

# Geographical variation in cardiovascular disease mortality in Norway: The role of life course socioeconomic position and parental health

Hye Jung Choi<sup>a,\*</sup>, Marissa LeBlanc<sup>b,c</sup>, Tron Anders Moger<sup>d</sup>, Morten Valberg<sup>a,c</sup>, Christian M. Page<sup>e,f</sup>, Geir Aamodt<sup>g</sup>, Øyvind Næss<sup>a,h</sup>

<sup>a</sup> Department of Community Medicine and Global Health, Institute of Health and Society, University of Oslo, Oslo, Norway

<sup>b</sup> Oslo Centre for Biostatistics and Epidemiology, University of Oslo, Oslo, Norway

<sup>c</sup> Oslo Centre for Biostatistics and Epidemiology, Oslo University Hospital, Oslo, Norway

<sup>d</sup> Department of Health Management and Health Economics, Institute of Health and Society, University of Oslo, Oslo, Norway

<sup>e</sup> Centre for Fertility and Health, Norwegian Institute of Public Health, Oslo, Norway

<sup>f</sup> Department of Mathematics, The Faculty of Mathematics and Natural Sciences, University of Oslo, Oslo, Norway

<sup>g</sup> Department of Public Health Science, LANDSAM, Norwegian University of Life Science, Ås, Norway

<sup>h</sup> Norwegian Institute of Public Health, Oslo, Norway

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## ABSTRACT

Despite substantial geographical variation in cardiovascular (CVD) mortality within countries, little is known about whether this variation can be explained by individuals' life course socioeconomic position (SEP) or differences in family history of premature CVD deaths. Cox proportional hazards models were used to investigate the association between the county of residence at ages 50–59 and CVD death in Norwegians born between 1940 and 1959 and survived to at least age 60, using national data. Individual life course SEP and family history of premature CVD death reduced the geographical variation in CVD mortality across Norwegian counties, but some significant differences remained. Furthermore, CVD risk varied by residents' migration histories between two counties with distinct CVD and socioeconomic profiles.

## 1. Introduction

Cardiovascular diseases (CVD) account for 32% of all global deaths, and 17.9 million people die from CVD every year (WHO, 2021). CVD mortality has decreased in high-income countries in recent decades (Mackenbach et al., 2000; Roth et al., 2020), however, geographical variation in CVD mortality between regions persists in countries with strong welfare systems, such as the UK (Bhatnagar et al., 2016; Exeter et al., 2011), Finland (Karvonen et al., 2002), Sweden (Nerbrand et al., 1991), and Norway (Ariansen et al., 2020). This regional health disparity is a major public health concern.

Descriptive presentations of CVD mortality of regional differences are a common source of information for healthcare planners and health policy makers (WHO, 2021). However, it is not well known to what extent these differences reflect socioeconomic composition and other related factors among the individual residents. A common approach has been to adjust for individual socioeconomic position (SEP), e.g. attained education or occupational class, and assess to what extent this

attenuates the differences between regions (Lawlor et al., 2003; Næss et al., 2005; Smith et al., 1998). Yet, SEP used in most previous CVD studies on regional variation was measured in adulthood, despite the growing awareness of a life course perspective on health inequalities in spatial epidemiology (Jivraj et al., 2020; Johnson et al., 2012; Norman and Boyle, 2014; Norman et al., 2005).

There is extensive evidence that socioeconomic factors from the full life course are related to CVD mortality (Galobardes et al., 2006; Næss et al., 2004; Smith et al., 1997). For example, Galobardes et al. found an association between adverse childhood SEP and risk of CVD mortality in later life, independent of adulthood conditions (Galobardes et al., 2006). Furthermore, the social class of one's father was more strongly associated with increased CVD mortality than the adulthood social class (Smith et al., 1997). Although Kamphuis et al. found the reverse to Smith's study, childhood SEP was still important, being associated with SEP and unhealthy behaviour in adulthood, especially among those with low adulthood SEP (Kamphuis et al., 2012). This may suggest that adjusting for the composition of individual socioeconomic factors in

\* Corresponding author. Postboks 1130, Blindern, 0318, Oslo, Norway.

E-mail address: [h.j.choi@medisin.uio.no](mailto:h.j.choi@medisin.uio.no) (H.J. Choi).

adulthood may be inadequate.

A family history is also important for CVD risk. Genetic traits, health-related behaviours and lifestyles are shared within a family (Lloyd-Jones et al., 2004; Næss et al., 2012), and a number of studies showed a family history of CVD as an independent risk factor for both CVD incidence and mortality (Fiskå et al., 2015; Lloyd-Jones et al., 2004). Given that families are spatially clustered (Lappegård, 2009; Sørli et al., 2012), regional variation might arise from the clustering of those from the same families with high risk of CVD (Plümper et al., 2018b). However, this aspect has not been considered yet in studies on regional differences in CVD.

