Relationship Between Cold Temperature and Cardiovascular Mortality, With Assessment of Effect Modification by Individual Characteristics  
– Ibaraki Prefectural Health Study –

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Background: Cold temperature has been reported to contribute to cardiovascular mortality, but it is not clear which people are more susceptible to cold temperature.

Methods and Results: The relationship between ambient temperature and mortality was examined in 3,593 subjects from the Ibaraki Prefectural Health Study who died of cardiovascular disease during a mean follow-up period of 9.7±4.0 years. Daily values of meteorological variables were obtained from the Japan Meteorological Agency. Time-stratified case cross-over analysis was used. The multivariate odds ratios (ORs; 95% confidence interval) per 1°C decrease in daily maximum temperature over the day of death and the 2 days prior to this day adjusted for relative humidity were 1.018 (1.003–1.034) for all cardiovascular deaths and 1.025 (1.003–1.048) for stroke deaths. Risk-stratified analysis showed that younger subjects aged <80 years and those with hyperglycemia were more susceptible to cold temperature. The OR of all cardiovascular deaths related to cold temperature was 1.034 (1.012–1.056) for subjects aged <80 years, and that of stroke deaths was 1.076 (1.023–1.131) for those with hyperglycemia.

Conclusions: Exposure to cold temperature triggers cardiovascular deaths. Additionally, younger age and hyperglycemia could enhance susceptibility to cold temperature. (Circ J 2013; 77: 1854–1861)

Key Words: Air temperature; Cardiovascular mortality; Cohort study; Cold weather

Weather conditions are known to be an important factor that can affect the morbidity and mortality of a population.1–3 In many studies from North America and Europe, decreasing temperature has been reported to be associated with increases in mortality.1–6 Some studies focused on the association between cold temperature and cause-specific mortality, and the short-term effect of cold weather is generally apparent in mortality due to cardiovascular disease (CVD).2 Previous studies have shown evidence of an association between increased cardiovascular mortality and cold temperature.6–11 Although some studies have suggested that temperature plays an important role in the seasonal variation of cardiovascular mortality, given the higher rate in winter,12,13 few studies have examined the association between ambient temperature and cardiovascular mortality or morbidity in Japan.14 It is necessary to be able to identify which people are easily influenced by weather conditions. Most previous studies examining the association between cardiovascular mortality and meteorological factors used vital statistics from the study area. Daily mortality data are sufficient to examine the relation between ambient temperature and mortality but do not allow identification of potentially susceptible people. Although some of the previous studies using vital statistics focused on sex and

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specific age groups, the dataset does not include detailed individual information on lifestyle and comorbidity. Thus, there are few findings describing which people are more susceptible to cold temperature and how susceptible they are. To promote behavior to prevent cardiovascular mortality related to cold temperature, it is important to identify the potentially susceptible people.

The Ibaraki prefectural government started a large cohort study, known as the Ibaraki Prefectural Health Study (IPHS), to obtain information on health status for the purposes of health education and policy making in 1993. Analysis of the data from this large cohort with individual-level information may help identify factors that affect susceptibility to cold temperature. Accordingly, we decided to use this cohort data to more precisely assess the relationship between air temperature and cardiovascular mortality. The aim of this study was to investigate the relationship between ambient temperature and cardiovascular mortality. Moreover, to identify those people who are susceptible to weather conditions, we conducted stratified analyses of risk factors that could be associated with cardiovascular mortality related to cold temperature.

**Methods**

**Subjects**

We used baseline subject information from the Ibaraki Prefectural Health Study. Details of this cohort study have been reported elsewhere.\(^{15-17}\) Briefly, the study group consisted of 97,042 individuals (33,130 men and 63,912 women) aged 40–79 years at the time of study entry who resided in Ibaraki prefecture and completed an annual community-based health checkup conducted by the Ibaraki Health Service Association in 1993. This study was approved by the Ethics Committee of Ibaraki Prefecture.

Baseline measurements were conducted in 1993. Blood pressure was measured on the right arm of seated subjects by trained observers using standard mercury sphygmomanometers. Height in stocking feet and weight in light clothing were measured, and body mass index (BMI) was calculated as weight (kg) divided by height squared (m\(^2\)). Blood samples were drawn from seated subjects. Plasma glucose level was measured by means of a glucose oxidase electrode method. An interview was conducted to ascertain smoking status, daily alcohol intake, and history of pre-existing diseases.

