



CHAPTER 7

PUBLIC HEALTH

The Metro East Coast Region (MEC) includes 20 million people in 31 counties in the tri-state area of New York, New Jersey, and Connecticut. The socio-economic, ethnic, racial, and genetic diversity of this area engenders an equally complex set of health issues unique to this part of the world. Among the distinguishing demographic features of the region's health profile are the number of inner city persons living in poverty, the number of immunocompromised persons, and the number of immigrants. The goal of this public health sector assessment is to evaluate the potential impacts of climate change on summer-season heat-stress mortality, water- and vector-borne diseases, and air pollution-related morbidity and mortality in the MEC Region. In addition, we present preliminary results of a modeling analysis of the future burden of respiratory morbidity in the region based on combining regional photochemical air pollutant projections and health impact coefficients from previous studies relating pollutant levels to respiratory hospitalizations.

BACKGROUND

With 20 million people, the cultural, ethnic, racial, and socio-economic diversity of the MEC Region makes it unique among world metropolitan areas (Tables 7-1 and 7-2). This is mirrored in a complex set of public health vulnerabilities. These include extensive areas of extreme poverty, particularly in the inner city; high population density; the constant influx of immigrants and transients; an unfiltered municipal water supply; and a sizeable population of immunocompromised persons (Hamburg, 1998).

Table 7-3 displays estimated mortality rates for counties comprising the MEC Region, for the region as a whole, and for the entire United States. Heart disease is the leading cause of death in both the MEC and United States,

with rates over 10% higher in the MEC Region. The mortality rate for HIV is over twice as high in the MEC as in the United States. Asthma mortality is similar in the MEC Region as a whole to the United States; however, large variations exist across counties within the MEC Region.

Trends over time (1981–1996) in cause-specific mortality rates are given in Table 7-4. Heart disease rates have been declining, with a much steeper decline from 1986 to 1991 in the MEC as compared with the total United States. From 1981 to 1991, cancer mortality rose in both the region and the country but seem to be leveling off in the most recent years for which data are available. Although mortality due to cerebrovascular disease has been declining for much of the past 20 years, there has been an increase in the trend from 1991 to 1996. Pneumonia and influenza mortality rates seem to be leveling off in both regions, with the MEC trend showing a little more instability than that for the total United States.

Chronic obstructive pulmonary disease and asthma mortality have been on the rise in both the MEC and the United States. By comparison to the national asthma mortality rate of 1.9 deaths per 100,000 persons, higher rates are seen in the MEC counties with the highest poverty rates (see Table 7-3), including the Bronx (6.3/100,000), Brooklyn (3.9/100,000), New York (Manhattan) (4.4/100,000), and Essex, NJ (2.7/100,000). For the rest of the MEC, asthma mortality rates are near or below the national average.

Asthma hospitalization rates have increased nationwide over the past two decades, particularly in the northeast (Mannino et al., 1998). In boys and girls under age 15,

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TABLE 7-1

Population of MEC Region by county and race, 1996

COUNTY	White	Black	Other	% Non-white
Connecticut				
Fairfield	716,988	88,845	26,380	13.8%
Litchfield	175,443	1,857	2,505	2.4%
N. Haven	688,123	88,004	16,510	13.2%
New Jersey				
Bergen	720,323	45,230	81,456	15.0%
Essex	401,636	323,507	30,582	46.9%
Hudson	413,371	85,828	50,836	24.8%
Hunterdon	113,667	2,524	2,507	4.2%
Mercer	246,504	67,814	15,201	25.2%
Middlesex	570,606	62,868	68,151	18.7%
Monmouth	510,081	55,687	24,479	13.6%
Morris	408,039	14,660	26,478	9.2%
Ocean	451,611	14,924	7,119	4.7%
Passaic	372,083	89,785	19,591	22.7%
Somerset	234,105	18,597	17,668	13.4%
Sussex	137,149	1,494	2,174	2.6%
Union	376,336	100,835	20,562	24.4%
Warren	94,414	1,667	1,350	3.1%
New York				
Bronx	627,554	508,703	52,610	47.2%
Dutchess	230,536	23,567	8,597	12.2%
Kings	1,174,762	928,081	150,714	47.9%
Nassau	1,119,948	126,252	57,298	14.1%
New York	967,055	407,461	153,899	36.7%
Orange	291,575	25,912	6,257	9.9%
Putnam	88,663	907	1,309	2.4%
Queens	1,199,615	459,234	315,359	39.2%
Richmond	336,943	35,913	25,296	15.4%
Rockland	231,131	30,817	16,359	17.0%
Suffolk	1,224,119	95,891	36,175	9.7%
Sullivan	62,904	6,387	1,048	10.6%
Ulster	154,440	8,987	3,229	7.3%
Westchester	712,042	134,512	46,465	20.3%
Total	14,963,103	3,855,843	1,286,855	25.6%

From Centers for Disease Control and Prevention, 1999.

asthma ranks second and third, respectively, for causes of hospitalization among Connecticut residents (CT DPH, 1998) and ranks first in New York City, where the childhood rates are three times the national average (NYC DOH, 1998). Hospitalization rates for asthma and total respiratory causes (including asthma, bronchitis, pneumonia, and emphysema) for selected MEC counties in 1996 are displayed in Table 7-5. These data demonstrate widely varying rates across counties similar to those observed for asthma mortality. In 1996, Bronx County, NY, had over four times the national rate and parts of Manhattan (e.g., in East Harlem) led the nation with seven times the national average. Within the city, asthma hospitalization rates for children in poor minority neighborhoods were over four times greater than for children in high-income neighborhoods. Per capita income and asthma hospital admissions (total numbers for 1997) for all ages are mapped in Figures 7-1 and 7-2.

