

Short-term association between air temperature and mortality in seven cities in Norway: A time series analysis.

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Title page

Title: Short-term association between air temperature and mortality in seven cities in Norway: A time series analysis.

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Abstract

Background: The association between ambient air temperature and mortality has not been assessed in Norway. This study aimed to quantify for seven Norwegian cities (Oslo, Bergen, Stavanger, Drammen, Fredrikstad, Trondheim and Tromsø) the non-accidental, cardiovascular, and respiratory diseases mortality burden due to non-optimal ambient temperatures.

Methods: We used a historical daily dataset (1996-2018) to perform city-specific analyses with a distributed lag non-linear model with 14 days of lag, and pooled results in a multivariate metaregression. We calculated attributable deaths for heat and cold, defined as days with temperatures above and below the city -specific optimum temperature. We further divided temperatures into moderate and extreme using cut-offs at the 1st and 99th percentiles.

Results: We observed that 5.3% (95%CI 2.0-8.3) of the non-accidental related deaths, 11.8% (95%CI 6.4-16.4) of the cardiovascular and 5.9% (95%CI -4.0-14.3) of the respiratory were attributable to nonoptimal temperatures. Notable variations were found between cities and subgroups stratified by sex and age. The mortality burden related to cold dominated in all three health outcomes (5.1%, 2.0-8.1, 11.4%, 6.0-15.4, and 5.1%, -5.5-13.8 respectively). Heat had a more pronounced effect on the burden of respiratory deaths (0.9%, 0.2-1.0). Extreme cold accounted for 0.2% of non-accidental deaths, and 0.3% of cardiovascular and respiratory deaths, while extreme heat contributed to 0.2% of nonaccidental to 0.3% of respiratory deaths.

SL-SONIC DISCOVER **Conclusions**: Most of the burden could be attributed to the contribution of moderate cold. This evidence has significant implications for enhancing public-health policies to better address health consequences in the Norwegian setting.

Keywords:

Climate

Norway

Cold Temperature / adverse effects* Hot Temperature / adverse effects* Cardiovascular Diseases / mortality Respiratory Tract Diseases / mortality Humans

Mortality*

Risk Assessment / methods

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Introduction

Norway is experiencing increasing temperatures and evident effects of extreme weather events (1, 2). Particularly, the Artic region has seen a steep increase, warming three times faster than the global average (3). The country has distinct differences between urban and non-urban areas and a wide range of climatic zones, with its population steadily moving to the cities and gradually ageing (4). This suggests a heightened vulnerability to climate change as older populations are particularly susceptible to thermal stress. Consequently, there is a pressing need to comprehend the effects of temperature on mortality.

The leading cause of death in Norway is ischaemic heart disease, with 104.53 deaths per 100 000 population in 2018, followed by dementia and chronic obstructive pulmonary disease (COPD) (5). One in five Norwegians live with a confirmed cardiovascular disease (CVD) or are at a high risk of developing one (6), and around 6% of the population over 40 years of age has been diagnosed with COPD (7).

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promises the pulmonary mec CVD and respiratory diseases are particularly affected by non-optimal temperature. Exposure to cold air temperature compromises the pulmonary mechanics, triggers vasoconstriction and may lead to an increase in cardiovascular and respiratory mortality (8, 9). Hot temperatures trigger a widening of blood vessels (vasodilation), the rate at which blood pumps increases, and sweating, which may originate in dehydration, and a series of pathological events that can result in a cardiovascular impairment (10) and induce airways inflammation or bronchoconstriction (11).

Specific research on the effects of extreme temperatures on mortality in Nordic cities is still limited. Some examples of recent studies in Nordic include Åström et al. (12), Orru et al. (13) and Fonseca et al. (14). Few studies on heat effects in Sweden and Finland have found significant excess morbidity and mortality, especially among older people, and mainly attributable to cardiovascular and respiratory diseases (15-17). According to (16), resilience to heat may have decreased over the last two decades, along with the increase in temperature and ageing population.

In this study, we aim to assess the temperature related mortality burden in seven urban municipalities in Norway for different causes of death in the 1996-2018 period. We focus on non-accidental and cause-specific mortality due to cardiovascular and respiratory diseases and stratify by sex and groups of age (18). To our knowledge, this is the first study of its kind in Norway.