On the basis of the results of this health checkup and questionnaire at baseline, we classified the subjects into 2 groups with regard to each of their risk factors. Smoking habit was classified into current smoker and non-current smoker, and alcohol intake was classified into regular intake (almost every day) and none/occasionally. Hypertension was defined as systolic blood pressure >140 mmHg, diastolic blood pressure >90 mmHg, or medication for hypertension, and in that case the subject was classified as hypertension (+). Hyperglycemia was defined as fasting glucose >110 mg/dl, non-fasting glucose >140 mg/dl, or treatment for diabetes, in which case the subjects was classified as hyperglycemia (+).

During the 1993–2008 follow-up period, cohort mortality data were obtained from a systematic review of death certificates, which are all forwarded to the local public health center of every community in Japan. These mortality data were centralized at the Ministry of Health, Labor and Welfare, where the underlying causes of death were coded for the National Vital Statistics records according to the International Classification of Diseases (ICD), 9th (1993–1994) and 10th (1995–2008) revisions. On the basis of the 10th revision of the ICD coding, cause-specific mortality was classified into deaths due to CVD (100–199), stroke (160–169), and cardiac disease (CD; 100–152 except for 110–113). We excluded subjects with incomplete data or those who had moved out of their community. During the follow-up period, a total of 11,246 deaths (5,066 men and 6,180 women) due to all causes except for injuries were observed, of which 3,593 deaths from CVD were included in analysis.

**Environmental Data**

Data on meteorological variables were obtained from the Japan Meteorological Agency. We calculated the daily ambient temperature and relative humidity using hourly measurements at the Mito local meteorological observatory located in Mito city and assigned them to all subjects. We also obtained data on air pollutants from the atmospheric environment database of the National Institute for Environmental Studies. Air pollutants included suspended particulate matter (SPM), photochemical oxidants (Ox), nitrogen dioxide (NO\(_2\)), and sulfur dioxide (SO\(_2\)). Under the Japanese Air Quality Standard, SPM is defined as airborne particles with a 100% cut-off level at an aerodynamic diameter of 10 \(\mu\)m. SPM monitored in Japan is theoretically assumed to be particles approximately 7-\(\mu\)m diameter, with a 50% cut-off level. Ox is defined as mixtures of ozone and other secondary oxidants generated by a photochemical reaction, which is considered as a proxy for ozone. For each city, we selected the monitoring station nearest that city and calculated 24-h mean concentrations of SPM, NO\(_2\), and SO\(_2\), and the 1-h maximum of the day from hourly measurements of Ox. We assigned to each subject the daily values of air pollutants of their corresponding city.

**Statistical Analysis**

We applied a time-stratified case cross-over design to examine the association between ambient temperature and cardiovascular mortality. A case cross-over analysis was developed to assess acute effects of transient exposure\(^{18}\) and was widely used to examine the association between short-term effects of environmental factors.\(^{18}\) Within-subject comparisons were made between a case period and control periods in this design. A case period was defined as the day of death. The selection of appropriate control periods is important for obtaining valid estimates. We defined the control periods as the same day of the week as the case period day in the same month and year of the case period (eg, if the second Friday of the month is defined as the case period, the other Fridays in the month are defined as the control periods). This control selection strategy was expected to adjust for the effects of long-term trend, seasonality, and day of the week by design.\(^{20}\) We estimated the odds ratio (OR) and 95% confidence interval (CI) of mortality associated with a change in maximum ambient temperatures using conditional logistic regression. We controlled for relative humidity and each air pollutant.

It was previously reported that the relationship between ambient temperature and mortality is non-linear, that is, it is either V-, U- or J-shaped in North America and Europe.\(^{1,2} \) In Japan, Honda et al also noted a V-shaped pattern of temperature and mortality, with optimum temperature as the point at which the mortality rate is lowest.\(^ {21}\) According to their analyses with data from 47 prefectures, an 80th or 85th percentile of daily maximum temperature was the best indicator of the optimum temperature. We referred to the V-shaped relationship with the breakpoint temperature between the 80th and 85th percentiles of daily maximum temperature during the study period and estimated the OR per 1°C decrease (cold effect).
and 1°C increase (heat effect) from the breakpoint temperature. Because the effect of cold temperature on mortality is reported to persist for a lag period of >1 week,4,22,23 in this model we examined lag for up to 10 days separately. To take into account multi-day lag effects, we also estimated the effects for a few days of moving-average temperature.