The regional clustering of health could also be related to domestic migration, as place of residence is influenced by the individuals' prior health status and SEP. Some may relocate to pursue better educational or employment opportunities, and healthy people from less affluent regions are likely to migrate to more prosperous regions (Norman et al., 2005). Conversely, individuals experiencing financial strain may move to more affordable locations, which can lead to selective domestic migration in a country (Holmager et al., 2021). Therefore, in regions with distinct cardiovascular disease features, it could be interesting to examine whether CVD risk would vary depending on domestic migration status.

In this study, we aimed to examine geographic variation in CVD mortality risk in counties of Norway, of Norwegians born between 1940 and 1959. We hypothesised that the regional variation would be smaller once individual life course SEP and parental premature CVD death were taken into account. In addition, if the regional variation persisted after adjustment for these factors, we investigated whether CVD mortality varied with different domestic migration histories in two counties with distinct CVD mortality and socioeconomic profiles.

## 2. Methods

### 2.1. Study population

We identified 1,386,442 Norwegians born from 1 January 1940 to 31 December 1959 in the Norwegian Population Registry. We followed our study population from age 60 years, excluding 264,842 individuals that died before that age. The remaining study population was linked to censuses to find residential history from birth to age 50–59. We excluded

those who lived in Svalbard at age 50–59 ( $n = 235$ ) due to their small number and few deaths, which yielded large uncertainty. We excluded those who were without residential information at age 50–59 ( $n = 16,200$ ), and those who were without parental information ( $n = 116,939$ ). Of the 1,006,226 study population, 3797 were immigrants [Fig. 1].

### 2.2. Outcome

The main outcome was CVD mortality, conditioned on survival to at least age 60, from January 1, 1991 to December 31, 2020 registered as the underlying cause of death in the Cause of Death Registry. CVD deaths were defined by European Shortlist for Cause of Death, coded 390–459 in the International Classification of Diseases (ICD) 9 and I00–I99 in ICD 10. We also included non-CVD deaths and all-cause mortality that occurred in the same period to consider a competing risk situation.

### 2.3. Exposure

The main exposure in this study was the county where individuals resided in the census conducted when they were 50–59 years of age, i.e. the most up-to-date residential information at inclusion. The rationale for selecting residential counties at aged 50–59 was based on the premise that at this age, most participants had resided in the same location for an extended period. Over half of Norwegians born between 1950 and 60 who migrated to bigger cities in their 20s, returned to their hometown by 34, with most migrations occurring within the same county (Sørli et al., 2012; Statistics Norway, 1995).

We used the former Norwegian county classification that was in official use from 1972 to 2018, comprising 19 counties. Changes in regional boundaries could make it difficult to compare other regions over time in longitudinal studies (Exeter et al., 2011). However, the county classification used in this study remained stable for the study period (Statistics Norway, 2020).

### 2.4. Covariates

#### 2.4.1. Life course socioeconomic position (SEP)

Education and income have been widely used for constructing SEP,

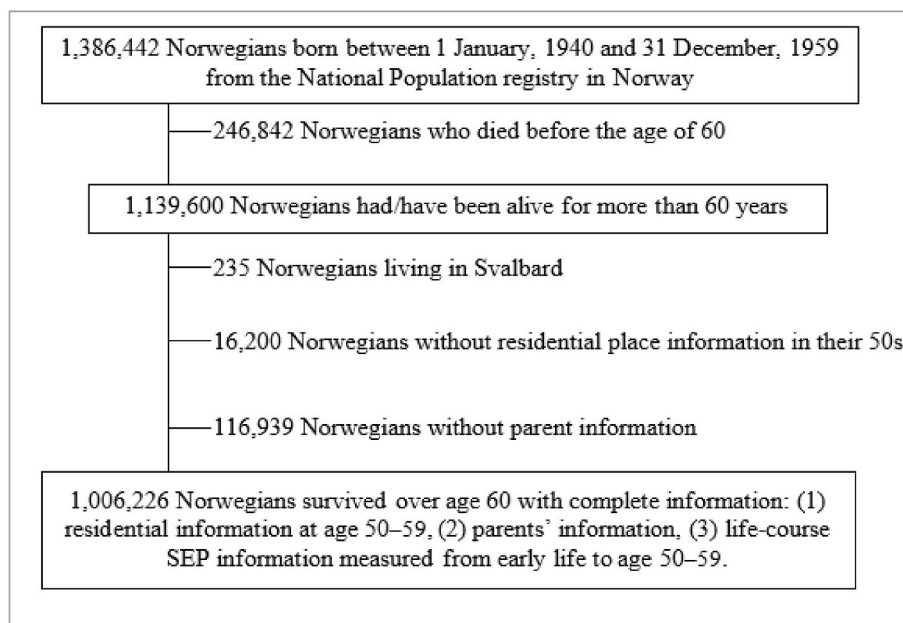


Fig. 1. Flow chart of the study population.

as these represent material and knowledge-related resources that affect individuals' behaviour and health outcomes (Galobardes et al., 2007; Katikireddi et al., 2020). Therefore, we used information on education from the National Educational Database, and income (housing conditions when income information was not collected) from six decennial censuses in 1960–2011 which had been repeatedly measured from early life (ranging from 0 to 20 years old) to the age 50–59 of the study population.

