The Impact of Heat Waves and Cold Spells on Mortality Rates in the Dutch Population

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We conducted the study described in this paper to investigate the impact of ambient temperature on mortality in the Netherlands during 1979–1997, the impact of heat waves and cold spells on mortality in particular, and the possibility of any heat wave- or cold spell-induced forward displacement of mortality. We found a V-like relationship between mortality and temperature, with an optimum temperature value (e.g., average temperature with lowest mortality rate) of 16.5°C for total mortality, cardiovascular mortality, respiratory mortality, and mortality among those ≥ 65 year of age. For mortality due to malignant neoplasms and mortality in the youngest age group, the optimum temperatures were 15.5°C and 14.5°C, respectively. For temperatures above the optimum, mortality increased by 0.47, 1.86, 12.82, and 2.72% for malignant neoplasms, cardiovascular disease, respiratory diseases, and total mortality, respectively, for each degree Celsius increase above the optimum in the preceding month. For temperatures below the optimum, mortality increased 0.22, 1.69, 5.15, and 1.37%, respectively, for each degree Celsius decrease below the optimum in the preceding month. Mortality increased significantly during all of the heat waves studied, and the elderly were most effected by extreme heat. The heat waves led to increases in mortality due to all of the selected causes, especially respiratory mortality. Average total excess mortality during the heat waves studied was 12.1%, or 39.8 deaths/day. The average excess mortality during the cold spells was 12.8% or 46.6 deaths/day, which was mostly attributable to the increase in cardiovascular mortality and mortality among the elderly. The results concerning the forward displacement of deaths due to heat waves were not conclusive. We found no cold-induced forward displacement of deaths. *Key words*: cold spells, heat waves, mortality, mortality displacement, Netherlands, temperature. *Environ Health Perspect* 109:463–470 (2001). [Online 3 May 2001]
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In healthy individuals, an efficient heat regulation system enables the body to cope effectively with thermal stress. Within certain limits, thermal comfort can be maintained by appropriate thermoregulatory responses such that physical and mental activities can be pursued without any detriment to health. Temperatures exceeding these limits, both with respect to heat and cold, substantially increase the risk of death.

In both the United States and Europe, an increase in the number of deaths has been recorded after winter cold spells and summer heat waves (*1*). Heat places stress on the thermoregulatory system (*2*). Exposure to high temperatures causes increases in blood viscosity and blood cholesterol levels (*3*). The number of deaths caused by extreme heat during heat waves is compensated for by a temporary fall in numbers in subsequent weeks (*3,4*). These compensatory effects suggest a mortality displacement or "harvesting" effect: heat principally affects those whose health is already compromised and who would have died in the short term anyway (*2,3*). Thus, only a part of the excess mortality due to extreme heat relates to avoidable deaths and therefore to a significant reduction in overall lifetime (*5*).

The immediate effects of cold on mortality rates are reported in several studies (*2,3,6–11*). In the Netherlands, annual cold-related mortality is higher than heat-related mortality (*12*). Bull (*13*) argued that excess mortality in winter may be due to physiologic changes in cellular and humoral immunity, with behavioral factors also playing a role. Exposure to cold can lead to direct cardiovascular stress due to changes in blood pressure, vasoconstriction, and an increase in blood viscosity and levels of red blood cell count, plasma cholesterol, and plasma fibrinogen (*2,7*). Low temperatures lead to thrombosis due to hemoconcentration (*8,9*), and rapid deaths occur due to the rupture of atheromatous plaques during hypertension and cold-induced coronary spasm (*8*). Indirectly, influenza contributes to cold-related mortality (*2,3,14*). Susceptibility to pulmonary infections may increase through bronchoconstriction, caused by breathing cold air (*15*). Less is known about the possibility of cold-induced mortality displacement.

We conducted this study to investigate the impact of ambient temperature on mortality in the Netherlands during 1979–1997, and the impact of cold spells and heat waves on mortality, in particular. We examined whether the cold spells and heat waves merely brought forward the deaths of those who would have died in the short term anyway or if the induced mortality made a substantial contribution to overall lost lifetime.

Methods

Data. The Netherlands Central Bureau of Statistics (Voorburg, the Netherlands) provided the numbers of deaths by the day on which the death occurred (1 January 1979–31 December 1997) and by selected causes of death and two age categories (0–64 years of age and ≥ 65 years of age, only for 1 January 1988–31 December 1997). The selected causes of death were malignant neoplasms [*International Classification of Diseases, Revision 9* (ICD-9: AM 12–19)], respiratory disease (ICD-9: AM 33–35), and cardiovascular disease (ICD-9: AM 25–32).

The Netherlands Royal Meteorological Institute (De Bilt, the Netherlands) provided 24-hr data on minimum and maximum temperatures. The average daily temperature was calculated as the average of the minimum and maximum temperatures. All data refer to the De Bilt station, which is located in the center of the country. Differences in climate within the Netherlands are small, and weather changes usually affect all parts of the country at roughly the same time.

Heat waves and cold spells. A heat wave is defined by the Netherlands Royal Meteorological Institute as a period of at least 5 days, each of which has a maximum temperature of at least 25°C, including at least 3 days with a maximum temperature of at least 30°C (measured at the De Bilt station). According to this definition, there were six heat waves in the past 19 years, and they lasted from 6 to 13 days (Figure 1).

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There is no official definition of a cold spell in the Netherlands. We searched for extreme cold periods between 1 January 1979 and 31 December 1997, which occurred with a frequency that resembled the frequency of the official heat waves in our study period. Because the minimum temperature during the winter months (December–February) correlates more closely with mortality (r = –0.058) than the maximum temperature (r = –0.034), the definition we adopted for a cold spell was based on the daily minimum temperature. In our definition, a cold spell is a period of at least 9 days with a minimum temperature of –5°C or lower, of which at least 6 days have a minimum temperature of –10°C or lower. Therefore, the duration of a cold spell according to this definition is longer than that of a heat wave. Kunst et al. (*16*) indicated that the effect of extreme cold might be more severe if the period of extreme cold is longer. During the study period, a cold spell occurred five times according to our definition, varying in duration from 9 to 17 days (Figure 1). The analysis of the excess mortality during cold spells was also performed with another definition of a cold spell, in which the duration of a cold spell was shorter and the requirements regarding the level of the minimum temperature were more stringent. This additional analysis, however, showed no clear excess mortality during these shorter and more severe cold periods (results not shown).