Data

We chose the seven largest cities in Norway by their population in 2018. These are in decreasing order: Oslo (673 469 inhabitants), Bergen, Trondheim, Stavanger, Drammen, Fredrikstad and Tromsø (75 638). The cities represent three different climates in the Köppen-Geiger climate classification: Humid continental, oceanic and subarctic (19). See Table S1 for more details.

For the temperature exposures at the city level, we used a high-resolution observational dataset of daily temperature for Norway, which is available at a 1 km resolution and is continuously updated (20). Making use of the geographical borders of each city, we averaged the mean daily temperatures for each city from 1st January 1996 to 31st December 2018.

We also used mean relative humidity (24-hour) data provided by the Norwegian Centre for Climate Services (NCCS) (21) and air pollution data, in particular measurements of ozone (O₃), particular matter with a diameter of 10 μ m and 2.5 μ m or less (PM₁₀ and PM_{2.5}) and nitrogen dioxide (NO₂) at a 1 km resolution. Pollution measurements for all cities come from the Nordic DEHM-UBM (Danish Eulerian Hemispheric Model- Urban Background Model) setup. All pollutants were computed as 24-hour averages but O₃, calculated as the daily maximum 8-hour running average from hourly measurements.

Concentrations of pollutants were weighted according to the population grid GHS 2019A (GHS-POP R2019A: GHS population grid multitemporal (1975-1990-2000-2015) [https://ghsl.jrc.ec.europa.eu/ghs_pop2019.php\)](https://ghsl.jrc.ec.europa.eu/ghs_pop2019.php), utilizing the year 2015 and averaging the daily values within the municipal borders as for the temperature exposure.

The health data comprised daily mortality counts for non-accidental, cardiovascular and pulmonary mortality (International Classification of Diseases, ICD-10: A00-R99, I00-I99 and J00-J99 respectively) over the study period of 1996 to 2018, provided by the Norwegian Death Registry (22). The data was also disaggregated by age groups (0-74, 75+) and sex.

Methods

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stimates of city-specific temperature-mortality association,
age regression included a natural We used a two-stage methodology. We first analysed the relationship between the mortality outcome and the average mean daily temperature using a quasi-Poisson distributed lag non-linear model for each city to derive estimates of city-specific temperature-mortality associations, reported as relative risk (RR). The first-stage regression included a natural cubic B-spline of time with 8 degrees of freedom per year to control for seasonal and long-term trends, and indicators for the day of the week and bank holidays. The temperature was modelled with a quadratic B-spline with 3 internal knots placed at 10th, 75th, and 90th percentiles of location-specific distributions and the lag-response curve with a cubic Bspline with an intercept and 3 knots placed at equally spaced values in the log scale. We opted to extend the lag period to 14 days (14) instead of 21 (23) in order to preserve statistical power and account for any harvesting effect, while also capturing the delayed impact of cold temperatures. This parametrization is commonly used (23) and yielded the final model:

No. daily deaths ~ intercept + cb(Avg. Temp) + ns(time) + holidays + weekday

In the second stage, we pooled the city-specific relative risks using a multivariate meta-regression model with the city-specific range of temperature as a covariate and fixed effects to obtain the overall pooled estimate. Then we calculated the best linear unbiased predictors (BLUPs) (24), which could provide more precise estimations, particularly for the cities with smaller numbers of deaths. We next derived the minimum mortality temperature (MMT) in each city from the BLUP of the overall cumulative association between temperature and each mortality outcome. We referred to the minimum mortality temperature as the optimum temperature and used it as the reference for calculating the attributable risk (23). The quadratic B-spline of temperature in the first stage analysis was then re-centred according to each city-specific MMT to obtain accurate risk estimates at a given temperature. We explored heterogeneity with the I² statistic and the Cochrane's Q test.

We conducted sensitivity analyses to explore the impact of different modelling choices on nonaccidental mortality, while accounting for air pollution and area-level vulnerability factors, such as the proportion of the population aged 65 years and above. In the first stage models, pollutants were included as daily covariates, whereas the vulnerability factors were incorporated in the second stage as 2018 annual values. Additional results and further details can be found in the Supplementary Material (Table S2 and Methods S1).