TABLE 7-2

Estimated percentage of population living in poverty, by county, MEC Region, and U.S., 1996

CONNECTICUT		NEW JERSEY		NEW YORK	
County	% Poverty	County	% Poverty	County	% Poverty
Fairfield	6.9	Bergen	4.6	Bronx	31.4
Litchfield	5.0	Essex	16.7	Dutchess	7.9
N. Haven	10.0	Hudson	17.3	Kings	29.3
		Hunterdon	2.9	Nassau	5.1
		Mercer	8.2	New York	22.7
		Middlesex	5.8	Orange	10.5
		Monmouth	6.3	Putnam	4.0
		Morris	3.3	Queens	16.3
		Ocean	7.3	Richmond	9.2
		Passaic	12.3	Rockland	9.0
		Somerset	3.4	Suffolk	7.4
		Sussex	3.9	Sullivan	15.1
		Union	7.8	Ulster	11.3
		Warren	6.2	Westchester	9.1
REGION					
MEC	10.2				
US	13.8				

Source: Bureau of the Census, 1999.

Table 7-6 shows that, across the United States, hospitalizations due to malignant neoplasms have been declining over the past 20 years while pneumonia has been on the rise. Nationwide asthma rates rose in the mid 1980's, but appear to have declined by 1996. Contrary to the national trend, evidence suggests that New York City's asthma hospitalization rates, already among the highest in the nation, were still on the rise through 1996.

Trends in New York City case reports for three important vector-borne diseases are displayed in Table 7-7. Recent increases have been observed for both Lyme disease and malaria. Too few years are available for cryptosporidiosis to detect any trends.

CURRENT PUBLIC HEALTH STRESSORS

Chief among the current public health vulnerabilities in the MEC region is poverty, which is endemic to pockets of many counties (see Table 7-2 and Figure 7-1). Diabetes, HIV, and asthma are three of the many diseases that have been associated with poverty in the MEC (Wallace and Wallace, 1999). Obesity and a high-sugar, high-fat diet may explain much of the association of poverty with diabetes. Drug abuse and drug-related behavior, e.g., sex for money, probably accounts for much of the association with HIV. Over the MEC region, HIV mortality is right skewed (median = 14.3), varying by more than 25-fold from the lowest HIV

TABLE 7-3

Selected age-adjusted mortality rates, by MEC county, for the entire MEC Region, and for the U.S., 1996. (deaths/100,000 persons/year)

	Heart Disease	Malignant Neoplasms	Cerebrovsc. Disease	Pneumonia/ Influenza	COPD*	HIV	Asthma
Connecticut							
Fairfield	271.2	190.6	51.1	28.7	26.4	11.1	1.4
Litchfield	245.4	189.4	44.4	28.4	33.1		
N. Haven	260.4	205.0	49.1	28.8	28.7	14.6	1.7
New Jersey							
Bergen	257.0	204.9	49.0	23.0	24.3	7.2	1.2
Essex	278.3	223.8	58.2	32.6	29.9	76.6	2.7
Hudson	277.5	214.8	49.6	34.4	32.4	48.0	2.0
Hunterdon	241.3	186.0	50.0	40.3	33.1		
Mercer	287.2	221.3	44.7	41.1	35.1	17.5	1.9
Middlesex	293.5	226.5	51.7	30.0	30.8	13.4	1.2
Monmouth	280.9	209.0	49.0	26.6	31.5	15.7	1.3
Morris	273.5	203.8	50.0	28.2	30.3	4.0	1.2
Ocean	307.7	219.1	43.6	20.7	30.2	11.0	.7
Passaic	272.4	211.6	47.9	23.4	27.4	26.9	1.8
Somerset	229.1	184.5	52.9	27.8	27.2	6.5	1.5
Sussex	263.1	216.0	49.2	34.6	47.0		
Union	254.4	202.6	51.8	27.1	27.6	27.5	2.2
Warren	302.3	198.5	47.7	31.7	38.2		
New York							
Bronx	371.2	196.1	41.1	40.2	26.2	108.7	6.3
Dutchess	328.1	223.9	51.9	38.8	42.0	11.2	1.2
Kings	406.4	195.5	29.6	34.7	22.4	66.0	3.9
Nassau	331.7	199.1	35.7	34.5	25.5	7.7	1.4
New York	264.8	180.4	29.6	33.8	22.9	85.4	4.4
Orange	325.0	225.1	52.0	34.0	41.7	9.4	1.8
Putnam	276.0	226.6	40.1	36.1	31.9		
Queens	369.5	172.6	24.9	26.6	19.4	29.7	2.1
Richmond	450.3	227.0	23.3	49.9	31.6	25.4	1.8
Rockland	322.9	204.0	37.3	42.5	35.3	5.8	1.1
Suffolk	344.3	228.6	50.4	33.1	40.9	8.8	1.8
Sullivan	349.2	224.0	46.0	38.7	46.1	18.6	
Ulster	314.4	238.8	45.8	35.8	49.3	12.7	2.0
Westchester	273.4	199.7	43.8	31.9	28.7	14.0	1.5
MEC Region	300.7	208.0	44.9	32.8	32.2	26.3	2.0
USA	276.4	203.3	60.0	31.4	39.8	11.6	1.9

*Chronic Obstructive Pulmonary Disease

Source: CDC, 1999

area (Morris, NJ, 4.0; 3.3% poverty) to the highest (Bronx, NY, 108.7; 31.4% poverty) with a regional rate more than twice that of the national average.