Excess mortality during cold spells and heat waves. To examine the impact of extreme temperatures on mortality, we calculated 31-day moving averages of daily mortality during the heat wave and cold spell days for the two preceding years combined (*4*). This was used to estimate the mortality during the heat wave or cold spell period in the absence of extreme temperatures. Excess mortality was calculated as the difference between the total number of deaths observed in the heat wave or cold spell and the corresponding 31-day moving average. Analyses were repeated for the selected age groups and causes of death. Excess mortality could not be calculated for the cold spell in winter 1978–1979 because this cold spell started before our study period began (1 January 1979) and because the necessary mortality data was not available prior to this day.

In addition to the method described above, Rooney et al. (*4*) investigated excess mortality during the 1995 heat wave in England using another approach. They compared the observed mortality with the 31-day moving average for the same year. The results for excess mortality during heat waves and cold spells using this method are reported in this paper but not discussed because we believe that this approach is less accurate. It is certainly more conservative, as the values for heat wave/cold spell days are included in the average values. This obscures the comparison between different heat waves and cold spells because of differences in their length.

Modeling the association between temperature and mortality. The daily numbers of deaths due to all causes, as well as those from the selected causes, were related to the daily average temperature using Poisson loglinear regression analyses over the whole dataset (1 January 1979–31 December 1997), controlled for the time trend and season. Time trend [the sequential number of the day (1 for 1 January 1979 and 7,305 for 31 December 1997)] was included to account for long-term trends resulting from changes in, for example, population structure, socioeconomic conditions, and the provision of health care over time. Without correcting for season, the mortality effects of seasonal variation in other factors, such as physical exercise, diet, stress, and blood pressure, are picked up by temperature (*6*,*17*). The variable representing season is kept constant between years (included as a dummy variable for each month, with December as reference month) (*3*).

Figure 2 shows the V-like relationship between mortality and temperature for mortality due to all causes as well as for mortality due to the selected causes and in the two age groups. Therefore, average daily temperature within the model was measured by two complementary variables, heat (0 if average temperature ≤ optimum value, otherwise average temperature minus optimum value) and cold (0 if average temperature ≥ optimum value, otherwise optimum value minus average temperature). The optimum temperature value corresponds to the average temperature with the lowest mortality level. We performed regression analyses to evaluate several optimum values (e.g., 14.0°C, 14.5°C, 17.0°C) until we found the one that best fit the lowest scaled deviance (*3*).

The regression equations for the assessment of the optimum values included values of heat and cold for previous days to account for lagged effects of temperature, in accordance with the study by Kunst et al. (*3*). To reduce multicolinearity, temperature variables were constructed for groups of subsequent days (lag periods) by averaging values for heat and cold over these periods. Lag times were grouped into lag periods that increased exponentially in size (1–2, 3–6, 7–14, 15–30 days), whereas lag times longer than 1 month were ignored (*3*).

The regression model can be described by

$$\log(y) = \beta_0 + \beta_1 \times t_i + \beta_2 \times x_2 + \ldots + \beta_j \times x_j,$$

where y is the number of deaths on day i (index day), t_i is the sequential value for day i (from 1 for 1 January 1979 to 7,305 for 31 December 1997), $x_2 \ldots x_j$ are the j independent variables (values for heat, values for