Given the diverse census times and a 20-year age span in the study population, housing conditions and income varied with time and age, consequently affecting their relationship with SEP. For instance, lacking access to an indoor water closet in the 1980s would be a strong indicator of poor SEP, but not in the 1960s. The absence of a telephone could indicate deprived housing conditions for individuals in their early 10s living with their parents, but it was common for those in their late teens and early 20s who had left their parents' home within the same census. Similarly, income levels were subject to inflation and seniority.

Therefore, to construct comparable SEP scores that were comparable across time and age groups for every individual, a standardisation was conducted. First, we categorised individuals as having low SEP if their income (household income in the 1990 and 2001 censuses, and individual income in the 2011 census) belonged to the lowest tertile among those of the same age in the same census from 1990 to 2011. An individual  $i$  receives a score  $P_{i,k} = 1$  if classified with low SEP in the  $k$ th census, whereas  $P_{i,k} = 0$  for middle and high SEP.

A similar method was applied to housing conditions in the earlier three censuses in 1960–1980 where income information was not available. We gave 0 point for each decent state and 1 point for a deprived state to each of six housing conditions: indoor water closet (0: indoor water closet, 1: not), indoor bath (0: indoor bath, 1: not), telephone (0: owned, 1: not), house ownership status (0: owned, 1: renting), house type (0: detached house, 1: apartments/other types), room per household capita (0: room per person  $\geq 1$ , 1: room per person  $< 1$ ). Then, we classified individuals with low SEP ( $P_{i,k} = 1$ ) if their total points of these housing conditions, ranging from 0 to 6, belonged to the highest tertile compared to individuals of the same age in the same census  $k$ .

Second, we summed these SEP scores ( $P_{i,k}$ ) from all censuses into a life course SEP score ( $S_i$ ), by dividing  $\sum_{k=1}^{n_i} P_{i,k}$  by the number of censuses ( $n_i$ ) that one participated in. Since we measured SEP from early life to age 50–59, the maximum of  $n_i$  was 6.  $S_i$  is thus given by  $S_i = \frac{1}{n_i} \sum_{k=1}^{n_i} P_{i,k}$ .

Lastly, we categorised three life course SEP groups based on this standardised  $S$  score (high life course SEP:  $S_i = 0$ , middle life course SEP:  $0 < S_i \leq 0.5$ , low life course SEP:  $S_i > 0.5$ ). For instance,  $S_i$  score  $> 0.5$  indicates that an individual  $i$  spent more than half of their lives with low SEP during the  $n_i$  censuses. Average propensity scores of housing conditions by life course group per census are provided in [STable 1](#). The mean scores are consistently lower by increasing SEP category across all housing variables, indicating that life course SEP groups are clearly separated using the approach above.

The highest level of attained education in the National Educational Database at age 50–59 was included as a separate independent variable. Those with none or completing only primary and lower secondary education were categorised as having a low education level, while any other higher education was categorised as high.

#### 2.4.2. Family history of CVD: premature CVD death in parents

We used premature CVD death of parents (age  $< 65$ ) of the study population to define family history of CVD. Parental relationships were built from the multigenerational database of the Norwegian Family Based Life Course Study, and those parents were linked to the Cause of Death Registry to identify their CVD death (ICD9: 390–459, ICD10: I00–I99).

## 2.5. Statistical analyses

Cox proportional hazards regression models with age as the time scale were performed to investigate the association between the county of residence at ages 50–59 and CVD mortality. The study population were followed from the day when they turned the age of 60 to CVD death, emigration, or the end of the study period (31 December 2020), whichever occurred first.

Model 1 included sex, birth year and county of residence at 50–59 years. Oslo, which has been the most populous county in Norway, was used as the reference. Model 2 consisted of the life course SEP (life course SEP group and education) and parental premature CVD death variables plus Model 1. To assess whether geographic variability in risk of CVD death was reduced after adjusting for life course SEP and premature CVD death in parents, we studied the change in hazard ratios (HRs) for the counties compared to Oslo between Model 1 and 2, and the standard deviation of the coefficients across the models. The regional risk of CVD mortality could be more prominent for those who lived in the same county for their entire life, thus, we ran Models 1 and 2 among those who resided in a county throughout their life as a subset analysis.