The large number of immuno-compromised persons in the MEC, mainly persons with HIV, are susceptible to a wide variety of co-morbidities and opportunistic infections (CDC, 1998). The municipal water supply is one potentially significant source of pathogen exposure for these people. As noted earlier, Cryptosporidiosis is a self-limiting enteric disease in individuals with normal immune function caused by an aquatic protozoan that is resistant to chlorination. In persons with HIV and in the very young and old, the disease may be life threatening (Meinhardt et al., 1996). A 1993 outbreak of cryptosporidiosis in Milwaukee, WI, caused over 400,000 cases of acute diarrhea and several deaths (MacKenzie et al., 1995).

TABLE 7-4

Trends in five year intervals from 1981 to 1996 in age-adjusted mortality rates for selected causes: a) MEC Region and b) U.S. (deaths/100,000 persons/year)

MEC Region						
	Heart Disease	Malignant Neoplasms	Cerebrovascular Disease	Pneumonia/ Influenza	COPD	Asthma
1981	361.1	191.2	58.3	24.8	21.9	1.3
1986	349.3	207.2	50.0	31.3	28.9	1.5
1991	303.3	210.9	41.9	31.1	30.7	1.9
1996	300.7	208.0	44.9	32.8	32.2	2.0
United States						
	Heart Disease	Malignant Neoplasms	Cerebrovascular Disease	Pneumonia/ Influenza	COPD	Asthma
1981	328.3	184.0	68.2	23.4	25.4	1.4
1986	318.8	195.2	59.4	28.9	31.4	1.5
1991	285.6	203.8	54.1	30.9	35.5	2.0
1996	276.4	203.3	60	31.4	39.8	1.9

A similar mass exposure in the New York metropolitan area could result in substantial mortality among the HIV positive population.

Housing characteristics and indoor air quality also affect the health status of the MEC population. Indoor environmental factors that have been linked to adverse health outcomes include lead paint, asbestos fibers, environmental tobacco smoke (ETS), emissions from gas stoves and space heaters, various volatile organic compounds including formaldehyde and organochlorine pesticides, fungi, and a wide range of allergenic particles associated with pets, house dust mites, cockroaches, and rodents (Samet et al., 1987; Gold, 1992). Levels and impacts of these factors are likely to vary across the MEC as functions of housing type, socioeconomic status (SES), age, and other factors. Several indoor agents, including ETS and biogenic allergens, have been linked with either the causation or exacerbation of asthma (NAS, 2000).

While considerable progress has been achieved over the past 30 years in reducing levels of some outdoor air pollutants (e.g., sulfur dioxide and carbon monoxide) in the MEC, other pollutants, especially ozone and particulate matter, continue to reach unhealthful levels on a regular basis (U.S. EPA, Air Quality Trends Report, 1998). The human-health based National Ambient Air Quality Standards (NAAQS) for ozone and particulate matter are often exceeded in the MEC Region, placing several counties out of compliance. Human health effects that have been associated with these two pollutants include mortality and hospitalizations for cardiovascular and respiratory diseases, increases in respiratory symptoms such as cough and wheeze, diminished lung function, and others (Kinney, 1999). Effects are greatest among the elderly, the young, and persons with compromised health status such as asthmatics.

TABLE 7-5

Hospitalization rates for total respiratory conditions and asthma, selected MEC counties and the United States, 1996. (hospitalizations/100,000 persons/year)

	Total Respiratory	Asthma
Connecticut		
Fairfield	748.3	130.9
Litchfield	874.5	85.6
N. Haven	982.9	167.5
New York		
Bronx	1964.7	846.9
Dutchess	873.2	105.1
Kings	1544.5	511.8
Nassau	877.6	160.7
New York	1256.8	420.7
Orange	1133.6	198.3
Queens	1083.4	281.9
Richmond	1270.4	246.6
Rockland	644.3	98.8
Suffolk	769.1	144.7
Sullivan	1194.2	133.6
Ulster	985.9	104.4
Westchester	814.8	124.1
USA	1226.5	179.5

Source: NY and CT State Departments of Public Health.

Another outdoor pollutant of concern, but for which no outdoor air regulations exist, is diesel exhaust particles (DEP), emitted in large quantities by trucks and buses throughout much of the MEC region. Diesel particles consist of tiny carbonaceous nuclei upon which are adsorbed a wide variety of organic compounds, including the carcinogenic polycyclic aromatic hydrocarbons (Kinney et al., 2000). Because of their small size, DEP can be inhaled and deposit deeply in the human respiratory tract. Occupational epidemiology studies have linked DEP exposures with lung cancer. Environmental epidemiology studies have linked exposure to traffic-related pollution—e.g., based on residential proximity to major roadways—with increased respiratory symptom rates and diminished lung function (e.g., see review in Kinney et al., 2000). Within the MEC, DEP exposure is often viewed as an environmental justice issue with respect to the siting of bus depots and other diesel-related sources. For example, seven of eight bus depots and stations in Manhattan are located north of 100th Street in the underprivileged and largely minority communities of Harlem and Washington Heights.

An important demographic feature of the MEC population that influences regional public health is its status as an intra- and international travel and immigration destination. The MEC continues to be a major port of entry to the United States for visitors, refugees, and immigrants. The high population density of the MEC region, and the constant flux of large numbers of people through it, favors the spread of communicable diseases within and beyond the area. Population movement through the inner city has been important for the intra-regional increased incidence,