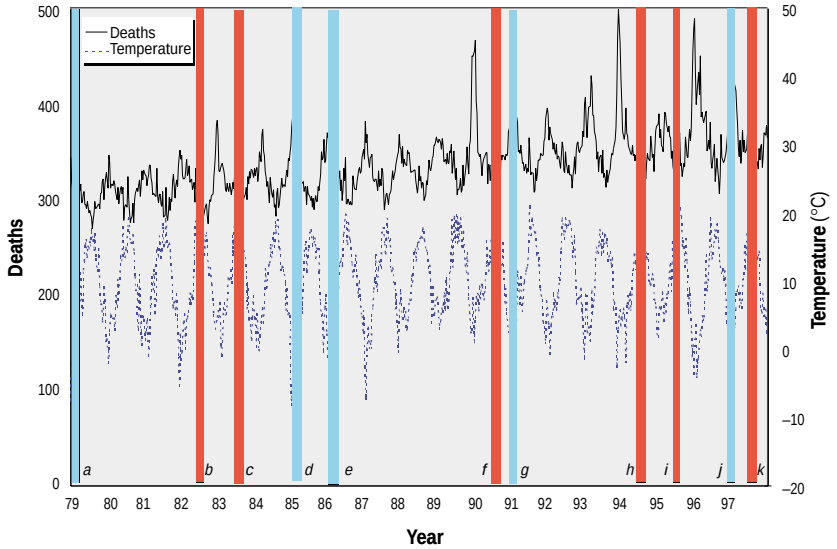

Figure 1. Daily average temperatures and total mortality rates in the Netherlands, 1 January 1979–31 December 1997. Abbreviations: max, maximum; min, minimum; temp, temperature.
[a]Cold spell: 30 December 1978–6 January 1979 (min temp: 1 day between –5°C and –10°C, 8 days at –10°C or less). [b]Heat wave: 29 July–4 August 1982 (max temp: 3 days between 25°C and 30°C, 4 days at ≥ 30°C). [c]Heat wave: 4 July–12 July 1983 (max temp: 6 days between 25°C and 30°C, 3 days at ≥ 30°C). [d]Cold spell: 4 January–20 January 1985 (min temp: 6 days between –5°C and –10°C, 11 days at –10°C or less). [e]Cold spell: 12 February–28 February 1986 (min temp: 11 days between –5°C and –10°C, 6 days at –10°C or less). [f]Heat wave: 26 July–4 August 1990 (max temp: 7 days between 25°C and 30°C, 3 days at ≥ 30°C). [g]Cold spell: 6 February–14 February 1991 (min temp: 3 days between –5°C and –10°C, 6 days at –10°C or less).[h]Heat wave: 19 July–31 July 1994 (max temp: 8 days between 25°C and 30°C, 5 days at ≥ 30°C). [i]Heat wave: 29 July–3 August 1995 (max temp: 3 days between 25°C and 30°C, 3 days at ≥ 30°C). [j]Cold spell: 24 December 1996–9 January 1997 (min temp: 9 days between –5°C and –10°C, 8 days at –10°C or less). [k]Heat wave: 5 August–13 August 1997 (max temp: 4 days between 25°C and 30°C, 5 days at ≥ 30°C).

cold, average values for heat during the different lag times, average values for cold during the different lag times, calendar months), and $\beta_2 \ldots \beta_j$ are the regression coefficients.

The regression coefficient (β) corresponding to a lag period was transformed, using the formula $100 \times (e^{\beta}-1)$, to the percentage change in mortality associated with a 1°C increase in the average value of cold or heat within the respective lag period (percental effect).

In order to construct an appropriate and parsimonious model for each cause of death and age group, variables were omitted if they met one of the two exclusion criteria:

- Adding the variable to the model causes a reduction in the scaled deviance associated with the null model (time trend only) of < 1%
- The regression coefficient corresponding to the variable was not significant ($p = 0.01$).

Forward displacement of deaths. Subsequent to the regression analyses, we used the same models to predict mortality in the 30 days after the heat wave or cold spell by removing the mortality effects of the extreme heat or cold itself. This was performed by assuming that, in the absence of extreme temperatures, the average temperature during the heat wave or cold spell period could be estimated by linear interpolation between the day before and the day after the heat wave or cold spell, and by recalculating the average temperature in the heat wave or cold spell. We then used average temperatures, including the recalculated values, in the models to predict post-heat wave or cold spell mortality in absence of the extreme temperatures. To study the forward displacement of deaths caused by extreme temperatures, we compared the predicted mortality values in the absence of a heat wave or cold spell to the observed post-heat wave or cold spell mortality during different lag periods after the heat wave or cold spell in the month after the event. Mortality displacement could not be studied for the cold spell in winter 1978–1979 because this cold spell started before the beginning of our study period (1 January 1979) and the necessary mortality data was not available before that day.

Because age-specific mortality data is only available for the two latest cold spells, we did not perform an age-specific analysis of a cold spell-induced forward displacement of mortality.

We used SAS version 6.12 software (SAS Institute, Cary, NC, USA) to analyze the data.

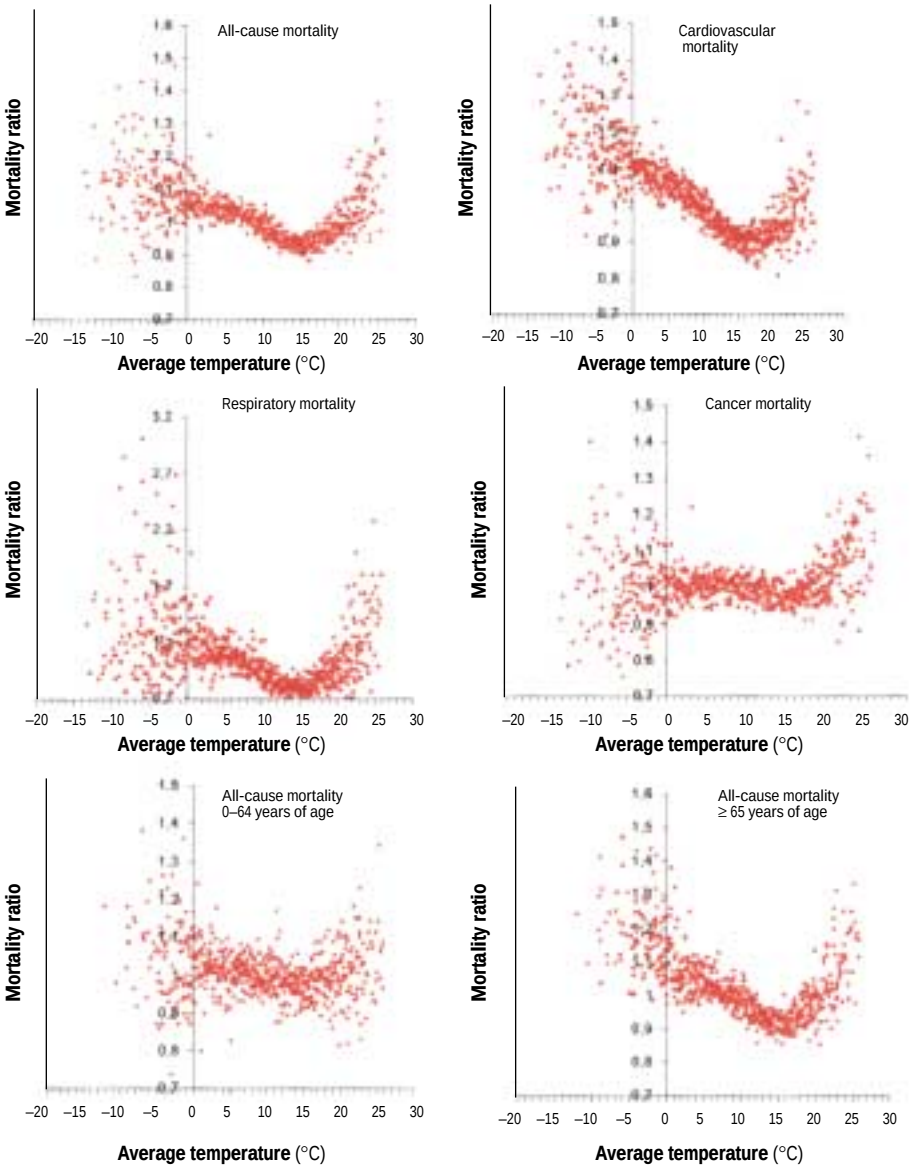

Figure 2. The relationship between mortality and average temperature (mortality ratio = observed number of deaths on day *i* ÷ mean number of deaths over the whole study period).