In addition to this, Models 3a and 3b were employed to investigate whether CVD mortality varied by different domestic migration histories (lifelong residents, emigrants, immigrants, and returners) among those who reside or have resided in counties with distinct features on SEP and CVD mortality. Model 3a included domestic migration history adjusted for sex and birth year, and Model 3b included life course SEP and parental premature CVD deaths plus Model 3a. These Models 3a and 3b were conducted separately in two counties with consistently higher (Finnmark) or lower (Akershus) risk of CVD mortality than Oslo. Akershus is known for the highest average income level among counties since 1960 and high life expectancy while Finnmark for the lowest income level most of the time and higher CVD mortality (Ariansen et al., 2020; Statistics Norway, 1962, 1982).

Clustered standard errors at a parent level were used in all models, to account for within-family dependence among the study population. The proportional hazard assumptions of all Cox regression models were checked by plotting Schoenfeld residuals, and we did not find a strong deviation against the assumption. All statistical and sensitivity analyses were done using Stata/MP 16.1.

## 2.6. Sensitivity analyses

To visualise the differences in CVD mortality incidence among 4 regions over 19 counties, we fitted cause-specific cumulative incidence functions (CIF) (Lambert, 2017). This was done for randomly selected 30% ( $n = 301,868$ ) of the study population, due to the large size of the datasets. We also fitted Models 1 and 2 for non-CVD deaths and all-cause mortality outcomes to consider a competing risk situation in the study population. Additionally, sensitivity analyses were conducted using SEP score ( $P_{i,k}$ ) from each census, and income as a continuous measure, to investigate the relationship with CVD mortality.

## 3. Results

### 3.1. Descriptive statistics

In the study population including 1,006,226 participants, the average birth year was 1950 and female participants accounted for 49% [[Table 1](#)]. The median follow-up time was 9.2 years, and 2.1% of the population died due to CVD during the follow-up. Individuals with low life course SEP, and those with a low education level accounted for 19.2%, and 22.8% of the population, respectively. Oslo and its neighbouring county, Akershus, were the counties where the highest proportion of the study population had lived in their 50s. Over 7% of the study population had parents who died before the age of 65 due to CVD. In sum, the study population came from 536,410 different parents.

**Table 1**

Descriptive statistics of 1,006,226 Norwegians who were born in 1940–1959 and survived over 60 years old.

		N (%)	
The average birth year ( $\pm$ SD)		1950 ( $\pm$ 5.4)	
Sex	Male	516,126 (51)	
	Female	490,100 (49)	
Follow-up years from age 60 (25th – 75th percentile)		9.2 (4.9–13.8)	
Deaths by the end of 2020	All-cause mortality	104,935 (10.3)	
	- CVD death	21,624 (2.1)	
	- Non-CVD death	83,311 (8)	
The average age of death ( $\pm$ SD)	All-cause mortality	68.5 (5.0)	
	- CVD death	68.6 (5.1)	
	- Non-CVD death	68.5 (5.0)	
Life course SEP group	High	211,342 (21)	
	Middle	601,551 (59.8)	
	Low	193,333 (19.2)	
Education level	High (high school, tertiary)	776,973 (77.2)	
	Low (none, primary, lower secondary)	229,253 (22.8)	
Premature CVD death (< age 65) in parents	None	927,031 (92.1)	
	One parent	77,664 (7.7)	
	Both parents	1531 (0.2)	
The number of families (parents) in the study population		536,410	
Residential county at age 50–59 (current county classification)		N (%)	Lifelong resident (%*)
South-Eastern	Østfold (Viken)	59,782 (5.9)	30,480 (51*)
	Akershus (Viken)	107,848 (10.7)	16,404 (15*)
	Oslo	92,311 (9.2)	30,595 (33*)
	Hedmark (Innlandet)	45,947 (4.6)	18,682 (41*)
	Oppland (Innlandet)	43,943 (4.3)	18,740 (43*)
	Buskerud (Viken)	55,994 (5.5)	22,364 (40*)
	Vestfold (Vestfold and Telemark)	51,246 (5.1)	22,649 (44*)
	Telemark (Vestfold and Telemark)	39,116 (3.9)	15,956 (41*)
	Aust-Agder (Agder)	24,227 (2.4)	6603 (27*)
	Vest-Agder (Agder)	33,294 (3.3)	12,247 (37*)
Western	Rogaland	79,221 (7.8)	31,627 (40*)
	Hordaland (Vestland)	95,210 (9.4)	52,653 (55*)
	Sogn and Fjordane (Vestland)	23,933 (2.4)	11,537 (48*)
	Møre and Romsdal	56,857 (5.6)	27,613 (49*)
Middle	Sør-Trøndelag (Trøndelag)	60,352 (6)	27,140 (45*)
	Nord-Trøndelag (Trøndelag)	30,358 (3)	11,162 (37*)
	Nordland	55,484 (5.5)	25,476 (46*)
Northern	Troms (Troms and Finnmark)	35,140 (3.5)	17,812 (51*)
	Finnmark (Troms and Finnmark)	15,963 (1.6)	8667 (51*)

\*% indicates the proportion for lifelong residents, who were born in a county and lived there until the ages 50–59 without any migration records, of the total number of people who lived in the same county in age 50–59 in the study population.