We also repeated the analysis for each risk factor. To test whether there were any differences in temperature effect between subgroups, an interaction term between low outdoor temperature and each risk factor was added to the model. P<0.05 was considered to indicate statistical significance. All statistical analysis was conducted with SAS version 9.1.3 (SAS Institute, Cary, NC, USA).

Results
Mortality and Environmental Data
Subject characteristics are listed in Table 1. Among the total of 11,246 deaths, 3,593 deaths were due to CVD, of which 1,709 were due to stroke, and 1685 were due to CD. The mean±SD of age for all 11,246 dead subjects was 76.8±8.8 years with a range of 41–94 years (median, 78 years). Mean follow-up period was 9.7±4.0 years. BMI, smoking habit, alcohol intake, and the existence of hypertension and diabetes for each cause of mortality are also listed in Table 1. For each cause of mortality, almost 30% of subjects were obese (BMI ≥25 kg/m²), 30% were current smokers, and almost 20% had regular alcohol intake. Almost 70% of the subjects had hypertension and 20–25% had hyperglycemia.

Maximum Temperature and Cardiovascular Mortality
The results for cold effect on univariate and multivariate analysis adjusted for relative humidity are summarized in Figure 1, which shows ORs of deaths due to CVD, stroke, and CD in association with a 1°C decrease of maximum temperature on the day of death (lag0) to 10 days prior to death (lag10). Generally, cold temperature was associated with increased mortality due to CVD and stroke. On univariate analysis, the asso-

Table 1. Subject Characteristics

<table>
<thead>
<tr>
<th>All CVD</th>
<th>Stroke</th>
<th>CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1,427 (39.7)</td>
<td>675 (39.5)</td>
</tr>
<tr>
<td>Female</td>
<td>2,166 (60.3)</td>
<td>1,034 (60.5)</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>18 (0.5)</td>
<td>8 (0.5)</td>
</tr>
<tr>
<td>50–59</td>
<td>102 (2.8)</td>
<td>49 (2.9)</td>
</tr>
<tr>
<td>60–69</td>
<td>345 (9.6)</td>
<td>147 (8.6)</td>
</tr>
<tr>
<td>70–79</td>
<td>1,309 (36.4)</td>
<td>641 (37.5)</td>
</tr>
<tr>
<td>80–89</td>
<td>1,654 (46.0)</td>
<td>797 (46.6)</td>
</tr>
<tr>
<td>≥90</td>
<td>165 (4.6)</td>
<td>67 (3.9)</td>
</tr>
<tr>
<td>Obesity BMI ≥25 kg/m²</td>
<td>998 (27.8)</td>
<td>439 (26.7)</td>
</tr>
<tr>
<td>Habit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoking</td>
<td>1,166 (32.5)</td>
<td>549 (32.1)</td>
</tr>
<tr>
<td>Regular alcohol intake</td>
<td>740 (20.6)</td>
<td>361 (21.1)</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>2,599 (72.3)</td>
<td>1,225 (71.7)</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>829 (23.1)</td>
<td>359 (21.0)</td>
</tr>
</tbody>
</table>

Data given as n (%). BMI, body mass index; CD, cardiac disease; CVD, cardiovascular disease.

Table 2. Summary of 1993–2008 Weather and Environmental Data (n=6,210)

| Mean temperature (°C) | 13.9 | 8.1 | –2.8 | 6.3 | 14.3 | 20.6 | 31.3 |
| Maximum temperature (°C) | 18.5 | 7.7 | –0.2 | 11.7 | 18.7 | 24.5 | 37.8 |
| Minimum temperature (°C) | 9.7 | 8.9 | –8.9 | 1.4 | 9.9 | 17.6 | 27.0 |
| Relative humidity (%) | 74.0 | 12.6 | 29.1 | 65.8 | 75.6 | 83.5 | 99.9 |
| Mean SPM (μg/m³) | 28.9 | 16.7 | 3.8 | 17.4 | 24.9 | 36.1 | 191.0 |
| Maximum Ox (p.p.b.) | 48.5 | 16.4 | 8.5 | 38.0 | 45.5 | 56.5 | 129.8 |
| Mean NO2 (p.p.b.) | 12.4 | 5.2 | 2.5 | 8.8 | 11.4 | 15.1 | 41.9 |
| Mean SO2 (p.p.b.) | 3.9 | 1.3 | 1.2 | 3.1 | 3.8 | 4.5 | 26.7 |

