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The role of air pollution in the relationship between a heat stress index and human mortality in Toronto

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Abstract

In this study we considered confounding from air pollutants and chronological variables in the relation between humidex, a summer temperature and humidity index, and nonaccidental mortality, from 1980–1996 in Toronto, Canada. Changes in the risk of death by age group, gender, and combined cardiac-respiratory cause of death were estimated for both 1°C and 50–95th percentile increases in humidex using a generalized additive linear model. With air pollution terms in the models, relative risk (RR) point estimates narrowly exceeded 1.0 for all groups. Humidex effects were most apparent for females (RR = 1.006, 95% CI = 1.004–1.008 per 1°C humidex and RR = 1.089, 95% CI = 1.058–1.121 for 50th to 95th percentile humidex). When air pollution was omitted from the model, RR in the 50–95th percentile analysis increased less than 1.71% for all groups except females, for which RR decreased 1.42%. Differences in RR per 1°C humidex were all less than 0.12%. Confidence intervals narrowed slightly for all groups investigated. Heat stress has a statistically significant, yet minimal impact on Toronto populations, and air pollution does appear to have a small, but consistent confounding effect on humidex effect estimates.

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Keywords: Heat stress; Humidex; Mortality; Air pollution; Toronto

1. Introduction

Human susceptibility to weather is considerable. Temperature and mortality studies that have controlled for confounding from season and disease have reported a U, J, or V-shaped relationship with minimum risk of mortality between 17° and 25° and increasing risk as temperature rises or falls (Knox, 1981; Kunst et al., 1993; Pan and Li, 1995; Braga et al., 2001; Curriero et al., 2002). More deaths occur in winter than in summer, presumably due to the incidence of infectious disease. However, summer heat waves can cause many deaths over a short period of time. Oppressive summer conditions, represented as either dry-bulb temperature or a multiple-variable weather index, can trigger heat stress that results in short-term increases in morbidity and mortality (Kalkstein and Valimont, 1986; Douglas and Al-Sayer, 1991; Smoyer et al., 2000a, b). Since the 1995 Chicago heat wave, which resulted in hundreds of excess deaths over the course of several days (CDC,

1995; Semenza et al., 1996), an increasing number of research efforts have focused on comprehensive reviews of retrospective climate/health associations (WHO/WMO/UNEP, 1996; Smith et al., 1998). Although most air pollution/health studies control for weather, few heat/health studies have controlled for the effects of air pollution.

Since air pollution levels are a function of atmospheric conditions, it is important to take both into consideration when examining the effects of weather on human health. We hypothesize that failing to account for air pollution may overestimate mortality risk estimates from weather. Thus, in our study of Toronto we employed a time-series approach to estimate the difference in relative risk of humidex (a hot weather index of temperature and humidity) on total nonaccidental deaths, controlling for the potential confounding effects of urban air pollution.

Toronto is Canada's largest city, with approximately 4.26 million people in the greater metropolitan area (Statistics Canada, 2001), and it is situated on the north shore of Lake Ontario (43°40'N). Toronto has cold winters and is susceptible to hot, humid summers,

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similar to US cities like Buffalo and Boston (Environment Canada, 2001). Air pollution levels in the Toronto area are relatively low, owing to government-regulated emission controls (Kenndall, 1993; OME, 1996); however, smog episodes are common during extended periods of warm weather. Climate model simulations for Southern Ontario under doubled CO₂ scenarios have estimated a warming of 2–5°C by the end of the 21st century (Smith et al., 1998). Hence, Toronto is likely to experience more frequent episodes of hot and humid summer weather. Annual mean ozone (O₃) levels have been increasing throughout Southern Ontario (Fig. 1) and are expected to increase further under climate change. The possible synergistic effects of air pollutants, including O₃, and heat stress on human mortality are uncertain.

1.1. Heat and health studies

Extreme heat is a well-known cause of heat stroke, heat syncope (fainting), and heat cramps, and it also exacerbates many preexisting health conditions. High risk populations include the elderly (Kilbourne et al., 1982; Macey and Schneider, 1993; Mackenbach and Borst, 1997), those on certain medications (Kilbourne et al., 1982; Semenza et al., 1996), and those with preexisting illnesses (Auliciems and Frost, 1989; Marshall et al., 1988; Enquesselassie et al., 1993; Khaw, 1995), particularly if they reside in cities (Kalkstein and Davis, 1989; Smoyer, 1998a, b). Prolonged hot and humid conditions are more stressful to human health than isolated hot days (Kalkstein and Smoyer, 1993a, b; Smoyer-Tomic and Rainham, 2001). It has also been suggested that the timing of exposures to hot weather is important, with heat waves occurring early in the season