### 3.2. Regional difference in CVD mortality risk

In the birth year and sex-adjusted model (Model 1), those who lived in the Northern part of Norway in their 50s experienced a higher CVD mortality than those in Oslo [Fig. 2, STable 2]. Conversely, counties located in the Western part of Norway and Akershus seemed to have relatively lower CVD mortality. In Model 2 adjusting for the life course SEP and premature CVD death in parents, the estimated CVD mortality in different counties changed slightly, ranging from –11% to 8% ( $\Delta$ ) compared to Model 1. Compared to Oslo, living in the northernmost county, Finnmark, was associated with increased CVD mortality (HR:

1.18, 95% CI: 1.07–1.31), whereas five counties in the Southern part had around 13% lower mortality (for example, Akershus HR: 0.87, 95% CI: 0.82–0.93; Aust-Agder HR: 0.86, 95% CI: 0.78–0.96). The standard deviation of the estimated HRs across counties in Model 1 was 0.114, and it decreased to 0.071 in Model 2. Hence, adjusting for life course SEP and premature CVD death in parents reduced the variation of CVD mortality risk across counties by 38%.

The average percent of lifelong residents across 19 counties was 42%, while Oslo (33%) and its former neighbour county, Akershus (15%), had the lowest [Table 1]. In Model 2 for the sub-population of lifelong residents, living in Finnmark for the entire life was associated with increased CVD mortality (HR: 1.16, 95% CI: 1.01–1.33) compared to living in Oslo [Table 2]. Yet, lifelong residents in Akershus were at lowered risk (HR: 0.90, 95% CI: 0.80–1.02) compared to Oslo's lifelong residents. Similar to the main analysis above, the adjustment for life course SEP and parental premature CVD death decreased the variation of estimated HRs across counties by 33%, from 0.113 in Model 1 to 0.075 in Model 2.

We compared prior and current residents with different domestic migration histories in Models 3a and 3b in two counties; Akershus with low HRs, and Finnmark with high HRs across the models [Fig. 3]. Interestingly, those with the same migration type in these two counties showed opposite results in Model 3b; Finnmark-born emigrants living in other counties at age 50–59 had lower CVD deaths (HR: 0.70, 95% CI: 0.58–0.85) compared to Finnmark's lifetime residents, but emigrants from Akershus were likely to have a higher CVD mortality (HR: 1.09, 95% CI: 0.94–1.26) compared to lifelong residents of Akershus. Furthermore, those who immigrated to Akershus had lowered CVD mortality (HR: 0.88, 95% CI: 0.78–0.99) than lifelong residents of Akershus.

### 3.3. Sensitivity analyses

The Northern region had higher incidences of CVD mortality compared to the other three regions in the crude cause-specific cumulative incidence functions (CIF) outcome [SFig. 1, upper]. However, there were no remarkable differences between regions in non-CVD mortality [SFig. 1, below].

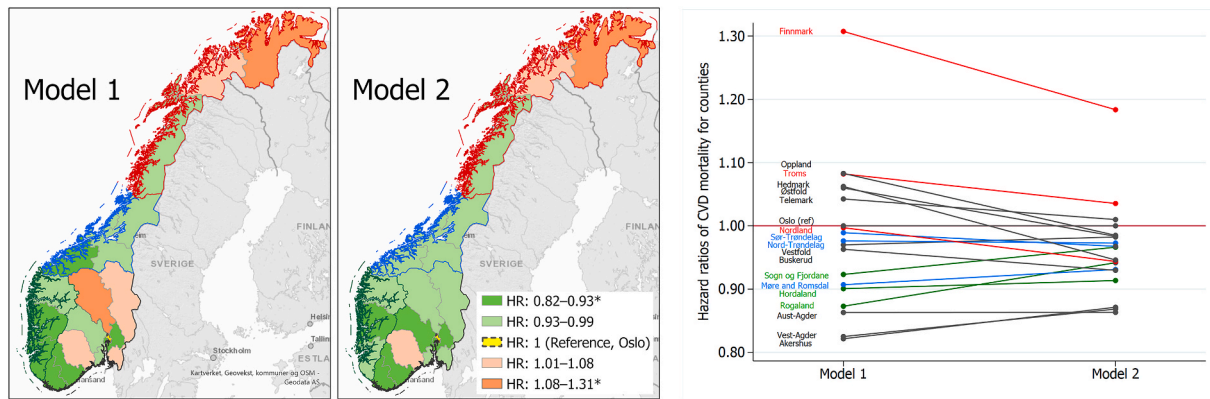
Almost all counties except a few in the South-Eastern part were associated with lower non-CVD and all-cause mortality than Oslo in Model 1 and Model 2 [STable 3]. Two Agder counties, with HR below 1 for CVD mortality in previous analyses, did not have HR below 1 for non-CVD and all-cause mortality compared to Oslo. Finnmark, which had consistently higher HRs of CVD death before, had higher HRs of all-cause mortality (HR: 1.06, 95% CI: 1.01–1.11), and non-CVD deaths (HR: 1.03, 95% CI: 0.97–1.08) in Model 2. Adjustment for life course SEP and parental premature CVD death in Model 2 reduced the non-CVD death variation across counties by 14%, from 0.078 in Model 1 to 0.067 in Model 2. The attenuation of adjustment for the factors was larger in all-cause mortality models, as the variation across counties decreased by 25% from 0.077 to 0.058.