Ox, photochemical oxidant; NO2, nitrogen dioxide; p.p.b., parts per billion; P25, 25th percentile; P75, 75th percentile; SO2, sulfur dioxide; SPM, suspended particulate matter.
In comparison with cold temperature effect on cardiovascular mortality, there were no specific trends for hot temperature effect in this study (Figure S1). Therefore, we mainly evaluated and discuss cold effects.

**Risk Stratification**

The subjects were then divided into 2 groups by each risk factor, and the analyses were repeated. Because we observed that the effect of cold temperature persisted for a few days, we used lag0–2 data for these analyses. The results of the stratified analyses of daily maximum temperature adjusted for relative humidity at lag0–2 are summarized in Figure 2. We observed significant associations of cold temperature with CVD mortality for the subjects aged <80 years (OR, 1.034; 95% CI: 1.012–1.056), for those with BMI <25 kg/m² (OR, 1.019; 95% CI: 1.001–1.038), and for those with hypertension (OR, 1.018; 95% CI: 1.000–1.037), and with mortality due to stroke for those with hyperglycemia (OR, 1.076; 95% CI: 1.023–1.131). Among them, the difference between subgroups was significant for age (P for interaction: 0.046) and for hyperglycemia (P for interaction: 0.035). The differences were not evident.
temperature. We observed that cold temperature was associated with increased mortality from CVD. The association was strongest at lag1 and lag2. Risk-stratified analysis identified the subjects aged <80 years and those with hyperglycemia as being more influenced by cold temperature. We did not observe evident differences in effect of cold temperature on cardiovascular mortality between the subgroups stratified by obesity, smoking habit, alcohol intake, and hypertension.

Table 3. Maximum Temperature and CV Death Adjusted for Environmental Factors†

<table>
<thead>
<tr>
<th></th>
<th>All CVD</th>
<th>Stroke</th>
<th>CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjusted for SPM</td>
<td>1.013 (0.996–1.030)</td>
<td>1.014 (0.991–1.040)</td>
<td>1.003 (0.979–1.028)</td>
</tr>
<tr>
<td>Adjusted for NO2</td>
<td>1.015 (0.999–1.031)</td>
<td>1.022* (1.000–1.046)</td>
<td>1.002 (0.980–1.025)</td>
</tr>
<tr>
<td>Adjusted for SO2</td>
<td>1.020* (1.004–1.037)</td>
<td>1.023 (0.999–1.048)</td>
<td>1.010 (0.988–1.035)</td>
</tr>
<tr>
<td>Adjusted for Ox</td>
<td>1.019* (1.003–1.035)</td>
<td>1.023* (1.001–1.047)</td>
<td>1.007 (0.984–1.030)</td>
</tr>
</tbody>
</table>

*P<0.05. †Adjusted for relative humidity and each air pollutant, averaged from lag 0–2. CI, confidence interval; CV, cardiovascular; OR, odds ratio. Other abbreviations as in tables 1,2.

Figure 2. Odds ratios (ORs) and 95% confidence intervals (whisker bars) for deaths due to cardiovascular disease (CVD), stroke, and cardiac disease (CD) in association with a 1°C decrease of maximum temperature adjusted for relative humidity at lag0–2. (A) Age, (B) obesity, (C) smoking, (D) alcohol intake, (E) hypertension, and (F) diabetes. BMI, body mass index. *P<0.05; †P for interaction <0.05.