having higher associated mortality than those later in the season (Kalkstein, 1993). Protective factors include access to air conditioning (Greenberg et al., 1983), low-rise housing, and the ability to avoid exposure (Semenza et al., 1996). In an effort to prevent heat-related illnesses and deaths, a number of cities across North America and in Europe have begun to develop and implement hot-weather response plans (Kalkstein et al., 1996; Sheridan and Kalkstein, 1998; Kalkstein, 1998). Many of these cities, including Toronto, also have air pollution advisories; however, most are separate from heat advisories. In response to an extended episode of hot weather in the summer of 1999, Toronto Public Health developed a heat response plan (Toronto Public Health, 1999). Thus, Toronto has systems in place that can issue pollution advisories or respond to heat events, but to date the two have not been linked.

1.2. Air pollution and health research

The potential magnitude of effect modification and/or interaction between air pollution and temperature or other weather variables remains unclear. Studies of the effects of air pollution on human mortality statistically control for weather variables (typically temperature) when deriving modeled risk estimates. Some of these studies have postulated combined temperature/pollutant effects even after controlling for the confounding effects of weather. For example, Lebowitz et al. (1973) identified a relationship between acute respiratory episodes and days with high air pollution, low temperatures, and high barometric pressure in New York City. Katsouyanni et al. (1993) reported a possible synergistic effect of air pollution and high air temperatures on human mortality in Athens. In Japan, the combined

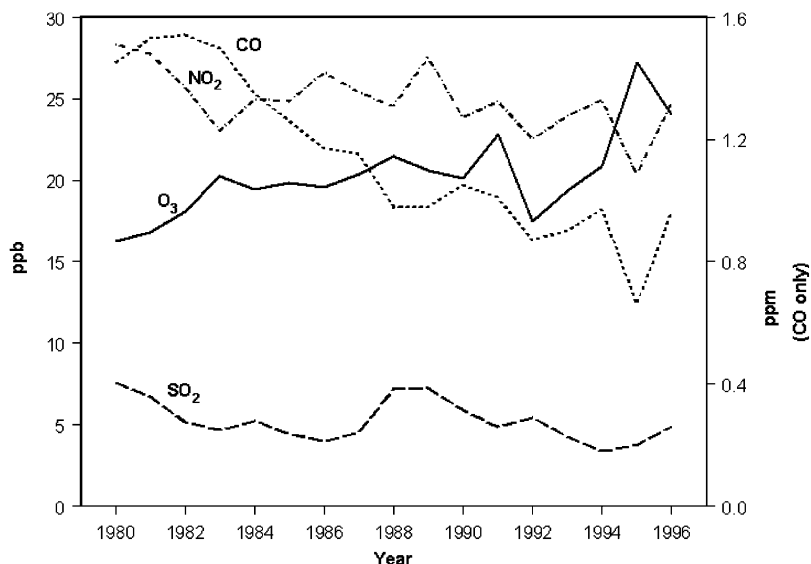


Fig. 1. Average ambient annual air pollutant concentrations for Toronto, Canada, from 1980 to 1996.

effect of nitrogen dioxide (NO₂) and high temperatures has been associated with lung cancer (Choi et al., 1997) and heat stroke (Piver et al., 1999). Evidence of interaction between total suspended particulates (TSP) and temperature has also been reported, with the association between mortality and TSP increasing in strength above 29°C (Wyzga and Lipfert, 1994). In a study of the effects of O₃ concentrations on daily mortality in Rotterdam, Biersteker and Evendijk (1976) found the relation between ozone and mortality to be inconclusive due to potential confounding by temperature. Styer et al. (1995) reported confounding in the summer months between weather and particulate matter (PM₁₀) and between weather and mortality in Cook County. Other research has revealed strong seasonally dependent associations between carbon monoxide (CO) and human mortality (Burnett et al., 1998a, b). In a US study of Birmingham and Philadelphia, Smoyer et al. (2000) found that hot and humid air masses had a greater impact on mortality than high concentrations of TSP or O₃. In Philadelphia, TSP and O₃ levels did not affect mortality when the hot and humid air mass was present, while in Birmingham they did (Smoyer et al., 2000). However, statistically significant effect modification by season has been reported for the association of mortality with particulate air pollution in Philadelphia (Samet et al., 1995). Many air pollution studies have addressed weather variables in some way (Dominici et al., 2000; Samet et al., 2000). However, few studies on the effects of weather on health have examined the role of air pollution in the relationship. Our objective is to identify the importance of air pollution confounding on humidex/mortality relationships in Toronto for several demographic groups and causes of death. Our results will contribute to ongoing discussions of the relative importance of weather, and air pollution, in acute human mortality.

