*		Naïve *OLS*
	CF	IV		*CF*	IV	
	(1)	(2)	(3)	(4)	(5)	(6)
Maternal G1 Smoking	−0.052** (0.020)		0.001 (0.013)			
Maternal G1 Smoking-hat		−0.039** (0.020)				
Paternal G1 Smoking				−0.013 (0.033)		0.035** (0.017)
Paternal G1 Smoking-hat					−0.533 (0.337)	
G2 Smoking	0.109*** (0.017)		0.111*** (0.016)	0.105*** (0.019)		0.104*** (0.018)
G2 Smoking-hat		0.635** (0.196)			0.506** (0.256)	
G2 Recent smoking	0.041*** (0.014)	0.069*** (0.013)	0.043*** (0.014)	0.057*** (0.018)	0.089*** (0.017)	0.060*** (0.017)
Control variables	Yes	Yes	Yes	Yes		Yes
IMR− 1	Yes			Yes		Yes
IMR− 2	Yes			Yes		
Observations	5725	5725	5725	4057	4057	4057
$_R2$			0.027			0.033
Akaike Inf. Crit.	7271.5	7306.1		5186.7	5219.5	
$Cov(\hat{w}, Z_1)$ [1]	0.011			0.005		
$Cov(\hat{v}, Z_2)$	−0.005			−0.006		
$Cov(\hat{w}, \hat{v})$	0.019			0.027		
$Cov(Z_1, Z_2)$	0.064			0.054		
$Cov(\hat{u}, \hat{w})$	0.002			0.035		
$Cov(\hat{u}, \hat{v})$	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		

Note: Table presents the marginal effects from the control function (CF) approach (columns (1) and (4)), alongside those obtained from the instrumental variable (IV) (columns (2) and (5)). Additionally, the coefficients from the naïve OLS regressions are presented in columns (3) and (6). Both maternal and paternal lineages are represented. *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)). In the maternal lineage column, G2 recent smoking refers to the mother's smoking behavior at the time of the survey. In the paternal lineage column, it refers to the father's recent smoking behavior. All models include control variables. 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. [1] *Cov*() stands for covariances. Covariances between residuals and two instrumental variables are close to zero, indicating the exogeneity of the instruments. The p-value of the likelihood ratio test indicates a better fit for the third-stage CF, including *IMR*− *1* and −*2*. 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.

and ease of understanding, especially when comparing marginal effects. Presenting the OLS estimates alongside the CF and IV analyses shows the discrepancy in estimating the G1 smoking effect due to uncontrolled endogeneity (Wooldridge, 2010, 2015).

In the maternal lineage, the CF model shows a statistically significant negative effect of G1 smoking on G3 smoking behavior, with a marginal effect of −0.052 (column (1)). The IV model for the maternal lineage similarly indicates a negative effect of G1 smoking on G3 smoking behavior, with a marginal effect of −0.039 (column (2)). Although there is a slight difference in the magnitude between the CF and IV models (−0.052 vs. −0.039), the direction of the effect remains consistent. Given our primary focus on the grandparents' influence, both models align in their findings and corroborate each other. These results suggest that maternal G1 smoking during the upbringing of G2 reduces the probability 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.

Besides, the smoking behavior of G2 plays a significant role in influencing G3 smoking behavior. The CF model shows that G2 smoking during G3's upbringing has a significant positive marginal effect of 0.109 (column (1)) on G3 smoking behavior. In parallel, the recent smoking behavior of G2, which represents G2's current smoking at the time of the survey, also exhibits a significant effect. Looking at the IV results, while we see the same level of significance for G1 smoking effect, the influence of G2 smoking on G3 smoking behavior presents differently across the two methods. In the maternal lineage, the IV approach yields a significant marginal effect of 0.635 for G2 smoking on G3 (column (2)), which is noticeably larger than the corresponding CF result of 0.109.

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 probability 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. Contrastingly,

the naïve OLS analysis shows a positive and statistically significant relationship between paternal G1 smoking and G3 smoking, reinforcing the importance of correcting for potential endogeneity. In the case of G2 smoking behavior, both lineages exhibit a strong, positive association with G3 smoking. Like the maternal lineage, the IV approach yields a significant and larger marginal effect for G2 smoking on G3 (column (5)) than the corresponding CF result (column (1). In the paternal lineage, G2 smoking plays a pivotal role in influencing G3's probability to smoke. As a result, both G2 smoking during G3's upbringing and their recent smoking at the time of the survey elevate the probability of G3 smoking, paralleling the patterns seen in the maternal lineage model.