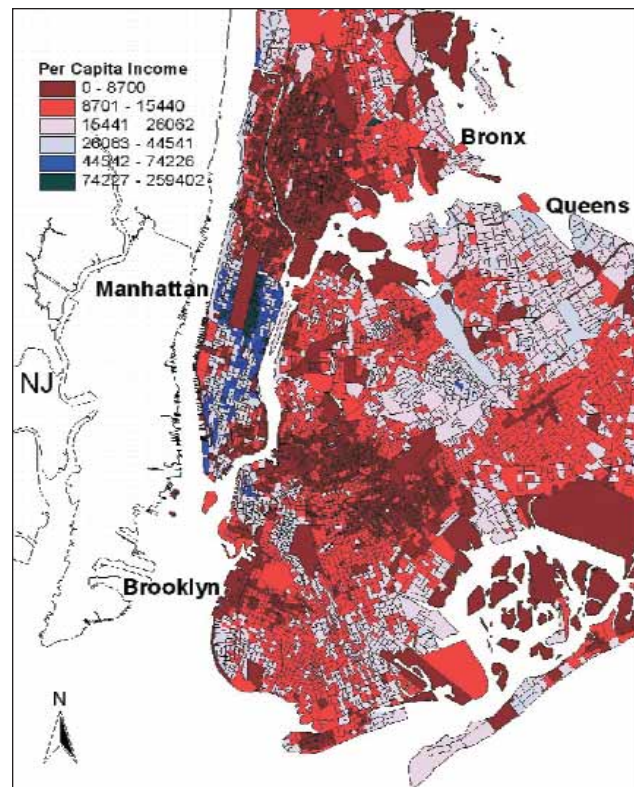


FIGURE 7-1 Geographic distribution of income per capita across New York City in 1990. Data aggregated at the census block group level.

Source: Bureau of the Census, 1999

and the extra-regional dispersion, of tuberculosis and AIDS in recent decades (Cantwell et al., 1994; Bifani et al., 1996). Also, the number of imported malaria cases among people returning or arriving from endemic countries has increased in New York City in recent years to over 100 cases annually (McNeeley et al., 1998).

CLIMATE-RELATED PUBLIC HEALTH STRESSORS

The most direct health effect likely to be associated with a warming and more variable climate is an increase in summer-season heat stress morbidity and mortality¹, particularly among the poor elderly. Though winter-season morbidity and mortality due to infectious diseases might decline if climate change results in shorter and less severe winters, this benefit would likely be offset by a rise in heat-wave associated illness and death as the number of days >90°F(32°C) increases. If hydrological regimes become more variable as predicted (IPCC, 1996), morbidity and mortality associated with extreme weather events, especially flooding, may also rise.

Indirectly, climate change in the MEC region could contribute to at least three classes of adverse health out-

¹Morbidity includes various measures of illness (e.g., doctor visits; hospitalizations) whereas mortality represents deaths.

TABLE 7-6

Trends from 1981-1996 in hospitalization rates for selected causes: U.S. and New York City (Hospitalizations/100,000 persons/year).

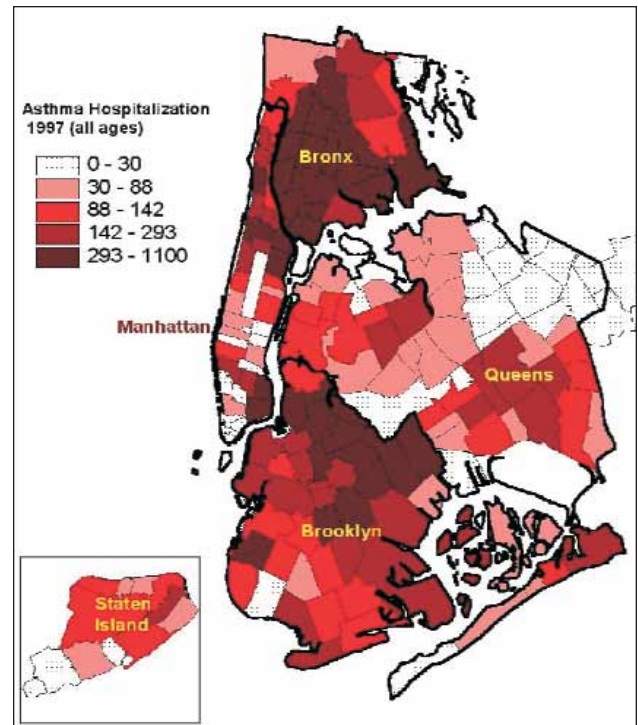
Year	Heart Disease	Malignant Neoplasms	Cerebrovascular Disease	Pneumonia	Asthma	NYC Asthma
1981	1466.7	856.1	354.0	337.8	183.6	unavailable
1986	1558.8	777.1	371.4	394.0	199.3	unavailable
1991	1478.3	636.2	333.2	434.2	195.6	434.4
1996	1605.7	520.5	361.7	455.3	179.5	480.8

Source: CDC, NHDS 1981, 1986, 1991, 1996

comes. The incidence of certain vector-borne diseases might rise as spring and fall warming extend the season in which disease reservoirs, vectors, and parasites are active and as wintertime survival increases. Secondly, water-borne disease organisms may become more prevalent depending on how rising temperatures affect wild animal populations in the watershed area, the viability of aquatic pathogens, and water availability. Finally, climate warming is likely to foster the formation of photochemical air pollutants such as O₃ and certain fine particles that have been associated with adverse human health effects including mortality (Davis et al., 1997). Complex feedback mechanisms, such as increasing pollen levels at higher CO₂ concentrations, and interactions between pollen and diesel particles, may exacerbate these impacts.

Climate change impacts on public health have undergone considerable research and debate for the past decade (IPCC, 1996; Epstein and Leaf, 1998). On the global scale, this has included examination of climate change impacts on risk of hunger (Rosenzweig and Hillel, 1998), vector-, water-, and food-borne disease (Martens, 1995; Patz et al., 1996; Lindsay and Martens, 1998), and direct effects of heat and cold on mortality (Martens, 1998). Much of the work on smaller geographic areas has focused on changes in the distribution of vector-borne disease for a particular country (Loevinsohn, 1994; Bryan et al., 1996) and the impact of heat-stress mortality on individual cities (Kalkstein and Smoyer, 1993; Katsouyanni et al., 1993).