Results

The relationship between mortality and average temperature was V-like, with an optimum temperature value corresponding to the lowest point in the curve (Figure 2). This optimum value was 16.5°C for total mortality, cardiovascular mortality, respiratory mortality and mortality among those ≥ 65 years of age, whereas for mortality due to malignant neoplasms and mortality in the younger age group, the optimum value was 15.5°C and 14.5°C, respectively.

Excess mortality during heat waves and cold spells. Tables 1 and 2 show the results of the analyses of excess mortality during the heat waves and cold spells in the study period.

Table 1 shows significant excess total mortality during all of the heat waves studied, particularly mortality due to respiratory causes. The average excess in all-cause mortality during these heat waves was 12.1% or 39.8 deaths/heat wave day. The largest excess is seen for the 1994 heat wave (24%), which was also the longest heat wave in the study period (13 days). The excess respiratory mortality of 120% during the 1994 heat wave is noteworthy. This is also significant in the 1982, 1983, and 1990 heat waves, and excess cardiovascular mortality is significant during the 1983, 1990, 1994, and 1995 heat waves. Mortality caused by malignant neoplasms increased significantly during the 1983, 1994, and 1995 heat waves. The total excess mortality is largely attributable to increases in mortality in the ≥ 65 age group, whereas the heat had little effect among those younger than 65.

There was significant excess mortality due to all causes in the first three cold spells. The average excess in all-cause mortality during all of the studied cold spells was 12.8%, or 47.6 deaths/cold spell day. In the

1984–1985, 1990–1991, and 1996–1997 cold spells, there was no significant excess respiratory mortality. In the same three cold spells, the mortality caused by neoplasms also did not increase significantly. Cardiovascular mortality increased significantly during the cold spells in the winters of 1984–1985, 1985–1986, and 1990–1991. During the first two cold spells, the percentage excess mortality was the highest for cardiovascular mortality compared to the other causes during the same cold spell.

Table 2 shows that in both cold spells for which data for different age groups are available (winter 1990–1991 and winter 1996–1997), the excess mortality in those ≥ 65 years of age is larger than that in the younger age group.

The lack in excess mortality during the cold spell in winter 1996–1997 is noteworthy. The only significant increase in mortality during this cold spell was among those ≥ 65 years of age.

Modeling the association between temperature and mortality. The reduction in scaled deviance of the null model (time trend only) is a measure of the explanatory power of the different models and, therefore, of the goodness of fit of the models. The explanatory power of the model for mortality due to all causes (including time trend, season, heat, cold, all lags heat, all lags cold) is relatively large compared to the other models; the reduction in scaled deviance of the model is 68%. The models for death due to cardiovascular diseases (including season, heat, cold, all lags heat, all lags cold) and mortality in the older age group (including time trend, season, heat, cold, all lags heat, all lags cold) have the greatest explanatory power, with a reduction in scaled deviance in the null model of 75% and 72%, respectively. These models are followed by the respiratory model (including time trend, season, heat, cold, all lags heat, all lags cold) with a reduction of 60%. The explanatory power of the models for mortality due to malignant neoplasms (including time trend, season, heat, cold, all lags heat except lag-days 15–30, all lags cold except lag-days 15–30) and for mortality among those 0–64 years of age (including heat, cold, lag-days 7–14 heat, lag-days 7–14 cold) is rather small, 41% and 25%, respectively.

The season variables met both inclusion criteria in all models, except in the model for the younger age group. In this model, the season variables were not significant.

Table 3 shows the results of the adjusted regression models. For example, the 0.27% effect of cold for the lag-days 1–2 in the model for all-cause mortality means that a 1°C increase in the average value of cold in the previous 2 days (i.e., a 1°C decrease in the average temperature below 16.5°C) is associated with a 0.27% increase in mortality. The aggregated effect for cold in the model for all-cause mortality of 1.37 means that the actual mortality rate is 1.37% higher for each degree Celsius increase in the average value of cold over the preceding month.

The estimates for heat show an immediate and positive effect, in particular for respiratory diseases. There is an inverse relationship between heat and mortality in the longer lag periods before the index day.

Table 1. Excess mortality during summer heat waves by selected causes of death and selected age groups.