2. Materials and methods

We analyzed daily counts of nonaccidental deaths (*International Classification of Diseases, Ninth Revision [ICD-9]* codes <800), which we categorized by census subdivision for residents of the City of Toronto from January 1980 to December 1996. This includes the census subdivisions of Toronto, Etobicoke, York, North York, East York, and Scarborough, which in 1996 had a combined population of 2,385,421 with 319,830 persons 65 years and older. The data were grouped by age (<65 years and ≥65 years), gender, and selected ICD9 codes for cause of death: combined cardiac and respiratory (390–459; 480–519) and other nonaccidental mortality unrelated to cardiac or respiratory causes. The final mortality series was structured to examine summer-only mortality/humidex associations so that days before May 1 or after September 30 were assigned missing values for modeling purposes, for a total of 2601 days.

The Meteorological Service of Canada provided hourly dry-bulb and dewpoint temperatures from a climate station at the Toronto Pearson International Airport, located approximately 15 km from the urban core. Daily values of humidex were calculated using the daily maximum dry-bulb temperature and the dewpoint temperature that occurred at the same hour as the maximum air temperature (Masterton and Richardson, 1979). Humidex is conceptually similar to apparent temperature, also known as the heat index. Like the heat index, humidex is a measure of thermal discomfort. Air of a given temperature and moisture content is equated in comfort to air with a higher temperature with negligible moisture content (Phillips, 1990). The Meteorological Service of Canada uses humidex to advise the public of the potential health risks associated with summer weather conditions (Table 1, second column).

Table 1
Comfort levels, health outcomes associated with different values of humidex, and days falling into each category May 1 through September 30 1980–1996

Humidex range in °C (°F)	Health Impact ^a	Number of days (%)	Mean days/summer	Year with least and most occurrences of humidex range (days in year)
<29 (<86)	No discomfort	1841 (70.8)	108.3	1991 (91 days) 1982/1992 (129 days)
30–39 (86–103.9)	Some discomfort	722 (27.8)	42.5	1982/1992 (23 days) 1991 (59 days)
40–45 (104–113)	Great discomfort	36 (1.4)	2.2	1984–85, 1989–90, 1996 (0 days) 1988 (8 days)
>45 (>113)	Dangerous	2 (0.1)	0.1	All but 1990 and 1995 (0 days)
>54 (>129.2)	Heat stroke imminent	0	0	1990 and 1995 (1 day)

°C = Celsius; °F = Fahrenheit.

^aSource: Environment Canada, http://www.msc-smc.ec.gc.ca/cd/humidity_e.cfm2.

Daily average concentrations of O₃, SO₂, NO₂, and CO were used to control for potential confounding effects of air pollution. Air pollution data were obtained from the National Air Pollution Surveillance Network (NAPS) database maintained by Environment Canada. Concentration values were derived from six ambient location monitors and averaged to provide a daily measurement. Respirable particle data (PM₁₀) were only available every six days and were not included in this study because short-term and acute effects would be difficult to capture without daily data.

Log-linear models with Poisson error were used to estimate the daily relationship between humidex and mortality and to account for time-varying trends, day of the week, and air pollution (Dominici et al., 2002; Curriero et al., 2002). The core model has the form

$$\log(\mu_t) = \beta \text{humidex}_t + \text{confounders}_t, \quad (1)$$

where β represents the log rate of mortality associated with the corresponding humidex value. We estimated the humidex effect using only short-term (daily) variations in mortality and humidex and controlling for potential confounding of humidex by longer term trends, such as those due to public health epidemics, summer smog events, and cyclical trends related to season and day of the week. Natural cubic splines of the form $ns(\text{time}, df)$ were used to approximate fluctuations of mortality over time, where the degrees of freedom (df) stipulate the smoothness of the parameter in the model. Time refers to the day in the study period with the first day labeled as 1 and each following day labeled sequentially for the 17-year period. We used a combination of measures, including partial autocorrelation plots, a modified Bartlett's Test (to examine residual structures), and Akaike's Information Criterion (AIC), to explore the relation between mortality and potential time-related confounders. Based on these diagnostics we selected eight degrees of freedom per year for total mortality and six degrees of freedom for all other mortality outcomes, for a total of 136 and 102 df , respectively, over the 17 years of record. The number of degrees of freedom selected adequately controlled for smooth seasonal variations in mortality.