In examining the effects across models, differential outcomes can be observed between the CF and IV methods. While both methodologies correct for endogeneity, ensuring a more robust comparison than the naïve OLS, they present different marginal effects of G2 smoking on G3 smoking behavior. The differences in magnitude between the CF and IV methods can be traced back to the inherent differences in their foundational assumptions and estimation techniques. Importantly, these differences are not attributed to variations in control variables or data structure since both approaches utilized the same dataset and set of controls. Rather than perceiving these differences as limitations, we consider them as offering a nuanced lens on the research question. The distinct outcomes between the CF and IV methods proffer alternative perspectives on the underlying mechanisms, underscoring the multifaceted nature of multigenerational transmission and the importance of methodological choice.

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 probability of bias due to endogeneity in the results for both lineage models.

Fig. 3 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 probability of G2 also smoking, which in turn increases the probability that G3 smokes. 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 probability 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 reverse it, resulting in an overall negative effect on the probability of G3 smoking. For the paternal lineage, the result 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.

Detailed results are available in Appendix A.5.

4.1. Robustness checks

4.1.1. The potential differential cohort effects across social groups

This robustness check explores 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 0.131 compared to 0.109 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 higher childhood economic conditions and the interaction between G2 smoking and G3's childhood 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 household economic conditions during childhood.

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 (0.142). 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.

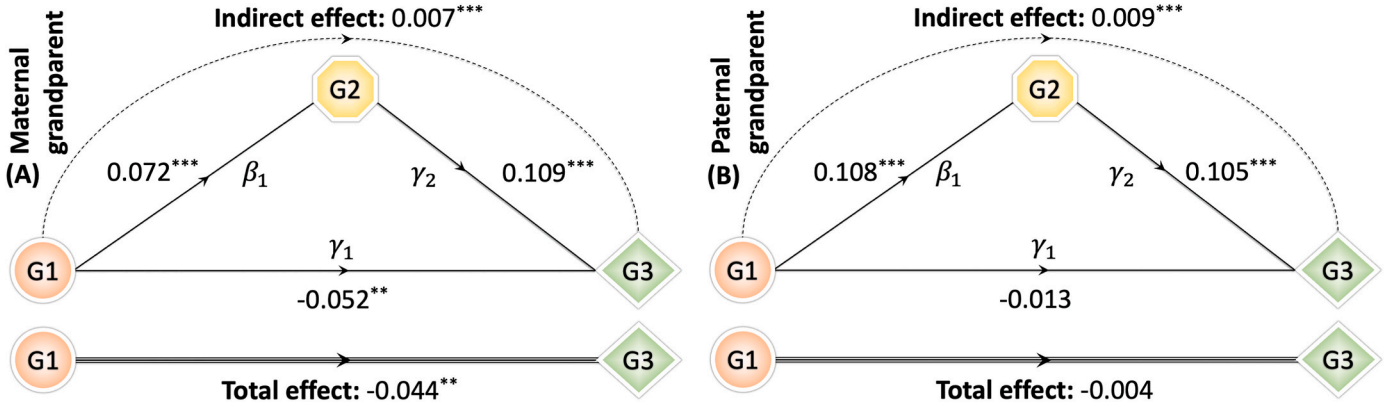

Fig. 3. Indirect and total effects of maternal and paternal grandparents on grandchildren.
Note: Figure (A) depicts the influence of smoking behavior during child-rearing by maternal grandparents (G1) on the smoking behavior of their grandchildren (G3), with the intermediate generation being the parents (G2). Similarly, Figure (B) represents the same relationship for paternal grandparents (G1) effect. Dashed lines in the figure indicate indirect effects, while the thickest lines at the bottom signify the total effects. The analyses show that the direct effect of G1 smoking is only significant for the maternal G1s. **p < 0.05; ***p < 0.01.

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	Paternal grandparents
	Marginal effects (CF)	*Marginal effects (CF)*
	(1)	(2)
Maternal G1 Smoking	−0.052** (0.020)	
Paternal G1 Smoking		−0.013 (0.033)
G2 Smoking	0.131*** (0.039)	0.142*** (0.053)
G2 Recent smoking	0.041*** (0.014)	0.058*** (0.018)
G3 Childhood economic conditions	−0.012 (0.036)	0.006 (0.050)
G2 Smoking X G3 Childhood economic conditions	−0.025 (0.040)	−0.041 (0.054)
Other control variables for G3	Yes	Yes
IMR− 1	Yes	Yes
IMR− 2	Yes	Yes
Observations	5725	4057
Akaike Inf. Crit.	7265.7	5188.2

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. *IMR − 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. Robust standard errors are shown in parentheses.
*** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

Table 4
Assessing the gender-specific effects of G1 and G2 smoking on G3 smoking.