Discussion

In this study, we combined daily meteorological data and mortality data from a large community-based cohort and evaluated the relationship between cardiovascular mortality and ambient temperature. We observed that cold temperature was associated with increased mortality from CVD. The association was strongest at lag1 and lag2. Risk-stratified analysis identified the subjects aged <80 years and those with hyperglycemia as being more influenced by cold temperature. We did not observe evident differences in effect of cold temperature on cardiovascular mortality between the subgroups stratified by obesity, smoking habit, alcohol intake, and hypertension.
Many previous studies have already indicated a relationship between ambient temperature and cardiovascular mortality.\textsuperscript{5-11} To our knowledge, however, there are no studies using cohort data to evaluate the effects of environmental factors on cardiovascular deaths. Using cohort data should make it possible not only to discover a relationship between cause-specific mortality and ambient temperature but also to evaluate the risk factors that could modify the association between mortality and ambient temperature. The present results suggested that subjects with certain risk factors were more susceptible to the effect of cold temperature. Thus, this is an important advantage of using a cohort study to assess the short-term effects of environmental factors.

Like these other previous studies,\textsuperscript{5-11} an association between decrease in maximum temperature and increase in mortality from CVD was seen in the present study. Various mechanisms are proposed to explain the increase in cardiovascular mortality associated with cold temperature. In experimental studies, exposure to cold temperature induces increases in heart rate, blood pressure, peripheral vasoconstriction, plasma fibrinogen concentration, blood cholesterol level, platelet viscosity, and red cell count.\textsuperscript{28-26} These changes in sympathetic nervous system and hemodynamic response to cold stress may contribute to morbidity and mortality related to cold temperature.

The present results also suggest that even when the effect of ambient temperature was adjusted for some air pollutants, the association between the cold effect and mortality from CVD remained. It was reported that air pollutants have short-term effects of ambient temperature but also to evaluate the risk factors that could modify the association between mortality and ambient temperature. The present multivariate analysis including relative humidity and air pollutants, which are potential confounders, may provide a more accurate analysis of the effects of ambient temperature on mortality.

This study, however, did not identify a hot temperature effect on CVD mortality. Some previous studies have reported that extremely hot weather could increase cardiovascular mortality.\textsuperscript{29,30} In the present study, we chose 27°C as a breakpoint for V-shaped analysis according to the previous study in Japan.\textsuperscript{21} This breakpoint may be too low to examine the effect of extremely hot temperature. The number of days with extremely high temperature, however, was so limited that we were unable to obtain a valid estimate. For more detailed evaluation of the hot temperature effect, a larger sample size is needed.

Although the effect of cold temperature on CVD mortality was more pronounced for those aged <80 years, in the present study it was not clear for those who died who were aged >80 years. In general, the elderly have long been considered physiologically susceptible to cold stress.\textsuperscript{31} Indeed, several epidemiological studies have reported that elderly people are more sensitive to cold weather.\textsuperscript{2,6,10} O’Neill et al. however, found stronger cold-related effects on mortality in younger subjects\textsuperscript{32} such as those in the present study. One possible explanation as to why the elderly people in the present study were less influenced by cold temperature might be that the elderly group included more people who were confined to bed or stayed in homes with heating systems during the cold winter. In contrast, the younger people tended to be outside more than the elderly and may have had more chance to be exposed to ambient temperatures that could make them more influenced by cold temperature.

Hyperglycemia may also enhance the effect of cold temperature on mortality due to stroke. Diabetes is characterized by a chronic state of low-grade inflammation, endothelial dysfunction, and hypercoagulability,\textsuperscript{33} and it was already reported that several factors, such as oxidative stress and protein kinase C, may contribute to macrovascular injury from hyperglycemia. Under these conditions of injured vasculature, cold temperature could more seriously affect patients with hyperglycemia.\textsuperscript{34} The reason why the effect of hyperglycemia was limited to stroke is still unclear.

A cohort study of male Japanese workers found that smoking, even at a low level, increased the risk of cardiovascular death.\textsuperscript{35} Another study observed greater seasonal change in blood pressure and heart rate for smokers and suggested that cardiovascular response to cold stress is enhanced by smoking.\textsuperscript{36} As noted here, cold temperature increases the activity of the sympathetic nervous system.\textsuperscript{25,26} A clinical study also showed that smoking has both acute and chronic effects on arterial stiffness.\textsuperscript{37} Given that the effects of smoking and cold temperature interact, it is possible that exposure to cold temperature could increase the cardiovascular response, which could result in more serious outcomes for smokers than for non-smokers. There were no significant effects, however, of smoking habit on cold-related CVD mortality in the present study, suggesting that smoking habit could have changed during the follow-up period, and there might be some misclassification in this study.