After controlling for long-term and seasonal trends, we focused our analysis on air pollution variables that were found to be the best predictors of summer mortality. To control for air pollution, we also considered parametrically smoothed functions of average measurements of ozone, sulfur dioxide, carbon monoxide, and nitrogen dioxide for the same day (ap_0) and for the previous three days (ap_{1-3}), each with between two and six degrees of freedom (df_{2-6}). Several models were fit with various combinations of pollutants and lag structures. A backward stepwise regression procedure was employed and AIC was used to guide the determination of appropriate lag structures, the smooth-

ness of the confounder, and, ultimately, inclusion of air pollution variables into the predictive side of the model (Bozdogan, 1987). We then examined the influence of air pollution on humidex/mortality associations by estimating risk values for models with and without air pollution terms. Factor variables were also included to control for day of the week (dow_{0-7}) and summer holidays.

Previous research has indicated the effects from temperature, and thus temperature indexes like humidex, to be short term (Lind, 1964; Giles et al., 1990). We decided a priori to examine mortality associated with humidex values on the same day ($humidex_0$), 1 day previously ($humidex_1$), and the cumulative average of both days. Limiting the analysis to these observations may help to ameliorate potential bias resulting from selective reporting of lags related to the largest effect estimates.

The full model was fit using the *gam* function in S-Plus (Mathsoft Inc., 1997). It has been recently noted elsewhere that using the default function parameters, especially when fitting models with nonparametric terms, can underestimate the variance of fitted model parameters, leading to type I error (Ramsey et al., 2003). Related research has also shown that use of the default convergence criteria in S-Plus can result in biased fitted linear parameters (Dominici et al., 2002). However, these issues are not necessarily applicable to all studies using the GAM function for reasons related to the modeling approach adopted by the researcher (Katsouyanni et al., 2002). To guard against problems related to concavity and bias in the regression estimates, we decided to adopt more stringent convergence parameters (Dominici et al., 2002) and used parametric natural cubic splines instead of nonparametric smoothers. The approach essentially fits a generalized additive linear model (McCullagh and Nelder, 1989) with more strict convergence criteria.

Many heat/health studies evaluate mortality risk for heat-wave (or heat-stress) conditions compared to mean weather conditions (e.g., Kilbourne et al., 1982; Semenza et al., 1996; Smoyer, 1998a, b). In some cases, studies examine health impacts over a range of values and provide risk estimates by degree (e.g., Curriero et al., 2002). To provide results comparable to both approaches, we used the final model to calculate relative risk point estimates and confidence intervals for 1°C increments as well as for the 50–95th percentile (14.2°C) increase in humidex, the latter representing normal versus heat-stress conditions. The 50th percentile value of 23.1°C and 95th percentile value of 37.3°C fall into the “no discomfort” and “some discomfort” categories, respectively.

3. Results

Most days between 1 May and 30 September fell into the lowest (70.8%) humidex category, which is

considered “no discomfort” (Table 1). The second largest percentage fell into the 30–39°C humidex category (“some discomfort”), with most observations below 35°C. Less than 2% of all days fell into the 40–45°C humidex range (“great discomfort”). Five summers had no days falling into this category, while in 1988, 8 days reached a humidex in this range. Only 2 days during the entire study period had humidex levels in the “dangerous” category, above 45°C, one in 1990 and another in 1995 (Table 1). Mean maximum daily humidex values in Toronto ranged from 5.5°C to 50.3°C, with a mean of 23.3°C (Table 2). For comparison, mean maximum daily air temperature for Toronto ranges from 18°C in May to 27°C in July.

The mean daily number of nonaccidental deaths in Toronto was 40.4, for a total of 105,127 summer deaths over the 17-year period (Table 2). Approximately 75% of total deaths were among the elderly (mean = 30.2), and one quarter of these were due to combined cardiac and respiratory related conditions (mean = 10.6).