When using SEP score ( $P_{i,k}$ ) from each census, low SEP (score = 1) across the censuses were consistently associated with higher CVD mortality [STable 4]. We also found an inverse association between income as a continuous measure and CVD mortality [data not shown].

## 4. Discussion

### 4.1. Principal findings

Our main result showed how the risk of CVD mortality in the 19 counties in Norway changed after taking into account individual-level factors known to be associated with CVD such as life course SEP and premature parental CVD death. In doing so we showed that some parts of regional health inequalities can be explained by these. The attenuation in the variation across counties was larger in CVD mortality than in non-



**Fig. 2.** Illustration of HRs of CVD mortality for living in a county when individuals were in their 50s (left, middle), and the changes in the variation of HRs across the counties (right) between models, visualising the outcomes from [Table 2](#).

\* indicates statistically significant HRs, with p-value <0.05. Red boundary/lines indicate the northern region, blue for the middle, green for the western, and black for the south-eastern region.

**Table 2**

The estimated HRs of CVD mortality in 19 counties for the subsamples (n = 408,407 with CVD deaths = 9051) of the study population, those who lived in the same county for their entire life.

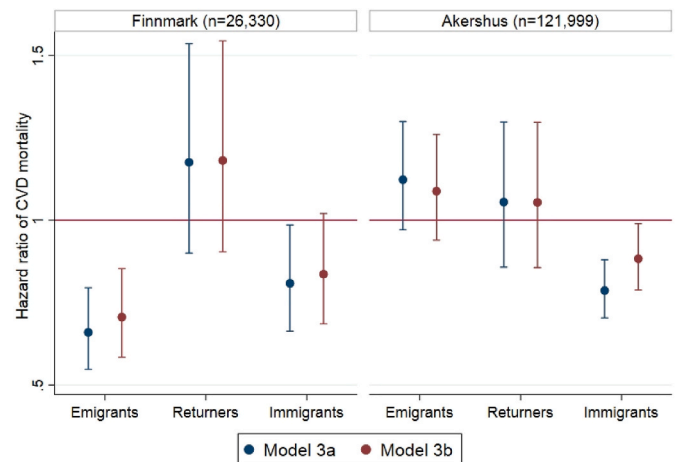
	Model 1 <sup>b</sup>	Model 2 <sup>c</sup>	Δ% <sup>d</sup>
County of residence for lifelong			
South-			
Eastern			
Østfold	1.00 (0.90–1.11)	0.96 (0.86–1.06)	-5
Akershus	0.90 (0.79–1.01)	0.90 (0.80–1.02)	1
Oslo (ref)			
Hedmark	0.99 (0.88–1.11)	0.89 (0.79–0.99) <sup>a</sup>	-10
Oppland	1.00 (0.89–1.13)	0.93 (0.83–1.05)	-7
Buskerud	0.96 (0.86–1.08)	0.94 (0.84–1.05)	-3
Vestfold	0.91 (0.81–1.02)	0.93 (0.82–1.04)	2
Telemark	1.02 (0.90–1.16)	1.00 (0.88–1.14)	-2
Aust-Agder	0.86 (0.71–1.04)	0.85 (0.70–1.03)	-1
Vest-Agder	0.75 (0.64–0.87) <sup>a</sup>	0.82 (0.70–0.96) <sup>a</sup>	10
Western			
Rogaland	0.86 (0.77–0.95) <sup>a</sup>	0.95 (0.85–1.05)	10
Hordaland	0.86 (0.78–0.94) <sup>a</sup>	0.88 (0.80–0.97) <sup>a</sup>	3
Sogn and Fjordane	0.82 (0.71–0.95) <sup>a</sup>	0.89 (0.77–1.04)	8
Middle			
Møre and Romsdal	0.80 (0.72–0.90) <sup>a</sup>	0.85 (0.76–0.95) <sup>a</sup>	6
Sør-Trøndelag	0.97 (0.87–1.07)	0.94 (0.85–1.05)	-3
Nord-Trøndelag	0.89 (0.77–1.03)	0.91 (0.79–1.06)	3
Northern			
Nordland	0.91 (0.81–1.01)	0.88 (0.79–0.98) <sup>a</sup>	-3
Troms	1.03 (0.92–1.17)	1.00 (0.89–1.13)	-4
Finnmark	1.26 (1.09–1.45) <sup>a</sup>	1.16 (1.01–1.33) <sup>a</sup>	-8

<sup>a</sup> indicates statistically significant HRs, with p-value <0.05.  
<sup>b</sup> Model 1 included the county of residence in the age 50s, adjusted for sex and birth year.  
<sup>c</sup> Model 2 included parental premature CVD deaths and life course SEP (life course SEP group and education) plus Model 1. Clustered standard errors were used in all models, to account for within-family dependence.  
<sup>d</sup> Δ%: percentage changes in the estimates of HRs from Model 2 compared to those from Model 1, calculating from  $[(\beta_{Model 1} - \beta_{Model 2})/\beta_{Model 1} * 100]$ .