The overall effect of temperature on a specific day on the RR of death was defined as the accumulation of the risk during the lag period (14 days). We calculated the attributable number of deaths and fractions of total deaths using the overall cumulative RR corresponding to each day's temperature in the next 14 days, and the respective empirical confidence intervals using Monte Carlo simulations assuming a normal distribution of the BLUPs of the reduced coefficients (25), using a previously described and applied method (23, 26). The sum of the contributions gives the total number of

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attributable deaths due to non-optimal temperatures and the ratio with the total number of deaths gives the total attributable fraction (AF).

We aggregated the contributions from the days below and above the point of MMT to calculate the AF due to cold and heat. Additionally, we classified AFs into moderate and extreme contributions by defining extreme temperatures as those falling below the $1st$ and exceeding the 99th location-specific percentiles.

We report pooled effects, defined as the cold and heat effects, represented by the RR at the $1st$ and 99th percentiles of the exposure-response association relative to the MMT, accompanied by 95% confidence intervals, for each outcome and subgroup*.* The model was separately fitted for sex and the two age groups: 0-74 years, and 75 years or older, based on cause of death.

All analyses were performed in software R version 4.1.0 using the packages *data.table*, *splines*, *dlnm* for the first stage and *mixmeta* for the second stage meta-analysis.

Results

The analysis included 230 979 non-accidental deaths, of which 83 051 were from cardiovascular causes and 24 217 were attributed to respiratory diseases. The average daily deaths from non-accidental mortality spanned from 12 in Oslo to 1 in Tromsø. The populations of the different cities are subject to different ranges of temperatures characterised by the specific climates (Table 1).

The overall exposure-response functions (ERF) curves (best linear unbiased predictions) presented in [Figure 1](#page-13-0) for non-accidental, cardiovascular, and respiratory mortality demonstrate risk increases for temperatures below and above the MMT, except for cardiovascular-related mortality above the MMT and the oceanic cities below the MMT for cardiovascular and respiratory disease mortality. Remarkably, oceanic cities exhibit a stronger effect with decreasing temperatures for non-accidental mortality. Additionally, at higher temperatures, the respiratory ERFs yield higher risks than the nonaccidental ERFs in all cities.

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d *mixmeta* for the second stage meta-analysis.

1230 979 non-accidental deaths, of which 83 051 were from

tributed to respiratory diseases. The average daily We report the pooled predicted overall cumulative association for the seven cities in [Figure 2,](#page-14-0) denoted as the RR at the $1st$ and $99th$ percentiles. These percentiles represent the extreme temperature thresholds that define the excess mortality attributed to non-optimal temperatures, specifically cold and heat effects. Regarding non-accidental mortality, statistically significant effects were observed in the entire population, as well as in women and the age group 75+. Both the female group and the oldest age group, 75+, exhibited a similar pattern with statistically significant associations between non-optimal weather conditions and mortality. This suggests that they are the most vulnerable among the groups analysed, including the effects of cold on CVD mortality and the effects of heat on pulmonary mortality.

In contrast, the 0-74 age group exhibited only one significant pooled effect concerning non-accidental mortality attributed to low temperatures. Remarkably, significant associations were observed solely for cold effects concerning CVD mortality, while heat effects were linked to respiratory disease mortality. Conversely, no significant pooled effects were found for males.

By city, statistically significant heat effects were identified in all cities except Bergen and Stavanger for non-accidental and pulmonary mortality in the 75+, women, and overall groups. Concerning cardiovascular mortality, we observed only one significant heat effect in Oslo for women. A similar pattern emerged for cold effects in the non-accidental and cardiovascular groups. Notably, all cities except Oslo yielded a significant cold effect in the non-accidental women's group. Additionally, the younger group exhibited significant cold effects related to pulmonary mortality in Oslo, Trondheim, and Drammen.

The largest significant heat effect was found in Trondheim, with a RR of 1.6 (95%CI 1.2-2.2) for the females, whereas for cold effects, Oslo had the highest impact with a RR of 1.9 (95%CI 1.2-3.0) in the younger group, both associated with respiratory mortality. In contrast, there were no statistically significant effects in any of the male groups and the younger cardiovascular group. All outcomes are available in Table S4 of the Supplementary Material.