Concentrations for most pollutants, except O₃, decreased over the study period (Fig. 1) and are generally within Canadian National Air Quality Objectives (WQAQOG, 1996). Ozone levels show increased variability over time and tend to correlate well with summer temperatures. Exceedences for O₃ (at 50 ppb), based on maximum acceptable tolerances for O₃ to prevent adverse health effects, were observed on 229 days. However, recent research has estimated the lowest observed adverse-effect level (LOAEL) for O₃, defined as the lowest ambient level of ground-level O₃ that can be statistically associated with an adverse health outcome, to be 20 ppb for Canada, based on nonaccidental mortality (Health Canada, 1999). Approximately 72%

of O₃ observations during the study period were equal to or above this level (Table 2).

Fig. 2 shows a plot of mortality and humidex, with a LOESS smooth line (50% span) superimposed to reveal the basic relation. Average daily deaths are relatively stable for moderate summer humidex values from 7°C to 25°C, with a linear increase in deaths as humidex rises above 25°C. An optimal comfort range characterized by a mortality minimum is seen between 20°C and 25°C.

The stepwise modeling strategy removed within-summer and long-term patterns in the mortality series. The model residuals with the best AIC revealed no detectable pattern. The final model included the following variables: an intercept, a spline function of time, a factor variable for day of the week, and spline functions for O₃ and SO₂, each with two degrees of freedom (Eq. (2)). The model with the best fit included ozone lagged one day and same-day sulfur dioxide both with two degrees of freedom. The final model has the form:

$$\log(\mu_t) = \alpha + \beta \text{humidex}_t + \eta \text{dow}_{(1-7)} + S(\text{time}, 8/\text{year}) + S(\text{ozone}_{t-1}, 2) + S(\text{sulfurdioxide}_t, 2), \quad (2)$$

where *time* represents a variable for the day of the study (1–2601) and *dow*_(1–7) indicates the day of the week.

With air pollution terms in the models, relative risk (RR) point estimates narrowly exceeded 1.0 for all groups (Fig. 3). Humidex effects were most apparent for females (RR = 1.006, 95% CI = 1.004–1.008 per 1°C humidex and RR = 1.089, 95% CI = 1.058–1.121 for 50–95th percentile humidex (Table 3)). The over-65-years group was more affected by humidex than the younger age group, and the “other” category had slightly higher RR than the cardiac-respiratory category. However,

Table 2

Summary statistics of daily number of nonaccidental deaths and daily observations of environmental variables in Toronto, May 1 through September 30, 1980 to 1996

	Percentiles						SD
	Min.	25th	50th	75th	Max.	Mean	
<i>Mortality</i>							
Total	20	36	40	45	63	40.4	6.7
Male	8	18	21	24	39	20.8	4.7
Female	6	16	19	23	34	19.6	4.6
<65 Years	1	8	10	12	22	10.2	3.2
≥65 Years	11	26	30	34	52	30.2	6.1
Cardiorespiratory	1	8	11	13	23	10.6	3.3
Noncardiorespiratory	12	28	32	36	54	32.2	6.1
<i>Other variables</i>							
CO (ppm)	0.0	0.8	1.0	1.3	4.0	1.0	0.4
O ₃ (ppb)	0.0	18.7	26.0	36.0	90.0	28.7	14.2
NO ₂ (ppb)	3.0	17.0	22.0	29.0	68.0	23.8	9.2
SO ₂ (ppb)	0.0	1.0	3.0	6.0	36.0	4.3	4.3
Humidex	5.5	18.9	23.1	30.3	50.3	23.3	7.4

SD = standard deviation; CO = carbon monoxide; O₃ = ozone; NO₂ = nitrogen dioxide; SO₂ = sulfur dioxide.

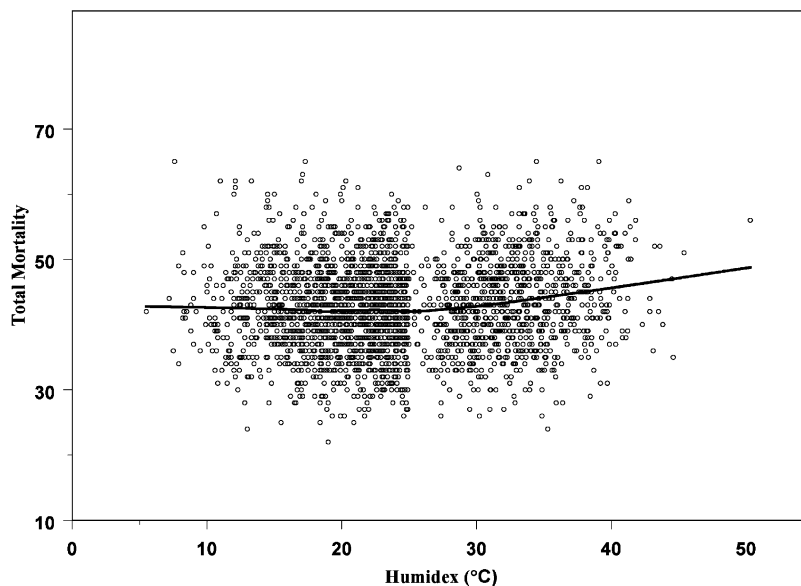


Fig. 2. Plot of summer mortality and humidex, with an LOESS smoothed (50% span) to help show the basic relation.