	Compared with 31 DMA for the preceding 2 years		Compared with 31 DMA for the same year	
Year/cause	No. (95% CI)	Percentage (average excess per day)	No. (95% CI)	Percentage (average excess per day)
1982				
All causes, all ages	184 (90–278)	8.7 (26.3)	159 (66–253)	7.5 (22.7)
Neoplasms	42 (−7–92)	7.2 (6.0)	34 (−16–83)	5.6 (4.9)
Cardiovascular disease	21 (−39–81)	2.3 (3.0)	38 (−22–98)	4.2 (5.4)
Respiratory disease	32 (9–55)	30.7 (4.6)	14 (−10–36)	10.9 (2.0)
1983				
All causes, all ages	267 (158–375)	9.6(29.7)	134 (26–243)	4.6 (14.9)
Neoplasms	69 (12–127)	8.8 (7.7)	42 (−15–100)	5.2 (4.7)
Cardiovascular disease	89 (18–159)	7.3 (9.9)	72 (1–143)	5.9 (8.0)
Respiratory disease	26 (1–51)	19.5 (2.9)	9 (−16–34)	6.2 (1.0)
1990				
All causes, all ages	339 (223–457)	10.6 (33.9)	182 (66–299)	5.4 (18.2)
Age 0–64	39 (−15–93)	5.4 (3.9)	49 (−5–104)	6.8 (4.9)
Age ≥ 65	301 (198–404)	12.2 (30.1)	133 (30–236)	5.0 (13.3)
Neoplasms	8 (−52–69)	0.9 (0.8)	28 (−32–89)	3.0 (2.8)
Cardiovascular disease	119 (46–191)	9.5 (11.9)	74 (1–146)	5.7 (7.4)
Respiratory disease	61 (34–87)	47.1 (6.1)	19 (−8–56)	11.1 (1.9)
1994				
All causes, all ages	1,057 (913–1,201)	24.4 (81.3)	240 (96–384)	4.7 (18.4)
Age 0–64	15 (−46–75)	1.6 (1.1)	1 (−59–61)	0.1 (0.1)
Age ≥ 65	1,043 (911–1,173)	30.7 (80.3)	239 (107–370)	5.7 (18.4)
Neoplasms	179 (105–253)	14.2 (13.8)	99 (25–174)	7.5 (7.6)
Cardiovascular disease	244 (159–328)	15.1 (18.8)	39 (−46–123)	2.1 (3.0)
Respiratory disease	247 (205–289)	120.0 (19)	37 (−5–78)	8.8 (2.8)
1995				
All causes, all ages	236 (141–332)	11.0 (39.3)	224 (128–320)	10.3 (37.3)
Age 0–64	23 (−19–64)	5.3 (3.8)	12 (−30–53)	2.7 (2.0)
Age ≥ 65	214 (127–300)	12.4 (35.7)	212 (126–298)	12.3 (35.3)
Neoplasms	60 (10–110)	10.1 (10.0)	61 (11–111)	10.3 (10.2)
Cardiovascular disease	81 (24–139)	10.5 (13.5)	69 (11–126)	8.7 (11.5)
Respiratory disease	13 (−11–38)	9.4 (2.2)	20 (−4–45)	15.1 (3.3)
1997				
All causes, all ages	256 (142–371)	8.2 (28.4)	47 (−67–161)	1.4 (5.2)
Age 0–64	26 (−25–78)	4.0 (2.9)	28 (−23–80)	4.3 (3.1)
Age ≥ 65	230 (128–332)	9.3 (25.6)	18 (−84–120)	0.7 (2.0)
Neoplasms	47 (−14–107)	5.1 (5.2)	29 (−32–90)	3.1 (3.2)
Cardiovascular disease	50 (−17–117)	4.5 (5.6)	−6 (−73–60)	–0.5 (–0.7)
Respiratory disease	16 (−1–55)	15.5 (1.8)	5 (−23–33)	2.4 (0.6)

Abbreviations, CI, confidence interval; 31 DMA, 31-day moving average.

The compensatory effects are the smallest for respiratory disease mortality, and the relative aggregated effect is the largest for this kind of mortality. The compensatory effects are the largest for deaths due to malignant neoplasms—nearly three-fourths of the effect within 3 days is compensated by a decrease in the number of deaths in the longer lag periods. In absolute terms, nearly 30% of all heat-related deaths (9.46 deaths/day/1°C heat during last month) are due to cardiovascular diseases (2.57 deaths/day/1°C heat during the last month) and nearly 25% is due to respiratory diseases (2.3 deaths/day/1°C heat during the last month) (Table 3).

In almost every lag period, there is positive relationship between cold and the actual mortality level. The negative percentage effect of cold on mortality at lag-day 0 is remarkable.

The positive relationships between cold and mortality are relatively weak for malignant neoplasms and relatively strong for respiratory diseases. The latter is probably due to the strong positive effect of cold on respiratory mortality in the 7–30 days before the index. The relative effect on mortality due to cardiovascular disease is slightly larger than that for total mortality, but much weaker than for respiratory mortality. In absolute terms, about one-half of all cold-related deaths (4.71 deaths/day/1°C cold during the last month) are due to cardiovascular diseases

Table 2. Excess mortality during winter cold spells by selected causes of death and selected age groups.

	Compared with 31 DMA for the preceding 2 years		Compared with 31 DMA for the same year	
Year/cause	No. (95% CI)	Percentage (average excess per day)	No. (95% CI)	Percentage (average excess per day)
1984–1985				
All causes, all ages	598 (439–756)	10.1 (35.2)	116 (−43–275)	1.8 (6.8)
Neoplasms	73 (−6–152)	4.7 (4.3)	37 (−41–116)	2.4 (2.2)
Cardiovascular disease	365 (265–474)	13.4 (21.5)	59 (−50–168)	1.9 (35)
Respiratory disease	19 (−19–57)	5.3 (1.1)	−5 (−43–33)	−1.3 (−0.3)
1985–1986				
All causes, all ages	1,736 (1,558–1,913)	26.8 (102.1)	306 (129–1,184)	3.9 (18.0)
Neoplasms	170 (86–253)	10.3 (10.0)	17 (−67–100)	0.9 (1.0)
Cardiovascular disease	683 (565–802)	23.0 (40.2)	170 (51–288)	4.9 (10.0)
Respiratory disease	462 (405–519)	117.2 (27.2)	69 (11–126)	8.7 (4.1)
1990–1991				
All causes, all ages	403 (38–276)	12.3 (44.8)	157 (38–276)	4.5 (17.4)
Age 0–64	103 (48–158)	15.0 (11.4)	85 (30–140)	12.1 (9.4)
Age ≥ 65	301 (195–406)	11.6 (33.4)	72 (–34–177)	2.5 (8.0)
Neoplasms	25 (−33–84)	2.9 (2.8)	−2 (−60–56)	−0.2 (−0.2)
Cardiovascular disease	244 (166–322)	18.1 (27.1)	109 (31–187)	7.4 (12.1)
Respiratory disease	16 (−12–43)	8.7 (1.8)	−13 (−41–14)	−6.4 (−1.4)
1996–1997				
All causes, all ages	137 (−32–306)	1.9 (8.1)	55 (−114–224)	0.7 (3.2)
Age 0–64	−20 (−92–52)	−1.5 (−1.2)	−9 (−81–62)	−0.7 (−0.5)
Age ≥ 65	157 (4–310)	2.6 (9.2)	64 (−89–218)	1.1 (3.8)
Neoplasms	45 (−39–129)	2.5 (2.6)	31 (−53–115)	1.7 (1.8)
Cardiovascular disease	18 (−86–123)	0.7 (1.1)	−4 (−108–100)	−0.1 (−0.2)
Respiratory disease	−15 (−63–33)	−2.4 (−0.9)	−3 (−50–45)	−0.4 (−0.2)

Abbreviations, CI, confidence interval; 31 DMA, 31-day moving average.