With respect to alcohol intake, it was reported that the use of alcohol in a cold environment could lead to an increase in heart rate, so there may be an unfavorable response by the cardiovascular system to alcohol intake in cold temperatures.\textsuperscript{38} We did not, however, see a clear difference in the effect of cold temperature on CVD mortality in relation to alcohol intake.

Hypertension is one of the main risk factors for cardiovascular mortality, and risk-stratified analysis in this study also suggested that CVD mortality in patients with hypertension was influenced by cold temperature. As noted here, cold temperature may increase blood pressure,\textsuperscript{9,26} therefore, it is reasonable that subjects with hypertension may be more sensitive to effects of cold temperature. The difference in OR, however, between hypertension subjects and non-hypertension subjects was very small. The stratification of hypertension was based on blood pressure, which was measured at the time of the health checkup. While some subjects with normal blood pressure at the checkup may have gone on to develop hypertension, others with high blood pressure may have successfully controlled their blood pressure during follow-up. There also may have been subjects with “white coat” hypertension that was misclassified as hypertension. Thus, such misclassifications may have affected the results.

**Study Limitations**

One of the limitations of the present study is that we could not elucidate the effect of extremely hot temperature on cardiovascular mortality. Separate analysis for cold and warm season may provide more precise information, especially on the effect of hot temperature. The present small study, however, did not allow for such analysis because the number of days with high temperature is low. Another limitation is that we could not adjust for influenza epidemics in this study. Influenza epidemics usually occur in winter and could confound the effect of cold temperature. It is also possible that upper respiratory tract infection could have adverse effects on the cardiovascular system during the cold season. Thus, studies examining the association between cold temperature and respiratory disease are needed.

We could not find a significant relationship between temperature and CD mortality in this study. Because the CD cat-
egory contains various etiologies such as coronary artery disease, heart failure, arrhythmia, and valvular disease, such etiologic heterogeneity of CD may have obscured the true relationship with temperature. The relationship between temperature and mortality due to hypertensive disease and atherosclerotic disease (110-113, and I70-79, respectively) was another subject of interest, but the number of subjects who could be assessed according to these 2 detailed classifications was too small.

Moreover, behavioral changes related to avoidance of cold outdoor temperatures may have obscured the effect of cold temperature on CVD mortality. In particular, the extent of exposure misclassification might be pronounced among the elderly subjects, who generally spend most of their time indoors. These factors were not considered in this study, so they would have modified the results. But because information on individual behavior is difficult to obtain, this point is an important limitation of a prospective cohort study.

In this study, we used fasting glucose >110 mg/dl and non-fasting glucose >140 mg/dl as the criteria for hyperglycemia. These were not the same as established criteria for diabetes mellitus (ie, fasting glucose >125 mg/dl and non-fasting glucose >200 mg/dl). Had we used the standard diabetes criteria, the subjects with diabetes would have comprised approximately 10% of the total subjects, which is too small a number of subjects to evaluate the effect of diabetes. Therefore, we used the hyperglycemia criteria mentioned here.

For risk-stratified analysis, the cohort data included information on risk factors at the time of the 1993 health checkup, and these factors may have changed during the follow-up period. This could lead to misclassification of category in stratified analysis and could have obscured the effect modification by individual characteristics. Unfortunately, however, information on risk factors for each subject could not be obtained from daily mortality data of each area, so this point was also a limitation of the study design. To solve these problems, more detailed modeling and cohort data would be required.

Conclusions

Short-term exposure to cold temperature increased cardiovascular mortality, and subjects with hyperglycemia were more strongly affected by cold temperature. The mechanism of susceptibility may be complex, and further effort is needed to identify the clinical factors responsible for susceptibility to cold temperature. Knowledge of the effects of meteorological factors on various health outcomes and of those who are more susceptible to meteorological factors, however, would facilitate effective preventive behavior.

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Disclosures

None.
Cold Temperature and Mortality


Supplementary Files

Supplementary File 1

Figure S1. Odds ratios and 95% confidence interval for each cause of mortality per 1°C increase of maximum temperature from the breakpoint (27°C) on the day of death (lag0) to 10 days prior to death (lag10).

Please find supplementary file(s): http://dx.doi.org/10.1253/circj.CJ-12-0916