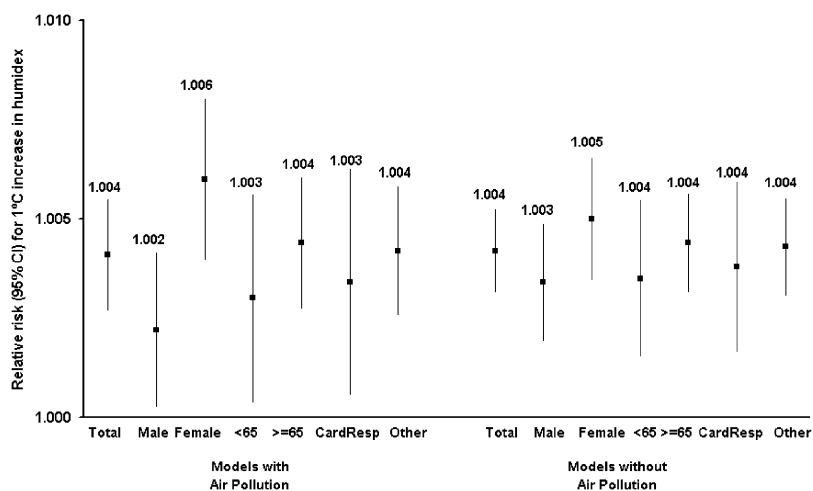


Fig. 3. Relative risk of mortality (and 95% CI) associated with a 1°C increase in humidex for models with and without adjustment for confounding by air pollution by demographic group and selected causes for Toronto, 1980–1996. Numerical values represent relative risk point estimates. (CardResp = cardiorespiratory deaths, Other = noncardiorespiratory deaths).

confidence intervals for the two age groups and causes of death overlapped.

Regression estimates for models without controlling for air pollution confounding were similar to those with models that included air pollution terms. When air pollution was omitted from the model, RR in the 50–95th percentile analysis increased less than 1.71% for all groups except females, for which RR decreased 1.42% (Table 3). The percent difference in point estimates per 1°C humidex, with and without air pollution in the model, was less than 0.12% for all groups, with females again demonstrating slightly decreased, rather than increased, risk when air pollution was not taken into account (values not shown). Confidence intervals

narrowed slightly for all groups investigated in models not controlling for air pollution (Fig. 3).

4. Discussion

The effect on human health of warm and humid summer conditions, including the potentially devastating impacts of lingering hot, humid air masses, has stimulated investigations into the size and shape of the association (Kunst et al., 1993; WHO/WMO/UNEP, 1996). In this study we employed GAMs to distinguish the relation between humidex, a summer heat-stress index, and nonaccidental mortality in Toronto,

Table 3

Association between humidex and mortality in summer for Toronto 1980–1996. Results are expressed as the relative risk (and the 95% CI) in mortality for an increase in the humidex between the 50th and 95th percentile range, and as the relative risk difference for models adjusted and unadjusted for air pollution

	Adjusted (heat stress) RR (95% CI)	Unadjusted (heat stress) RR (95% CI)	RR difference (%)
Total	1.059 (1.038–1.080)	1.061 (1.045–1.077)	0.15
Male	1.031 (1.003–1.060)	1.049 (1.027–1.071)	1.70
Female	1.089 (1.058–1.121)	1.074 (1.050–1.097)	–1.42
<65 years	1.044 (1.006–1.083)	1.050 (1.021–1.080)	0.64
≥65 years	1.064 (1.040–1.090)	1.065 (1.046–1.084)	0.02
CardResp	1.050 (1.008–1.093)	1.055 (1.023–1.088)	0.52
Other	1.061 (1.037–1.086)	1.062 (1.044–1.081)	0.10

CI=confidence interval; RR=relative risk; CardResp=Cardiorespiratory mortality; Other=Noncardiorespiratory mortality.