CVD and all-cause mortality analyses. We also found the risk of CVD mortality varied by different domestic migration history among those who reside or have resided in two counties; one with the highest average income level and the other with the lowest.

4.2. Comparison with previous studies

Geographic variation in CVD was found in other high-income



**Fig. 3.** CVD mortality risk in pre- and current residents of Akershus and Finnmark, by their domestic migration history in Model 3a and 3b. The red horizontal line indicates the reference group (those lifelong residents) in each of the two counties. Emigrants mean those born in one of these two counties but not lived in their home county at the age of 50–59, returners were those born in one of these counties and lived in their home county at the age of 50–59 after living in other places at earlier ages, and immigrants were those born in other counties resided in one of these counties in the age of 50–59. Model 3a included domestic migration history (lifelong, emigrants, returners, immigrants), adjusted for sex and birth year. Model 3b included parental premature CVD deaths and life course SEP (life course SEP group and education) plus Model 3a. Clustered standard errors were used in all models, to account for within-family dependence.

countries with strong welfare systems ([Havulinna et al., 2008](#); [Lawlor et al., 2003](#); [Morris et al., 2003](#)). For example, a “north-south gradient” still existed in Britain after adjusting for established CVD risk factors, as those who lived in Scotland had a higher incidence of CVD compared to those in South England ([Lawlor et al., 2003](#); [Morris et al., 2001](#)). Moreover, the CVD burden in Scotland remained high despite the large reduction in CVD mortality from 1980 to 2013 in the UK, and this indicated that CVD burdens between the countries of the UK were not equally improved ([Bhatnagar et al., 2016](#)). Similarly, all Norwegian counties had increased life expectancy both for men and women from 1990 to 2019, and the different life expectancies between counties became smaller over the period ([Clarsen et al., 2022](#)). However, we found that CVD deaths that occurred during a similar period (1991–2020) were associated with the county of residence in age 50–59,

which indicated that regional inequalities in CVD deaths remained.

The regional variation found in this study was similar to previous studies demonstrating adverse health outcomes in northern Norway (Clarsen et al., 2022; Syse et al., 2018). These studies have found that inhabitants in northern Norway had a lower life expectancy than in other counties, especially for men, and this inequality existed even with the general health improvement among the Norwegian population (Clarsen et al., 2022; Syse et al., 2018). Regional inequality in CVD mortality also persists in Norway, although overall CVD mortality has decreased over decades. The northern part of Norway, particularly Finnmark, has had a higher standardised CVD mortality than other regions since 2005, while CVD mortality in the other regions was relatively similar to each other (Ariansen et al., 2020; Borgan and Pedersen, 2007).

The persistent geographical variation in CVD could be attributed to unadjusted factors associated with CVD that were not included in our analysis. Inequalities in health between regions have often been related to the distinct characteristics of inhabitants in regions in terms of compositional explanation (Kravdal et al., 2015; Morris et al., 2001). Inhabitants of Finnmark have traditionally had the least favourable composition of CVD risk factors (Clarsen et al., 2022; Vedøy and Sæbø, 2022), and the highest CVD mortality (Ariansen et al., 2020). As Morris et al. showed that geographic variation in the incidence of coronary heart disease in Britain could be explained by established CVD risk factors (Morris et al., 2001), including further individual information on these traditional CVD risk factors could further reduce the geographical variation in CVD mortality. In addition, there are geographical variations in the physical environment that potentially could explain parts of the remaining geographical variation in CVD mortality. For example, the northernmost countries have harsher weather conditions, with e.g. heavier snowfalls, that have been associated with higher incidence of myocardial infarctions in older north Norwegians over 65 years old (Hopstock et al., 2012).

Most previous studies have focused on the role of regional socioeconomic conditions, and they showed that geographical health inequalities were largely attenuated when the socioeconomic characteristics of regions were included (Kravdal et al., 2015; Plümper et al., 2018a, 2018b). Compared to these studies observing a 70–80% reduction, the extent of the reduction in this study was smaller as of around 38%. This small reduction in the variation could be related to the choice of regional level in this study. Administrative districts may not appropriately reflect the socio-geographic context of residing individuals (Merlo et al., 2013), and a county consists of more heterogeneous features than a small areal level, for example, a municipality or neighbourhood. Furthermore, the different extents of reduced variation between this study and others could arise from the outcomes of interest. While ours focused on CVD, the other studies analysed the regional inequalities in all types of death. Nevertheless, we showed that individual life course SEP and premature parental CVD death reduced the regional CVD mortality variation, accordingly, and regional health inequalities can be partly explained by these individual factors.