Two reports have assessed climate change impacts in the MEC Region. A recent qualitative analysis by the Environmental Defense Fund (1999) discussed impacts of climate on heat-stress mortality, mosquito-borne disease,

**FIGURE 7-2** NYC asthma hospital admissions in 1997 by zip code.

and asthma. Based on a review of previous studies, it was concluded that all three outcomes had the potential to increase as a result of climate warming. Previously, Kleinman and Lipfert (1996) examined the potential effects of higher temperatures and air pollutant levels on mortality and respiratory hospital admissions in New York City. For a 2°C increase in annual mean temperature, the authors estimated that annual mortality would rise 0.67% and summertime respiratory hospital admissions by 1.3% in New York City. These annual estimates reflect the averaging of very large impacts on a few summer days with negligible impacts during the remainder of the year. Most of the projected increase in mortality was attributed to higher temperature, whereas ozone accounted for the bulk of the rise in hospitalizations.

Here we briefly review the literature on three potential health impacts that could result from climate change in the MEC Region: heat stress, vector-borne and water-borne diseases, and respiratory effects of photochemical air pollution.

TABLE 7-7

Trends in cryptosporidiosis, Lyme disease, and malaria, New York City.

Rates per 100,000 population

Year	77	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96
Cryptosporidiosis																		3.9	6.4	4.5
Lyme disease										0.3	1.5	2.2	1.5	1.3	1.9	1.4	2.9	1.3	6.2	5.7
Malaria	0.8	0.9	0.6	0.9	0.9	1.0	0.6	0.6	0.9	0.4	0.4	1.3	1.4	1.6	1.8	2.3	2.0	1.4	3.0	3.7

From NYC DOH, 1998.

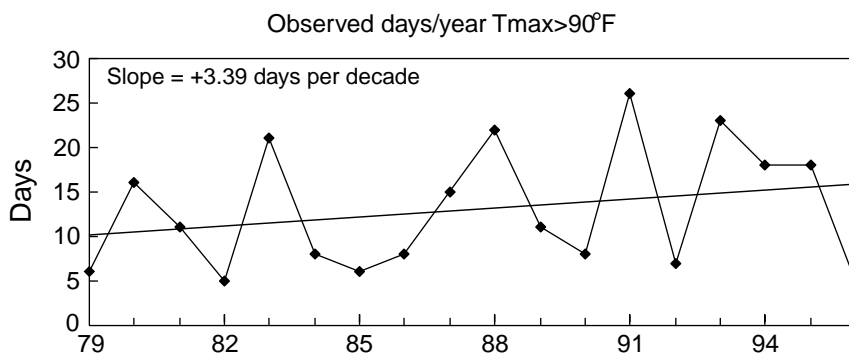


FIGURE 7-3 Average number of observed days per year with $T_{max} > 90^{\circ}\text{F}$, New York City (1979 to 1996).

Heat Stress

Increased morbidity and mortality due to heat stress is one potential direct impact of climate change in the MEC (Marmor 1975; Kalkstein, 1991; IPCC, 1996). Heat-induced illnesses and deaths in large cities have been noted at least since the early part of this century (Gover, 1938). In 1993, New York City led the nation in heat stress mortality with over 300 deaths (Kalkstein, 1993). It has been estimated that this toll could increase by two to seven times over the next century as the number of days with temperatures $>90^{\circ}\text{F}$ (32°C) increases through the 2090s (Kalkstein and Greene, 1997; EDF, 1999). Figure 7-3 shows the trend in observed days/year above 90°F from 1979 through 1996 in New York City, indicating a steady increase from an average of about 10 per year to over 15 per year (Rich Goldberg, personal communication). Note the substantial interannual fluctuations around the trend line. Figure 7-4 plots projected average numbers of days/year above 90°F for decades starting from 2000 to 2090 for the four alternative GCM scenarios, as well as for an extrapolation of the current trend. These models show increases from about 20 days per year in the decade starting 2000, to between 27 and 80 days/year in the final decade of this century.

Heat stress interacts with pre-existing disease states to precipitate acute morbidity and mortality. Kilbourne (1997) found that persons with cardiovascular disease, respiratory ailments, and a history of stroke have greater

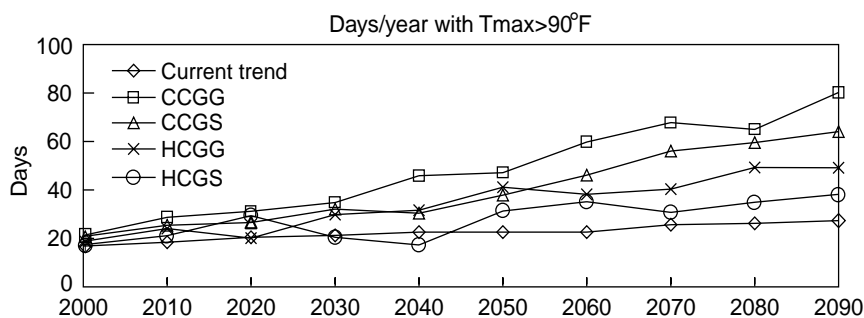


FIGURE 7-4 Change in the average number of days per year with $T_{max} > 90^{\circ}\text{F}$ for projected changes in climate, relative to 1961–1990. Scenarios depicted include extrapolation of current trend, and outputs from the four GCM scenarios: CCGG, CCGS, HCGG, and HCGS.

risk of mortality during heat waves. Because certification due to excess heat is rare, it has been difficult to get an accurate estimate of the total mortality impact of heat stress (Oechli, 1970). Many heat-related deaths are not a clear result of heatstroke or other heat-related illnesses, and thus are not reported as such in medical records. The use of different criteria and definitions for heat-related mortality in different studies has led to some confusion regarding which diseases contribute

most. Other difficulties in heat-related mortality studies are the incorporation of the time delay between exposure to heat and onset of fatal illness, collinearity among variables generated over common time periods, non-linear time trends, and inconsistent criteria for temperature and mortality determination.