Canada's most populated city. Although common among studies estimating the association between air pollution and mortality, GAMs have been used less extensively among previous reports of temperature and mortality relationships. A majority of these studies do not statistically adjust for air pollution, an established human health risk.

Our results revealed a statistically significant, yet small association in Toronto between humidex and mortality for a variety of groups. The results are generally comparable and consistent with similar studies demonstrating a relation between summer weather and health in Toronto (Kalkstein and Smoyer, 1993a; Tavares, 1996; Smoyer et al., 2000; Smoyer-Tomic and Rainham, 2001). A clearly detectable portion of non-accidental mortality is attributable to humidex values lagged 0–1 day. AIC value comparisons revealed that the time response to humidex was acute, with associations weakening proportionally to an increase in lag time. Thus, our results also confirm other findings that health impacts from hot and humid conditions are immediate (Lind, 1964; Giles et al., 1990; Kalkstein and Smoyer, 1993b).

Although the association was stronger for elderly (65+ years), mortality and humidex than for the younger age group, the confidence intervals for the two groups overlapped. Confidence intervals for the elderly, however, were narrower, thus indicating a more robust humidex effect. Most US heat/health studies have identified the elderly as more susceptible to warmer temperatures than younger age groups (Ellis, 1972; Kalkstein and Davis, 1989; Whitman and Good, 1997). A previous study of weather and mortality for Canada found little difference in health impacts between the 65+ age group and younger populations (Kalkstein and Smoyer, 1993a). Thus differences in heat-stress risk for elderly versus younger

age groups should be re-visited, and results from Canadian populations compared to those from the United States and elsewhere. Due to increased life expectancy and better health among seniors over time, it may also be desirable in future studies to raise the cutoff age of the elderly group so as to isolate the frailest of the population.

Females demonstrated the highest relative risk of mortality from humidex in models with and without air pollution. Although there was some overlap in the tails of the 95% confidence interval for females and males, the overlap is so minimal that the point-estimate differences appear to be robust (Fig. 3, Table 3). The differences in female versus male risk estimates are most likely due to the greater number and proportion of women in the oldest age groups. Unlike the other groups we examined, however, models controlling for air pollution had higher, rather than lower, risk estimates. Without further study, it is unclear whether this finding is due to chance or represents a difference in mortality response to humidex and air pollution among females.

Plausible explanations for the importance of age as a risk factor are consistent with investigations of underlying biological mechanisms. Significant humidex/mortality associations with age are likely due to changes in physiologic tolerance to heat. A decline in thermoregulatory response and a reduction in sensitivity to thermal variation, proportional to an increase in age, have been shown to facilitate the onset of hyperthermia (Natsume et al., 1992). The stress that warm temperatures place on the thermoregulatory system is also closely linked with effects on the circulatory system. Experimental studies have found associations between arterial thromboses (Keatinge et al., 1986), increasing resting heart rate due to sympathetic activation associated with an increase in core body temperature (Yamazaki et al., 1997), and warm external temperatures. These conditions are more common among the elderly age group.

In our study, mortality risk from humidex for combined cardiac-respiratory outcomes was not statistically different from other causes of death. This, however, may reflect a limitation of the model or of combining the two mortality outcomes because the mechanism between heat stress and cardiac stress is well documented, as shown above.

We recognize that bias may arise from misclassification of the underlying cause of death, although our use of broad categories of gender, age, and ICD coding may reduce the likelihood of such bias. Although not performed here, an analysis of the daily correlation between misdiagnosis and humidex would reveal possible bias among effect estimates. We do not believe this to be the case in this investigation. We furthermore restricted calculation of risk estimates to acute humidex exposure to reduce bias associated with selection of largest effect estimates.

Of note is the magnitude of the humidex/mortality effect we identified for Toronto. The small but robust heat stress/mortality relation is consistent with the magnitude found in a previous study of Toronto using different methods (Smoyer-Tomic and Rainham, 2001). However, our estimates for Toronto are smaller than the range of regression coefficients reported for other locations, including Hong Kong (Yan, 2000) and cities in the United States (Curriero et al., 2002). Although not directly comparable because of different methods, units, and heat indicators used, the largest relative risk we identified in Toronto, 0.6% (95% CI=0.4–0.8%) per 1°C humidex for females, is considerably smaller than the 5.83% per 1°F in air temperature Curriero et al. (2002) identified for warm temperatures in Boston or the 2.45% per 1°F they identified for Chicago. Relative risk estimates for extreme (95th percentile) versus baseline (50th percentile) humidex, ranging from 3.1% (males) to 8.9% (females), were closer in magnitude to the Curriero et al. (2002) estimates. Summer weather conditions may partially explain the smaller heat/mortality effect in Toronto: mean July air temperature is 21°C in Toronto and 23°C in both Boston and Chicago, with both minimum and maximum temperatures in the summer months lower in Toronto than in the two US cities (The Weather Network, 2002). Thus Toronto populations are not exposed to the temperature extremes experienced in larger northern US cities susceptible to heat stress. The role of socioeconomic factors, urban planning, housing, and health and social policies may also be important in Canadian/US differences and require careful examination. Canadian results should also be compared to those from more northerly European cities.