Table 3. Association between temperature (cold and heat) and daily mortality (due to different causes and in different age groups) controlled for the long-term mortality trend and season in the Netherlands for 1979–1997.

	Total mortality[a]	Age (years) 0–64[b,c]	Age (years) ≥ 65[a,b]	Malignant neoplasms[d]	Cardiovascular disease[e]	Respiratory disease[a]
Optimum	16.5	14.5	16.5	15.5	16.5	16.5
Mean no. deaths ± SD	344 ± 41	75 ± 10	287 ± 36	94 ± 12	143 ± 19	18 ± 8
Heat[f]						
Lag-day 0	1.59	0.98	1.51	1.34	1.42	2.43
Lag-days 1–2	1.18	—	1.46	0.46	1.12	3.89
Lag-days 3–6	0.41	—	1.11	–0.79	0.19	4.38
Lag-days 7–14	–0.10	–0.80	0.37	–0.54	–0.38	2.92
Lag-days 15–30	–0.36	—	–0.07	—	–0.49	–0.80
Aggregate heat						
Relative[g]	2.72	0.18	4.38	0.47	1.86	12.82
Absolute[h]	9.46	0.14	12.50	0.44	2.57	2.3
Cold[f]						
Lag-day 0	–0.31	0.02	–0.37	–0.45	–0.07	–0.66
Lag-days 1–2	0.27	—	0.30	0.20	0.39	0.15
Lag-days 3–6	0.38	—	0.39	0.32	0.33	0.57
Lag-days 7–14	0.56	0.48	0.79	0.15	0.61	2.04
Lag-days 15–30	0.47	—	0.94	—	0.43	3.05
Aggregate cold						
Relative[g]	1.37	0.5	2.05	0.22	1.69	5.15
Absolute[h]	4.71	0.38	5.88	0.21	2.42	0.93

[a]Calculated with full model (including time trend, season, heat, cold, all lags heat, all lags cold). [b]Only 1988–1997 data. [c]Calculated with model without time trend, season, lag-days 1–2, lag-days 3–6, and lag-days 15–30. [d]Calculated with model without lag-days 15–30. [e]Calculated with model without time trend. [f]Percental effects estimated from regression analysis of the temperature–mortality relationship; different adjusted models were used for the different causes of death. [g]The sum of the percental effects associated with the significant lag periods. [h]Calculated as the relative aggregated effect × the average daily number of deaths, according to Kunst et al. (*3*).

(2.42 deaths/day/1°C cold during the last month) (Table 3).

Forward displacement of deaths. Tables 4 and 5 show the results of the forward displacement of deaths due to the heat waves and cold spells. The mortality from malignant neoplasms and mortality in the younger age group was excluded from this analysis because of the lack of explanatory power of the regression models.

Table 4 shows a mortality deficit (all causes) in the longer lag period after the 1983, 1990, 1995, and 1997 heat waves. In the other two heat waves, there was significant excess total mortality. For the selected mortality groups, the results are inconclusive: some heat waves show a decline in mortality in the following period, while others cause an increase in mortality (some of which is significant).

Excess all-cause mortality, as well as that caused by cardiovascular and respiratory diseases, seems to continue during the whole month after the cold spells (Table 5). A clear decline in mortality, which would suggest that cold has a harvesting effect, is not evident. Thus, Table 5 shows that the cold spells studied probably did not lead to any considerable forward displacement of deaths among those who would have died in the short term anyway.

Discussion

Excess mortality during heat waves and cold spells. The 31-day moving average for the same year contains the possible excess mortality during the heat wave or cold spell days and is therefore expected to be larger than the 31-day moving average for years without extreme heat or cold during the same period (Tables 1 and 2).

A difficulty arises when one or both of the 2 years before the heat wave or cold spell year also contains a period with extreme high or low temperatures; these can influence mortality and, subsequently, the 31-day moving average of the 2 years before the heat wave or cold spell. This would lead to an underestimation of the excess mortality during the heat wave or cold spell. For example, during winter 1984–1985 a cold spell occurred during 4 January 1985–20 January 1985, while the mortality during 12 February 1985–27 February 1985 was used to determine part of the 31-day moving average of the 2 years before the cold spell in 1985–1986. It is possible that the cold spell in January 1985 had an influence on the mortality during 12 February 1985–27 February 1985, despite an intervening 3-week period. Such interference will result in a larger 31-day moving average for the 2 years before the cold spell in winter 1985–1986. However, during the heat waves (1983, 1995, 1997) and cold spell (1985–1986) for which this could be a problem, the excess mortality is still significant.

We found significant excess total mortality (8.7–24.4%) during the six heat waves in the study period. The increase in all-cause mortality was highest during the 1994 heat wave, which was also the longest heat wave.

Heat waves seem to have the potential to affect mortality from all of the selected causes. Relative excess mortality is generally largest for respiratory diseases, particularly during the 1994 heat wave, when it reached 120%. This can probably be explained by contributing factors such as high levels of air pollution.