Simultaneously, we found that selective migration might contribute to those differences in risk of CVD mortality between counties. The risk of CVD mortality varied among residents by domestic migration history in Akershus and Finnmark, even after life course SEP and premature CVD death in parents were considered. Our finding indicated the selective migration that healthy people from less affluent areas migrate to affluent areas while the unhealthy stay in less affluent areas or move to least affluent areas (Norman et al., 2005). Similar selective migration was found in Denmark (Holmager et al., 2021) and in the UK (Norman et al., 2005), which aggravated geographical inequalities in health.

Regarding the association between life course SEP and CVD mortality, our finding (data not shown) was consistent with previous research that found the greater impact of cumulative life course SEP on CVD mortality, than on non-CVD and all-cause mortality (Katikireddi et al., 2020; Næss et al., 2004; Smith et al., 1997). In a UK study using a

cumulative measurement of social classes in three different life stages, the association between cumulative SEP and CVD mortality was stronger than that of all-cause or other cause-specific mortality, even after adjusting for traditional risk factors (Smith et al., 1997).

We found a strong association between parental premature CVD deaths and CVD mortality in the study population (data not shown). Compared to those with non-premature CVD death in parents, CVD mortality was 2.43 times higher for those with both parents having premature CVD deaths, and 49% higher for those with one parent having premature CVD death. This finding corresponded to other studies that showed individuals with CVD in their parents had higher risk of CVD than those without family history of CVD (Fiskå et al., 2015; Lloyd-Jones et al., 2004).

#### 4.3. Strengths and weaknesses of this study

This study used national databases and registries to investigate CVD mortality risk in 19 Norwegian counties at the individual level, taking both life course SEP and history of premature parental CVD death into account in the analyses. Individual life course SEP has been associated with risk of CVD (Galobardes et al., 2006; Næss et al., 2004; Smith et al., 1997) and place of residence through selective migration (Holmager et al., 2021; Norman et al., 2005). In spite of this, the possible effects of SEP have not been comprehensively examined in prior research on regional CVD variation. Also, we showed to what extent different regional variations in health were explained by individual-level factors, while most studies on regional health disparities are interested in socio-demographic differences at the regional level based on aggregated data.

It could be questioned how well housing conditions in 1960–1980 represented individual SEP and how adequate our SEP calculation was as a proxy for income when income information was not available. The same data and approach have been used in previous CVD research with the life course perspective in Norway (Degerud et al., 2018; Næss et al., 2004), and this method seems to indicate the different features of SEP groups on CVD outcomes well. We refined the method used in these previous studies by using age-specific distribution for each housing condition and income to identify low SEP individuals. Thus, it is likely that our revised approach may capture SEP with more precision compared to these earlier studies.

Furthermore, we have investigated the association between each SEP score ( $P_{i,k}$ ) from different time points and CVD mortality. Notably, the HRs of low SEP based on income from 1990 onwards were stronger compared to those based on housing conditions from 1960 to 1980. This difference could be attributed to variations in the precision of representing individual SEP between housing conditions and income, or the different impacts of SEP on CVD mortality across different life stages. It is worth mentioning that using the same SEP measurement over time would have provided more reliable results; however, the income data for the years 1960–1980 was not available. If other socioeconomic information, such as occupation or employment status, had been available in our datasets, it could have been incorporated in our life course SEP variable.

Smaller geographic units such as municipalities may capture more pronounced variations compared to our analysis at the county level, however, we chose the county level due to well-known variations in socioeconomic conditions and health between counties as studied in previous literature. Nonetheless, future studies may benefit from delving into internal differences within counties or exploring variation at an even smaller geographical level.

## 5. Conclusion

Individual life course SEP and family history of premature CVD death partly explained the geographical variation in CVD mortality across Norwegian counties. The differences in CVD mortality between counties

were attenuated after adjusting for these two factors, however, a few counties had notable differences in CVD mortality compared to other counties. There was an association between domestic migration status and CVD mortality, which could contribute to these differences. The findings may have implications for interpreting regional variation in CVD mortality in terms of the role of individual CVD-relevant factors which are geographically clustered. Further, the findings indicate the underlying inequalities which might have been overlooked due to the recent decreasing trend of CVD mortality in high-income countries.

### Ethics approval

This project obtained ethical approval [REK 2012/827] from the Regional Committees for Medical and Health Research Ethics, Norway.

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### Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: M. LeBlanc has served as speaker for MSD outside the submitted work.

### Data availability

The authors do not have permission to share data.

### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.healthplace.2023.103095>.

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