The overall non-accidental mortality AF due to non-optimal temperatures was 5.3% (95%CI 2.0-8.3), which translated into 12 286 deaths in 23 years (1996-2018). Of these deaths 11 729 were attributed to cold temperatures and 557 to heat. Examining causes of death, overall AF for CVD mortality was 11.8% (95%CI 6.4-16.4) and 5.9% (95%CI -4.0-14.3) for respiratory mortality. Cold was responsible for most of the mortality burden (AF for non-accidental 4.9% (95%CI 1.7-7.8), cardiovascular 11.4% (95%CI 6.0-15.4) and respiratory deaths 5.1% (95%CI -5.5-13.8)).

The city-specific attributable fractions, further itemised into extreme and moderate heat and cold components, are presented in Figure 3. Additionally, results categorised by sex and age groups can be found in the Supplementary Material (Tables S5-S8 and Figures S7-S12). Extreme temperatures were associated with relatively smaller risks portions compared to moderate temperatures, aligning with the ERFs depicted in Figure 1 and the city-specific temperature distributions.

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tory deaths 5.1% (95%CI -5.5-13.8)).

ibutable fractions, further itemised into extreme and mo

ssen The MMTs shifted to higher temperatures in the CVD groups compared to non-accidental and respiratory in most of the cities. These corresponded to an average minimum mortality percentile (MMP) per cause, rising from 70 to 94 for non-accidental, 80 to 99 for cardiovascular diseases, and 49 to 91 for respiratory diseases mortality. Consequently, this explains that the absence of heat effects in cardiovascular diseases. MMTs are detailed per cause, group, and city in Tables S6, S7 and S8 of the Supplementary Material.

We also plotted the joint distributed lag graphs using a maximum lag of 14 days for the RRs comparing the effects of extreme cold and heat to the MMT on both non-accidental and cause-specific mortality. Cold effects peaked in general on the first day (lag 1) and lasted longer, while the heat effects were immediate. Cold effects were significant for non-accidental and respiratory mortality, and heat effects were statistically significant for all three causes of mortality in females (Figures S13-S17 of the Supplementary Material).

Our sensitivity analyses suggested that our results were consistent and independent of modelling assumptions, also when controlled for PM_{25} , PM_{10} , NO₂, ozone, humidity, and vulnerability factors. These results are shown in Tables S2 and S3 of the Supplementary Material.

Discussion

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We examined the associations between exposure to air temperature and mortality due to nonaccidental, cardiovascular, and respiratory diseases in Norwegian urban areas from 1996-2018. Additionally, we analysed these associations in different subgroups, stratifying by age and sex. to ensure the accuracy and integrity of our results, we also examined climatic and geographical characteristics as potential confounders and selectively included only those variables that acted as confounders in our analyses, prioritising their impact. Our findings indicate that non-optimal temperatures were associated with an increased mortality risk for all the causes and subgroups analysed. Particularly moderate cold temperatures were responsible for a higher proportion of the mortality burden in the seven Norwegian cities. We estimated higher relative risks for colder $\mathbf{1}$

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temperatures in contrast to warmer temperatures, except within the pulmonary groups: overall, those aged 75 and above, and men, for which the relative risks for hotter temperatures were slightly higher. In an Estonian study by Orru et al. (13), greater effects of heat were noted compared to cold for mortality due to external causes, whereas Åström DO et al. (12) in the Baltics found no association between respiratory disease mortality and cold effects but identified an adverse effect with cold temperatures on CVD mortality. In contrast, our findings reveal more pronounced cold and heat effects for respiratory mortality compared to CVD mortality, with the exception of males experiencing cold effects. Additionally, only the heat effect for non-accidental mortality in the younger group was larger than the effect on respiratory related mortality. Changes linked to age have been associated with physiological differences in thermoregulation, which could account for differences in our results (9). Gender differences are evident in our analysis, with women displaying greater susceptibility to temperature-related mortality. However, it is worth considering that the observed gender disparities could be influenced by various additional factors, such as the social context (15). These results in which the estimated pooled overall heat effects are larger for respiratory than CVD mortality, are consistent with other studies (27). In our study, we did not identify a significant increase in cardiovascular mortality due to heat, similarly to this multi-city study (28); however, another found a clear high effect in southern Swedish locations and not in the northern ones (29).