Some of these difficulties have been resolved by the use of time-series analysis. This method is particularly appropriate in studying heat-related mortality data because it optimally evaluates short-term effects of time-varying exposure. Using total daily mortality as the outcome variable enables an assessment of the total impact of heat-stress across causes of death. Time-series analysis also allows researchers to evaluate multiple weather and other environmental variables, and can elucidate the lag time between exposure and manifestation of effect, which has typically been observed to be 1–2 days (Pope, 1996).

The relationship between ambient temperature and risk of death is a complex one. Populations appear to adapt to prevailing meteorological conditions, both through physical and physiological mechanism. It is when temperatures exceed normal limits, especially early in the summer, that risk of heat-related mortality is greatest. In a 1938 study of U.S. mortality in the summer months, Gover noted that positive deviations from normal temperatures during summer predicted mortality in 86 large cities. This was confirmed by Rogot and Padgett in a 1976 study of temperature and stroke mortality in the United States, as well as by MacFarlane in a 1978 study of daily mortality during three summer hot spells in London. Another common observation is a super-linear relationship between mortality risk and temperature as temperature rises above normal levels (e.g., Sartor et al., 1997).

The observation by Kalkstein (1991) that minimum temperature was a key predictor of mortality has led to the hypothesis that high over-night tem-

peratures play a key role in heat-related mortality risk. Specifically, excessively hot nights exacerbate the stress of extremely hot days, since the cooler nighttime temperatures usually serve to mitigate the adverse effects of hot daytime conditions. It is also thought that high winds decrease the adverse effects of hot weather by increasing body cooling (Kunst, 1993).

The elderly appear to be the most vulnerable population subgroup. MacFarlane (1978) found that the increase in mortality during hot spells in London were largely restricted to individuals older than 60 years. More recently, Whitman reported that during the Chicago heat waves of 1994 and 1995, individuals over the age of 65 accounted for 63% and 72% of all deaths, respectively. With the general aging of the MEC population, the proportion of persons in the older age groups will increase in coming years.

Urban living increases the risk of heat-related mortality. Higher temperature in cities as compared with surrounding suburban areas, combined with the higher proportion of socioeconomically disadvantaged, are factors that contribute to excess deaths. The temperature disparity between cities and surrounding suburbs is attributed to the fact that a large proportion of urban surface area is covered by man-made materials that absorb daytime radiant heat and radiate this heat during the night. This “urban heat island” leads to higher minimum temperature in the cities which, as discussed earlier, appears to exacerbate heat stress. The high concentration of disadvantaged populations living in urban core neighborhoods interacts with this phenomenon to enhance vulnerability.

The distribution of heat-stress impacts across the MEC will likely vary both as a function of local surface temperatures as well as by the residential distribution of the disadvantaged elderly. In addition to the urban heat island effect, strong gradients in local surface temperatures also occur within urban areas due to variations in vegetation cover as well as reflectivity of man-made surfaces, as shown in Figure 7-5 (Small, 2000). In addition, the residential distribution of disadvantaged persons is very heterogeneous in the MEC (Figure 7-1).

Factors associated with poverty include deteriorating and poorly maintained housing, and inadequate interior climate control due to the expense of owning and operating air conditioners (Semenza et al., 1996). Reduced mobility of the elderly poor, due in part to fear of crime, leads to greater time spent indoors. Anecdotal evidence suggests that vegetation may increase with affluence in some urban areas, enhancing protective shading from heat effects. Thus, locally elevated surface temperatures may interact with poverty over small geographic scales to create even greater heat-stress impacts in the disadvantaged neighborhoods of the MEC. Future work by our group will analyze the small-scale geographic distribution of heat-stress

impacts in the MEC using remote sensing in conjunction with available demographic and health data.

The extent to which heat stress will impact public health in the MEC as a whole over the next century is not known, but will depend on individual adaptability, the prevalence of publicly and privately accessible air-conditioned environments, and the ability of weather services and local health agencies to warn the population of upcoming heat waves (Kalkstein, 1991). The distribution of impacts by age and socioeconomic status are likely to be marked. The effects of summer heat stress may be partially offset by reductions in wintertime mortality due to cardiovascular disease and respiratory infections as warming reduces the duration and severity of winters (Langford and Bentham, 1995; Martens, 1995).

While the direct effects of climate change on human health in the MEC due to increased heat stress have the potential to be significant, adaptive responses involving increasing access to air conditioning and improved early