The heat-induced mortality increases mainly occur among those ≥ 65 years of age. This group was expected to be more sensitive because, in general, the health status of older persons is more compromised than the health of younger people (*18*). For example, artheromatous arteries are much more common among older people, which exacerbates the hematologic changes induced by extreme temperatures (*7*). The elderly are also at greater risk due to a

Table 4. Difference between predicted mortality in the absence of the extreme heat and the observed mortality for time intervals after the heat waves.

	Excess[a]			
Cause/year	Lag-days[b] 1–2	Lag-days 3–6	Lag-days 7–14	Lag-days 15–30
All causes				
1982	27	25	4	112**,##
1983	36	35	–45	–289**,††
1990	142#	–7	–58	–103
1994	142	70	103**,†	275**,†
1995	30	22	–90	–278**,†
1997	117	168#	–1	–177#,†
Cardiovascular diseases				
1982	–2	37**	–4	66
1983	11	1	11	–139#,††
1990	59	–13	–62**,†	–63
1994	65	5	–2	160#
1995	11	–13	–15	–26
1997	31	37*	–7	–19
Respiratory diseases				
1982	23	14	38#,††	23**,†
1983	9	13	–6	40**,†
1990	31	7	7	–16
1994	47*	40#	70#,††	43**,†
1995	20	24*	15	12
1997	20	39*	41*	12
Age ≥ 65				
1990	150#	15	–73	–78
1994	163	113**	118**,†	403#,††
1995	22	10	–8	–169*,†
1997	133*	233#	134**	64

[a]Excess = observed number of deaths minus predicted number of deaths in absence of extreme temperatures (model). [b]Days after end of heat wave. *Student's *t*-test, $p < 0.1$. **Student's *t*-test, $p < 0.05$. #Student's *t*-test, $p < 0.01$. ##Signed rank test, $p < 0.1$. †Signed rank test, $p < 0.05$. ††Signed rank test, $p < 0.01$.

Table 5. Difference between predicted mortality in the absence of the extreme cold and the observed mortality for time intervals after the cold spells.

	Excess[a]			
Cause/years	Lag-days[b] 1–2	Lag-days 3–6	Lag-days 7–14	Lag-days 15–30
All causes				
1984–1985	80*	10	–24	71
1985–1986	234*	362#	465#,††	338#,††
1990–1991	51	–15	8	153**,†
1996–1997	90	51**	180#,††	285#,††
Cardiovascular diseases				
1984–1985	55*	48	39	62
1985–1986	112*	124#	168#,††	287#,††
1990–1991	19	24	–21	13
1996–1997	40	28	46	50
Respiratory diseases				
1984–1985	3	17*	13*	22
1985–1986	73*	101#	173#,††	67*,##
1990–1991	6	–9	–12	32
1996–1997	6	50**	50#,††	177#,††

[a]Excess = observed number of deaths minus predicted number of deaths in absence of extreme temperatures (model). [b]Days after end of heat wave. *Student's *t*-test, $p < 0.1$. **Student's *t*-test, $p < 0.05$. #Student's *t*-test, $p < 0.01$. ##Signed rank test, $p < 0.1$. †Signed rank test, $p < 0.05$. ††Signed rank test, $p < 0.01$.

reduced thermoregulatory response and less sensitive thermal perception (*2*).

We found a significant excess all-cause mortality in all of the cold spells studied, except during the winter of 1996–1997. During the cold spells that showed a significant increase in the total number of deaths, the excess mortality was between 10.1% and 26.8%. The most striking increase in mortality occurred in the winter of 1985–1986. Mortality increased significantly for all the selected causes and the percentage of excess mortality was highest in this period, compared with the other cold spells. Excess mortality due to respiratory causes during this cold spell is 117.2%. An influenza epidemic in the same period (February–March 1986) (*17*) is probably responsible for this sharp increase.

Our results show that in the absence of such an influenza epidemic, respiratory mortality does not increase during a period of extreme cold. It is possible, however, that the respiratory effects of low temperatures influence mortality after the end of the cold spells. Respiratory mortality peaks after mortality due to cardiovascular causes, suggesting different lag times in the effect of cold (*2,3,6–9,19*). Respiratory cross-infection may explain the delayed effect of cold (*3,6,8,9,17*). In the Netherlands, the occurrence of influenza and influenza-like conditions have been found to be strongly correlated with low temperatures, in particular 14 days after cold weather (*6*).

Cardiovascular mortality increased during cold spells, confirming the rapid effect of cold on this type of mortality, except during the cold spell in winter 1996–1997 (Table 2). The absence of an increase in cancer mortality during cold spells except in February 1986 suggests that unusually low temperature has no impact. Kunst et al. (*6*) stated that the relationship between mortality due to malignant diseases and cold is rather weak.

In the two cold spells for which data for the age groups are available, the excess mortality is much greater among the older age group than in the younger group. As discussed earlier, the ≥ 65-year-old group would be expected to be the most sensitive to extreme temperatures.

The lack of excess mortality during the 1996–1997 cold spell could be explained by increased influenza mortality during the last few weeks of 1995, which resulted in a higher 31-day moving average of the two preceding years of the 1996–1997 cold spell and, as a consequence, to an underestimation of the excess mortality during this cold spell.

Modeling the association between temperature and mortality. We found a V-shaped association between temperature and mortality, with mortality rates lower on days when the temperature was closer to the level corresponding to the lowest point on the curve (Figure 1). Various investigations have indicated that the prevailing climate of a geographic area may be a determinant of this optimum temperature level (*2–4,10,20*). It seems that the slope of the relationship between warmth and all-cause mortality in Figure 2 becomes steeper above a second turning point in the graph (approximately 22°C), but adding a third temperature variable, extreme heat, did not lead to an increase in the explanatory power in any of the models.

Our model on the whole data set showed an immediate effect of heat on mortality in all of the selected categories. These rapid influences are followed by compensatory effects, which suggests that some of the heat-induced increase in mortality can be attributed to those whose health was already compromised. In line with this, the largest compensatory effects relate to deaths due to presence of a malignant disease, which is often terminal.