Previous studies have alluded to air pollution/mortality associations being temperature dependent (Katsouyanni et al., 1993; Katsouyanni, 1995). There seems to be some epidemiological evidence relating to an interaction between air pollution and the tail ends of the summertime temperature distribution. One study of Toronto demonstrated relatively weaker air pollution and health associations in summer than during other seasons (Burnett et al., 1997). We noticed consistent, yet small differences in risk estimates between models with and without air pollution terms. This could be due to relatively low air pollution concentrations for Toronto, where average ozone concentrations were 28.7 ppb. For comparison, Birmingham, Alabama, and Philadelphia, Pennsylvania, had mean summer ozone concentrations of 65 and 85 ppb, respectively, between 1975 and 1988 (Smoyer et al., 2000). Toronto air quality records indicate that concentrations for most pollutants, except ozone, declined over the study period (Fig. 1). Thus, air pollution levels in Toronto during the study period were relatively low and may not have been large enough to exert a notable influence on mortality.

The results indicate the potential for interaction between air pollutants and temperature, and confirm hypotheses presented in previous research (Katsouyanni, 1995; Ballester et al., 1996; Morris and Naumova, 1997). To our knowledge, however, ours is the first study to examine the influence of air pollution in modifying the relationship between summer weather and human mortality.

We postulated that excluding air pollution terms from a weather/mortality analysis could lead to inflated relative-risk estimates. Our results confirm this hypothesis, but the effect is minimal. Percent relative-risk estimates were slightly larger for all mortality outcomes when air pollution was omitted from the regression analysis, but the confidence intervals overlap for risk estimates from the pollution-adjusted and nonadjusted regression models (Fig. 3, Table 3). The results also reveal that including air pollution terms increases the standard error of the coefficient estimates. The overlap of confidence intervals indicates that coefficient estimates are not significantly different; however, the results do show that failure to account for air pollution among studies with small point estimates may artificially add confidence to these estimates when, in fact, the true value may include unity.

The overall implication of not controlling for air pollution in heat/health risk estimates appears to be relatively minor, at least for Toronto. Nevertheless, we believe it would be unwise not to control for the potentially confounding influences of air pollution in studies of weather/health relationships, unless some quantitative assessment of air pollution confounding were included.

Further research of the association between warm weather and human health is required. Our analysis was limited to one Canadian city and we did not address how warm weather/mortality associations may change by ethnicity, preexisting illness (Kilbourne, 1999), or by factors associated with housing (Semenza et al., 1996), neighborhood (Smoyer, 1998b), or socioeconomic conditions (Kilbourne et al., 1982; Smoyer, 1998a). In a previous study of weather-related mortality in Toronto, we evaluated the difference between humidex and apparent temperature, the index widely used in the United States. Our research concluded that both are sufficient as weather indices for predictions of impacts to public health and that relatively few excess deaths were associated with high humidex and apparent temperatures in Toronto (Smoyer-Tomic and Rainham, 2001). It would be useful, however, if a universal thermal weather index could be adopted so that international studies could be directly compared.

We do not believe that this study currently reveals a large public health problem because, at present, exposures to high humidex levels are rare in Toronto. However, our findings here confirm our previous ones in

that the Toronto population is sensitive to elevated humidex levels when they do occur. Projections for temperate climates indicate a 2–5°C increase in average summer temperatures (Smith et al., 1998), which would result in more frequent heat waves (Kalkstein and Smoyer, 1993a). Under such conditions, summer mortality in Toronto could increase substantially. Recent research from the Netherlands, however, has shown that increases in summer mortality may be offset by reductions of mortality resulting from warmer winter temperatures (Martens and Huynen, 2001). The modeling approach adopted in this study would be useful for estimating weather/health associations under scenarios of future climate change, while taking into account air pollution.

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