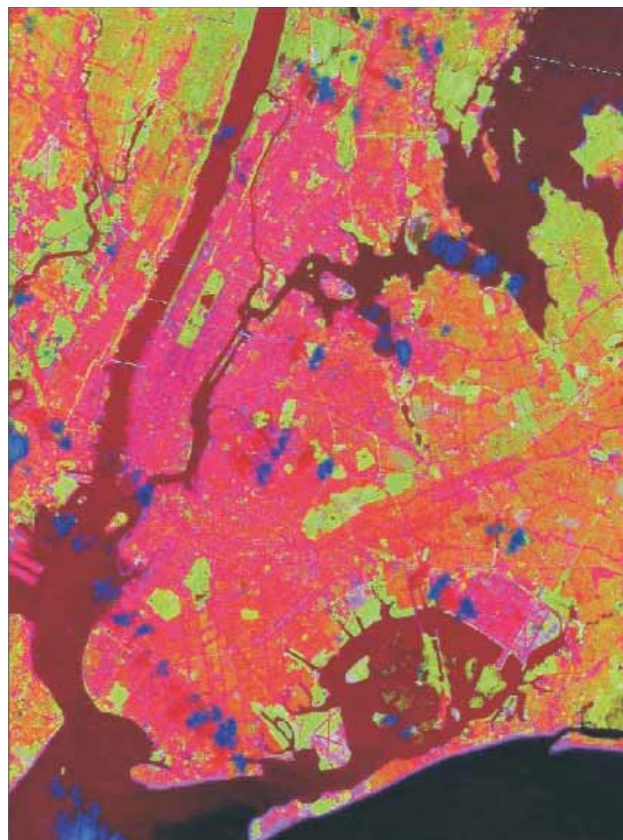


FIGURE 7-5 New York City surface temperature and vegetation fraction. The image is a reduced resolution false color composite combining surface temperature and vegetation abundance information derived from Landsat TM data acquired 2 June, 1996. Red indicates surface temperature, green indicates vegetation abundance and blue indicates uncertainty in the vegetation fraction estimate. Red and pink areas are characterized by higher surface temperatures and lower vegetation abundances. Green and yellow areas are characterized by higher vegetation abundances and lower surface temperatures. Blue and black areas have lower surface temperatures and little or no vegetation. Source: Christopher Small, Lamont-Doherty Earth Observatory

warning mechanisms are relatively straightforward, given sufficient political will and economic resources. The impacts of increased peak energy demand during summer heat waves will need to be addressed via new supplies however.

The more complex set of indirect effects of climate change, including vector- and water-borne diseases and air-quality impact on respiratory diseases, may have an even greater cumulative impact on health in the MEC region over the coming century. In addition, due to their complexity, the indirect impacts will be more difficult to forecast, prevent, and effectively adapt to.

Vector-Borne Diseases

The distribution, dispersion, and transmission potential of many important disease vectors and reservoirs are largely determined by climatic factors. Warming of sub-tropical and temperate regions supports the poleward movement of arthropods, arachnids, and mammals from the lower latitudes and increases the wintertime survival rate of endemic vectors (Rodriguez-Tan and Weir, 1998; Kovats et al., 1998; Gratz, 1999). Here we briefly discuss three vector-borne diseases: malaria, West Nile Encephalitis, and Lyme disease.

An increased incidence of locally acquired malaria may be one adverse outcome of climate warming in the MEC. Competent vectors of malaria exist in the MEC (Zucker, 1996) but do not presently support sustained transmission of the disease for two main reasons. First, most aspects of *Anopheles* population dynamics and behavior are limited by an intolerance of temperatures below 9°C (48.2°F); this threshold is even higher for the *Plasmodium* parasite, which cannot reproduce below 14.5°C (58.1°F) (Lindsay and Birley, 1996). Second, on average, the U.S. population spends less than two hours a day outside (Godish, 1997), minimizing the opportunity for exposure. Both of these protective factors may be undermined as the climate of the MEC warms.

Prior to 1991, local malaria transmission had not occurred in the region since 1966 (Zucker, 1996). Both 1991 cases, which occurred in southern New Jersey at the MEC-Mid Atlantic Region (MAR) border, were *attributed to unusually warm, humid weather*; the first case was also linked to the proximity of documented and undocumented immigrant workers. In 1993, three more cases of locally acquired malaria occurred in Queens, NY (Layton et al., 1995). It was concluded that a recent immigrant living in northern Queens, possibly of Latin American or Caribbean origin, had served as the source of mosquito inoculation; and that the mosquito population had been favorably affected by *warmer, more humid temperatures*.

Several climate models suggest that the annual mean temperature will increase largely as a result of higher winter and spring temperatures. These trends may expand the mosquito season into the spring and improve the survival

of the parasite, increasing the possibility of exposure. While public health, environmental, and residential infrastructure in the MEC probably preclude the possibility of a large-scale outbreak, the frequency of isolated cases may rise as climatic conditions and the increasing incidence of imported cases (associated with immigration to the MEC region) continue to favor local transmission.

The West Nile Encephalitis epidemic of 1999, also mosquito-borne, provides an alarming illustration. Seven confirmed fatalities occurred during a very wet August that followed an unusually mild winter and dry July. It appears that this pattern of temperature and precipitation selectively favored the prevalence of two mosquito species, *Culis pipiens* and *Aedes vexans*, that can carry and spread the West Nile virus. This epidemic highlights the magnitude of both public health and societal impacts that changing patterns of vector-borne diseases may have as the climate changes and becomes more variable over the next century.

The incidence of Lyme Disease may also change with the climate (McMichael and Haines, 1997). In a study of black-legged tick (*Ixodes*) populations in New Jersey, Vail and Smith (1998) found that temperature and humidity accounted for most of the variation in behavior. Two recent papers have noted a northward movement of ticks in Europe coincident with warmer winters (Lindgren et al., 2000; Tälleklint et al., 1998).

Water-Borne Disease: Cryptosporidiosis

In 1993, an outbreak of cryptosporidiosis in Milwaukee, WI, caused over 400,000 cases of acute diarrhea and several deaths (MacKenzie et al., 1995). Though it is uncertain how the municipal water supply became contaminated with *C. parvum*, it has been suggested that heavy spring rains carried large quantities of wild and domestic animal manure into the watershed area (Nadakavukaren, 1995; Scott et al., 1994). As the ranges of the mammal hosts of the parasite (e.g., deer and mouse) overlap with the watersheds of the MEC, and as the chief means of municipal water purification (chlorination) is relatively ineffective against *C. parvum*, the elements for significant outbreaks currently exist in the MEC region. With a relatively high number of immuno-compromised persons in the MEC region, a mass exposure similar to the Milwaukee episode could have serious consequences in the MEC region.