Consisting with other studies (27), we found that cold effects lasted longer than heat effects. On average, the effect of cold temperatures peaked at lag one day and lasted almost ten days, while heat effects appeared immediately and lasted two or three days, followed by a mortality displacement.

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1.227). In our study, we did not id We found that temperature is responsible for a substantial fraction of 5.3% of the non-accidental mortality burden. This percentage translates to 469 deaths in 2018 alone for the seven cities. Other studies have reported similar mortality burden due to cold temperatures, and in similar latitudes to the overall we describe. Åström et al. (30) recount that temperature is responsible for 4.5% in Stockholm, Sweden for all-cause mortality in the period 1990-2002 and Gasparrini et al. (23) 3.7% for the same period. Another study conducted in Tallin and Riga reported AF values of 7.4% and 8.3% respectively, although only November to March months were analysed from the years 1997-2015 and 2009-2015 (12). The former finding aligns more closely with our results for non-accidental mortality in Oslo, Trondheim and Drammen (7%, 6.8% and 6.2% respectively). Achebak et al. (18) documented an overall AF of 13.1% for CVD mortality which is comparable to our own findings of 11.8% (6.4-16.4).

Our analysis had several strengths. As the first study to focus exclusively on Norwegian cities, it captures their distinctive idiosyncrasy stemming from subpolar latitudes and comparatively milder temperatures than Russia or Canada. Our results provide novel and robust evidence on mortality risk and burden from both non-accidental causes and cardiopulmonary diseases. Another considerable strength of our data is the completeness of Norwegian registries, with close to 100% registrations of data such as residence addresses and time of death. Additionally, the reliability of the Norwegian death certification system bolsters our findings' credibility, supported by careful diagnosis and cause attribution procedures.

We acknowledge some limitations inherent to our study. Our scope centred on urban populations. Similar to numerous ecological epidemiological studies, assessing individual exposures remained unfeasible. The small number of daily deaths in some locations, particularly for respiratory mortality, produced wide confidence intervals and thus low precision estimates. Therefore, the reported effects should be interpreted cautiously, particularly the cold effects as we have not considered the possible confounding during the cold season for influenza activity. Lastly, an underlying assumption was made that children's deaths attributable to temperature are exceedingly scarce, thereby exerting minimal influence on our findings.

Conclusions

This study confirmed that non-optimal temperatures are associated with increased mortality for nonaccidental and cardiopulmonary-related causes in Norway. We further identified that moderate temperatures are responsible for most deaths, and women and the elderly are the most vulnerable groups. Further research is needed to delimit the excess mortality that could be preventable. These results could contribute to improving public health preparedness and response measures for both cold and hot seasons.

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Table 1. Descriptive statistics by city.

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Figure 1. Overall cumulative exposure–response associations for non-accidental and cardiopulmonary mortality in seven cities and the respective pooled association. Exposure–response associations are showed as best linear unbiased predictions. RR=Relative risk.

Figure 2. Pooled effects for non-accidental, cardiovascular, and respiratory mortality are reported at the 1st and 99th percentiles as cold and heat effects, respectively, with 95% confidence intervals. These effects are stratified by sex and age group.

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Figure 3. Proportion of non-accidental and cardiopulmonary mortality attributed to moderate and extreme hot and cold temperatures across different cities. Extreme and moderate high and low temperatures were determined using the minimum mortality temperature at the 1st and 99th percentiles of temperature as thresholds.

Supplementary Material

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Short-term association between air temperature and mortality in seven cities in Norway: A time series analysis.

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Figure S7. Fractions of non-accidental mortality attributable to moderate and extreme hot and cold temperature by city and age group.

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Methods S1: First stage sensitivity analysis

An initial sensitivity analysis was performed on the non-accidental mortality models, where we checked the city-specific average and range temperature as meta-predictors. We further considered two modelling frameworks: fixed-effects and random-effects (restricted maximum likelihood) metaanalysis and sorted them according to their Akaike Information Criteria (AIC). The model with the lowest AIC was invariably the one with no covariates, followed by the temperature range singlepredictor model. We also varied the choice of lag from 1 to 28 days and internal knots of the temperature distribution from 2 to 4. We chose a lag period of 14 days and three internal knots to model the exposure-response curves based on previous studies (1). [Table S2](#page-22-0) contains results from the tests of these modelling choices for this sensitivity analysis.