Third-step: G3 Smoking	Maternal grandparents	Paternal grandparents
	Marginal effects (CF)	
	(1)	(2)
Maternal G1 Smoking	−0.045* (0.025)	
Paternal G1 Smoking		−0.005 (0.038)
G2 Smoking	0.109*** (0.017)	0.105*** (0.019)
G2 Recent smoking	0.041*** (0.014)	0.058*** (0.018)
G3 Female	−0.051** (0.021)	−0.037 (0.027)
Maternal G1 Smoking X G3 Female	−0.014 (0.027)	
Paternal G1 Smoking X G3 Female		−0.033 (0.033)
Other control variables for G3	Yes	Yes
IMR− 1	Yes	Yes
IMR− 2	Yes	Yes
Observations	5725	4057
Akaike Inf. Crit.	7265.8	5187.7

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. Robust standard errors are shown in parentheses.
*** Significant at the 1% level.
** Significant at the 5% level.
* Significant at the 10% level.

4.1.2. The gender-specific grandparental influences (G1)

This analysis helps us to 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 and Hertwig, 2010; Tu et al., 2021; Wang and 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 (−0.045 vs. −0.052) and is now significant only at the 10% level. The interaction term between maternal G1 smoking and G3 being female is not significant, suggesting that the intergenerational transmission of smoking behavior from the maternal G1 does not vary by the gender of the G3.

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 probability. Similar to the maternal lineage, the interaction term between paternal G1 smoking and G3 being female is not significant, suggesting no differential effect of paternal G1 smoking on granddaughters compared to grandsons.

In terms of control variables, the 'G3 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.

5. 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 underlying 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 the smoking behavior of maternal grandparents has a significant negative direct effect on their offspring. This impact persists even after controlling for parental smoking both during the upbringing of their children and the survey times, 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.

While our findings underscore the importance of grandparents, particularly from the maternal lineage, in influencing smoking behaviors, it is important to discuss the magnitude of this effect. The effect size from the maternal grandparents is smaller compared to the effects of the parental generation. In addition, while parents' smoking behavior during the upbringing of their children indicates a more long-term or sustained influence, the parent's recent smoking also underscores the significance of immediate parental behavior in shaping the smoking behavior of the subsequent generation. This suggests that while grandparents play a role, the immediate parental environment remains the most influential effect of smoking behaviors in the younger generation (Duarte et al., 2016). The smaller effect size from the grandparents, however, should not be overlooked. Moreover, the mere presence of an effect, regardless of its size, emphasizes the multi-generational nature of

health behaviors and the need to consider broader family dynamics in interventions and policy-making (Sari, 2023; Vandewater et al., 2014). As a result, the combined influence of both grandparents and parents underscores the complex origins of risky health behaviors like smoking. The influence of parents' smoking, regardless of generation, as Vandewater et al. (2014) say, has a significant effect on their children's smoking behaviors and that these behaviors are passed on from one generation to the next.

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.

The differences between paternal and maternal lineages in our findings indicate a matrilateral bias in the transmission of smoking behaviors, which could be derived from both evolutionary and socio-cultural factors. From an evolutionary perspective, the assured biological link mothers and maternal grandparents have with offspring can lead to increased influence on their health behaviors (Trivers, 1972). Socio-culturally, maternal grandparents often have a more hands-on role in upbringing due to traditional family dynamics and cultural norms (Coall and Hertwig, 2010). Socio-economic aspects, such as financial or emotional support, can further amplify this influence (Danielsbacka et al., 2011). However, with evolving family structures and gender roles, the distinction between maternal and paternal influences might be intertwined, such as a large degree of similarity in gender roles in contemporary Norway (Christiansen, 2014). The actual impact may hinge less on biological lineage and more on the depth of interaction between grandparents and grandchildren. Thus, while our findings suggest a matrilateral emphasis, it is important to frame this within both evolutionary and socio-cultural contexts.

This research is pivotal for two main reasons. Most significantly, our study illuminates the role of grandparents, which has been under-explored 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. Also, 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.

This nuanced understanding of grandparental investment and the evidence of matrilateral bias delineates a paradigm shift in the multigenerational transmission of health behaviors. Children, according to the social learning theory, tend to emulate the behaviors and attitudes they observe in their parents. However, our research 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 grandparent's 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).

5.1. 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). Health professionals can also 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. This grandparent-focused approach may complement existing parent-focused strategies, providing a more comprehensive and effective approach to reducing smoking in the younger generations. However, we acknowledge that the practical implications of the grandparents' influence might be limited, given the magnitude of the effect.

5.2. 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. Also, the geographical specificity of our sample - Northern Norway, Tromsø - might limit the generalizability of our findings. Therefore, caution must be taken when extrapolating our results to other geographical or cultural contexts. While the study provides data on the smoking behavior of parents who cohabited with their offspring during childhood, our data do not provide 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).

Future research can expand on our findings by incorporating a more diverse geographic and cultural sample (Sari, 2023), 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. It would be interesting to examine whether these factors could moderate or mediate the relationship between grandparental and grandchild smoking behaviors.

6. 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. The 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. 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.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used Grammarly and OpenAI in order to improve the readability and language of the manuscript. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

Declaration of competing interest

None.

Data availability

The authors do not have permission to share data.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2023.116339.

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