The negative percentage effect of cold on the same day is also observed in other studies (*3,10*), but this has not been adequately explained. Furthermore, our model showed a lack of compensatory effects on mortality after exposure to cold temperatures. This supports the findings of Kunst et al. (*3*) in their study of temperature and mortality in the Netherlands.

It has been suggested that the effects of cold could be influenced by influenza and influenza-like conditions during the winter (*3,5,9,17*). Kunst et al. (*3*) found that influenza incidence in their regression models could only partly explain the effect of cold temperatures and that this was only the case for the effect of cold temperatures in the previous 7–30 days. In the same study, more than one-half of the unexplained mortality occurred within the first week, which strengthens the hypothesis that the relation between cold weather and mortality is largely attributable to the direct effects of cold. The significant increases in mortality found during the periods of extreme cold temperatures that we studied provide a further indication of the likelihood of this hypothesis being correct.

Forward displacement of deaths. Earlier studies (*2–5*) supported the hypotheses that high temperatures result in the forward displacement of deaths. Our results relating to the heat-induced forward displacement of deaths are inconclusive. Some heat waves show a decline in mortality in the longer lag periods after the extreme heat, which suggests that heat has a harvesting effect, whereas others do not show this decline in the number of deaths. It is possible that contributing factors (for example, earlier episodes of relatively warm weather) influence the potential of heat waves not merely to cause the forward displacement of deaths but to make a substantial contribution to overall lost lifetime.

We did not observe a decrease in mortality after the cold spells. This suggests that extreme cold does not lead to any mortality displacement. This contrasts with our findings and those of others relating to mortality after heat waves (*2–5*). There is a significant and relatively large excess of mortality from all causes, as well as from cardiovascular and respiratory diseases during a whole month after the 1985–1986 cold spell. This is probably due to the influenza epidemic in this period (*17*), because our regression models did not correct for the incidence of influenza. Furthermore, the relatively high excess mortality due to respiratory diseases after the 1996–1997 cold spell is probably due to the small increase in influenza incidence during the same period.

Limitations and assumptions. When considering our conclusions regarding the separate heat waves and cold spells, it is important to bear in mind that this study is based on a very small number of heat waves and cold spells. Also, these results depend strongly on the definition of heat waves and cold spells used.

One assumption was that the 31-day moving average method produces an accurate approximation of mortality in the absence of extreme temperatures. If the daily mortality numbers used for the two preceding years differ from normal values (i.e., due to high influenza incidence, very high or low temperatures), the 31-day moving average for this period can result in a biased approximation of mortality in the absence of a cold spell.

We assumed that the models were able to accurately predict mortality in the absence of heat waves and cold spells. The models for total mortality and respiratory and cardiovascular mortality, however, cannot explain 32%, 40%, and 25%, respectively, of the variation in daily mortality. The fact that we were not able to correct for the effects of influenza is one of the flaws in our models. Only the effect of temperature—controlled for time trend and seasonal influence—was determined. However, it is conceivable that the influences of other weather components should also be included [i.e., atmospheric pressure, fronts, number of hours of sunshine per day, relative humidity, wind speed (*3,11*) and air pollution (*3,11*)]. The influence of relative humidity and wind speed has been observed (*3,21*), whereas controlling for SO_2 density did not alter the relationship between mortality and temperature in the Netherlands (*3*).

Conclusions

Our modeling results support earlier findings that temperature has a relatively small influence on mortality due to malignant diseases and among those < 65 years of age. Looking at extreme temperatures, our study shows that mortality increased significantly during all of the heat waves studied. Those ≥ 65 years of age were most affected by extreme heat. The heat waves led to increases in mortality from all of the selected causes, especially respiratory mortality. The excess mortality during the cold spells was mostly attributable to the increase in cardiovascular mortality and mortality among the older group. Respiratory mortality did not seem to increase during these cold spells, but this was expected because cold has a more lagged effect on this kind of mortality. This was confirmed in our study by the positive effect of extreme cold during the longer lags in the periods studied after the cold spells.

The results concerning the forward displacement of deaths due to heat waves were not conclusive. However, looking at the relation between the ambient temperature and mortality over the whole period studied, our results showed compensatory effects on mortality in the longer lag periods after warm weather (average temperature above optimum temperature level). This could be an indication of a harvesting effect of warmer temperatures. However, as previously discussed, this was not clearly shown by the analysis of heat waves. We found no cold-induced forward displacement of deaths.

In the future, further research may produce more conclusive results. For example, the same study could be conducted over a longer period with a larger number of heat waves and cold spells. Adopting different definitions of "heat wave" and "cold spell" could lead to new insights. As an avenue for further research, we recommend using more accurate models to study the possibility of forward displacement of deaths due to extreme hot or cold periods.

The formulation of health policy could benefit from the results of research on the association between temperature and mortality (*10*). Where substantial resources are dedicated to the treatment of diseases (*9*), it may be appropriate to study prevention of these diseases by reducing the effects of temperature, especially among those at high risk. Bearing in mind that cold temperatures could be responsible for a substantial amount of lost lifetime, health policy designed to prevent the adverse effects of cold spells should be considered. For example, during the winter of 1986–1987 a media campaign in the United Kingdom, which advised elderly people to avoid outdoor exposure, was accompanied by a dramatic fall in winter deaths compared to the numbers predicted by the trend over the previous decade (*7*).

The results of our study may also shed new light on future changes in mortality related to the global warming predicted by several climate models. Although global climate change is likely to be accompanied by an increase in the frequency and intensity of heat waves, winters will likely be milder as well. Therefore, it is possible that in some regions, including the Netherlands, a continuing decreasing trend in winter mortality would tip the scales to the increasing excess summer mortality rates. However, the overall balance would also depend on adaptive responses and future health levels, as people will acclimatize to warmer climates via a range of behavioral, physiologic and technologic adaptations.

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