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COLL CLL CLL CLL CLL CLL CLL CLL CLL With regards to the cut-offs for defining the extreme temperatures, we did not choose the 2.5th and 97.5th as the minimum mortality temperature in colder weathers is normally very high, in the range of the 90ths' percentiles (averaged 93rd in non-accidental mortality). In temperate regions the temperature percentile of minimum mortality varies from about the 80th to the 90th percentile (1).

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Table S1. Extended descriptive statistics

Table S2. Sensitivity analysis.

Computed on the fraction (%) attributable to temperature (total, heat, and cold components), by varying modelling choices, fitting the models to non-accidental mortality, and controlling for PM25, PM10, NO2, ozone, humidity, and vulnerability factors. Brackets indicate 95% empirical confidence intervals.

Table S3. Second-stage meta-regression models.

AIC, BIC, multivariate Cochran Q test for heterogeneity (p-value), and I² statistic (%) in different metaregression models.

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Table S4. The relative risks of cold temperature and hot temperature on deaths at lags of 0–14 days in the seven cities. Estimates were calculated from the non-linear model. Cold effects (1st VS minimum-mortality temperature (MMT)) and heat effects (99th VS MMT) correspond to Figure 2 where the pooled ones are shown.

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Table S5. Attributable fractions (%) on non-accidental and cardiopulmonary deaths in the seven cities for cold and heat defined as temperatures below and above the minimum mortality temperature (MMT) and extremes defined as cut-offs at the 1st and 99th percentiles. Brackets indicate 95% empirical confidence intervals. AFs for CVD are presented in italics.

Table S6. Attributable fractions (%) on non-accidental, cardiovascular and respiratory deaths in the seven cities for moderate cold and heat defined as temperatures below and above the minimum mortality temperature (MMT) and extremes defined as cut-offs at the 1st and 99th percentiles. Brackets indicate 95% empirical confidence intervals.MMP is minimum mortality percentile.

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Table S7. Attributable fractions (%) on non-accidental, cardiovascular and respiratory deaths by sex in the seven cities for moderate cold and heat defined as temperatures below and above the minimum mortality temperature and extremes defined as cut-offs at the 1st and 99th percentiles. Brackets indicate 95% empirical confidence intervals.

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Table S8. Attributable fractions (%) on non-accidental, cardiovascular and respiratory deaths by age group in the seven cities for moderate cold and heat defined as temperatures below and above the minimum mortality temperature and extremes defined at the 1st and 99th percentiles. Brackets indicate 95% empirical confidence intervals.

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Table S10. Pooled attributable fractions (%) and number of deaths (AN) for non-accidental, cardiovascular, and respiratory mortality. Brackets indicate 95% empirical confidence intervals.

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Overall cumulative exposure–response associations for non-accidental mortality in seven cities by sex. Exposure–response associations as best linear unbiased prediction (with 95% empirical CI, shaded) in the seven cities. RR=Relative risk.

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Overall cumulative exposure–response associations for CVD mortality in seven cities by sex. Exposure– response associations as best linear unbiased prediction (with 95% empirical CI, shaded) in the seven cities. RR=Relative risk.

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Overall cumulative exposure–response associations for respiratory disease mortality in seven cities by sex. Exposure–response associations as best linear unbiased prediction (with 95% empirical CI, shaded) in the seven cities. RR=Relative risk.

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Figure S4

Overall cumulative exposure–response associations for non-accidental mortality in seven cities by age group. Exposure–response associations as best linear unbiased prediction (with 95% empirical CI, shaded) in the seven cities. RR=Relative risk.

Overall cumulative exposure–response associations for CVD mortality in seven cities by age group. Exposure–response associations as best linear unbiased prediction (with 95% empirical CI, shaded) in the seven cities. RR=Relative risk.

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Figure S6

Overall cumulative exposure–response associations for respiratory mortality in seven cities by age group. Exposure–response associations as best linear unbiased prediction (with 95% empirical CI, shaded) in the seven cities. RR=Relative risk.

Fractions of non-accidental mortality attributable to moderate and extreme hot and cold temperature by city and age group. Extreme and moderate high and low temperatures were defined with the minimum mortality temperature and the 1st and 99th percentiles of temperature as cut-offs.

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Figure S8

Fractions of cardiovascular mortality attributable to moderate and extreme hot and cold temperature by city and age group. Extreme and moderate high and low temperatures were defined with the minimum mortality temperature and the 1st and 99th percentiles of temperature as cut-offs.

CVD deaths: 0-74 years >75 years Trondheim Tromsø For Personal Product Cold Products Cold Stavanger Oslo Fredrikstad **Drammen Bergen** $\overline{0}$ Extreme heat

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Figure S10

Fractions of non-accidental mortality attributable to moderate and extreme hot and cold temperature by city and sex. Extreme and moderate high and low temperatures were defined with the minimum mortality temperature and the 1st and 99th percentiles of temperature as cut-offs.

Non-accidental deaths: men women Trondheim Tromsø For Personal Product Cold Products Cold Stavanger Oslo Fredrikstad **Drammen Bergen** $\overline{2}$ $\bf 8$ $\mathbf 0$ Extreme heat

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Fractions of cardiovascular mortality attributable to moderate and extreme hot and cold temperature by city and sex. Extreme and moderate high and low temperatures were defined with the minimum mortality temperature and the 1st and 99th percentiles of temperature as cut-offs.

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Figure S12

Fractions of respiratory mortality attributable to moderate and extreme hot and cold temperature by city and sex. Extreme and moderate high and low temperatures were defined with the minimum mortality temperature and the 1st and 99th percentiles of temperature as cut-offs.

Overall lag structure in the effects of extreme cold temperature on daily cause-specific mortality in seven Norwegian cities, from 1996 to 2018, classified by cause of death. The effects were determined by comparing the risks at -11.2°C (that is, the mean of the 1st centile of temperature distributions) to the mean minimum temperature (MMT). The solid lines represent the mean estimates, while the shaded areas represent the 95% confidence intervals. Natural refers to all non-accidental mortality.

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Figure S14

Overall lag structure in the effects of extreme hot temperature on daily cause-specific mortality in seven Norwegian cities, from 1996 to 2018, classified by cause of death. The effects were determined by comparing the risks at 19.5°C (that is, the mean of the 99th centile of temperature distributions) to the estimated minimum mortality temperature (MMT). The solid lines represent the mean estimates, whilst the shaded areas represent the 95% confidence intervals. Natural refers to all non-accidental mortality.

Overall lag structure in the effects of extreme cold and hot temperatures on daily non-accidental mortality in seven Norwegian cities, from 1996 to 2018, distinguished by sex. The effects were determined by comparing the risks at -11.2°C and 19.5°C (that is, the mean of the 1st and 99th centiles of temperature distributions) to the estimated minimum mortality temperature. The solid lines represent the mean estimates, while the shaded areas represent the 95% confidence intervals. Natural refers to all non-accidental mortality.

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Figure S16

Overall lag structure in the effects of extreme cold and hot temperatures on daily cardiovascular mortality in seven Norwegian cities, from 1996 to 2018, distinguished by sex. The effects were determined by comparing the risks at -11.2°C and 19.5°C (that is, the mean of the 1st and 99th centiles of temperature distributions) to the estimated minimum mortality temperature. The solid lines represent the mean estimates, while the shaded areas represent the 95% confidence intervals.

Overall lag structure in the effects of extreme cold and hot temperatures on daily respiratory disease mortality in seven Norwegian cities, from 1996 to 2018, distinguished by sex. The effects were determined by comparing the risks at -11.2°C and 19.5°C (that is, the mean of the 1st and 99th centiles of temperature distributions) to the estimated minimum mortality temperature. The solid lines represent the mean estimates, while the shaded areas represent the 95% confidence intervals.

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Figure S8.

Directed acyclic graph with the variable *Smoking* as an example. Variables included in the sensitivity analysis were considered confounders and included in the model one at a time.

Exposure: Temperature

Exposure: Mortality

Party

Par

References

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