As the climate in the MEC warms, the conditions for *C. parvum* outbreaks will become more favorable if some combination of the following three conditions are met. First, if warming substantially increases evapo-transpiration rates, concentrations of *C. parvum* in municipal water supplies would rise as the watermark falls. Second, warming of the aquatic environment may improve parasite viability (Colwell, 1996). Finally, if warmer winters facilitate the

survival of deer and other significant wild sources of contamination, human infection rates may similarly rise. Again, precipitation patterns may ultimately determine if and how the epidemiology of this disease is impacted. If, as some GCMs project, the frequency of severe storm events increases over the next century, we would expect the probability of cryptosporidiosis outbreaks to similarly rise.

Air Quality and Respiratory Diseases

In 1971, the U.S. EPA established National Ambient Air Quality Standards (NAAQS) for “criteria pollutants,” a small set of ubiquitous outdoor air pollutants with well-established human health effects. The criteria pollutants include ozone (O₃), PM_{2.5}, PM₁₀, lead, carbon monoxide (CO), nitrogen dioxide (NO₂), and sulfur dioxide (SO₂). The current NAAQS are summarized in Table 7-8.

Over the past three decades, considerable progress has been made in controlling levels of several of the criteria pollutants (e.g., sulfur dioxide, carbon monoxide, and lead). However, concern remains about the human health impacts of O₃ and particulate matter (i.e., PM_{2.5} and PM₁₀), for which concentrations remain elevated in many parts of the country, including the New York metropolitan area. The atmospheric reactions that produce O₃ and PM_{2.5} from combustion precursors are strongly dependent on temperature. Thus, climate change is likely to foster increasing concentrations of these pollutants if precursor emissions are held constant, and will make it more difficult to reduce concentrations even with reductions in precursor emissions.

O₃ is a strong oxidant gas that occurs naturally in the stratosphere (i.e., 30–50 km altitude) but which is an unwelcome pollutant in the troposphere (the lowest 10 km of the atmosphere). Tropospheric O₃ is a secondary pollutant (i.e., not directly emitted to a substantial degree) that is produced via complex chemical reactions involving nitrogen oxides, reactive hydrocarbons, and sunlight (U.S. EPA 1996a). In populated areas, the primary sources of O₃ precursor pollutant emissions are motor vehicles and the fuel supply system that supports them. Vegetation can be another important source of reactive hydrocarbons. Because of the importance of solar radiation and temperature in O₃ photochemistry, significant concentrations of O₃ appear only in the warmer months, i.e., May through October. Further, O₃ production occurs only during daylight hours, resulting in a characteristic mid-day concentration peak in urban areas. However, O₃ concentrations often remain elevated late into the evening, especially in regions downwind of major urban areas. As a result, residents of downwind regions, such as the Connecticut coastline and Long Island, typically experience longer periods of elevated O₃ levels than do urban dwellers, such as those living in New York City. Because of its reactive

TABLE 7-8

Current primary National Ambient Air Quality Standards for criteria pollutants

Pollutant	Standard	Averaging Time	Year Last Revised
Ozone	80 ppb	3-year average of the annual 4th-highest 8-hour concentration	1997
PM _{2.5}	15 µg/m ³	annual	1997
	65 µg/m ³	24-hour	
PM ₁₀	50 µg/m ³	annual	1987
	150 µg/m ³	24-hour	
Lead	1.5 µg/m ³	quarterly	1978
Carbon Monoxide	9 ppm	8-hour	1994
	35 ppm	1-hour	
Nitrogen Dioxide	53 ppb	annual	1995
Sulfur Dioxide	30 ppb	annual	1996
	140 ppb	24-hour	

nature, O₃ only partially penetrates indoors, with indoor/outdoor ratios ranging from 0.1 to 0.8, depending on the degree of natural ventilation (penetration is greatest when open windows are used for ventilation).

PM_{2.5} represents the mass concentration of airborne particles with aerodynamic diameters smaller than 2.5 micrometers (µm). PM_{2.5} particles vary widely in size, composition, and origin (U.S. EPA, 1996b). Some are emitted directly by fossil fuel combustion, such as fly ash and soot from coal and diesel fuel combustion. Others form as secondary pollutants by chemical reactions in the atmosphere that convert gases emitted by fossil fuel combustion, such as sulfur dioxide, to particles, such as acid sulfates. Important chemical components of PM_{2.5} include sulfates, nitrates, elemental carbon, organic molecules, and a variety of trace elements (Spengler and Wilson, 1996). Outdoor PM_{2.5} particles penetrate readily to the indoor environment (Ozkaynak and Spengler, 1996). Unlike O₃, significant indoor sources of PM_{2.5} exist, the most prominent being smoking and cooking.

HUMAN HEALTH EFFECTS OF OZONE

O₃ is a strong oxidant gas which upon inhalation deposits throughout the respiratory system. Epithelial cells in the deep lung are especially vulnerable to oxidant damage, both because the delivered dose of O₃ is greatest there and because these cells lack a protective mucous layer. Acute O₃-induced lung injury is characterized by epithelial cell destruction, pulmonary edema, and inflammation (U.S. EPA, 1996a).

Human chamber studies have shown that brief O₃ exposures at or above 80 ppb cause reversible drops in lung volumes, increases in non-specific bronchial responsiveness, and pulmonary inflammation (U.S. EPA, 1996a;

Horstman et al., 1990; Devlin et al., 1991). There is a broad distribution of responsiveness across human subjects for all of these effects, with some individuals exhibiting responses several fold higher than the population mean response, and others showing no response. Epidemiology studies involving repeated measures across days have also demonstrated the acute effects of low-level O₃ exposures on lung function in children and adults (Hoek et al., 1993; Kinney et al., 1989; 1996a; Spektor et al., 1991).

Epidemiology studies also have reported acute associations between O₃ and daily asthma exacerbations, emergency room visits, hospital admissions, and deaths (Burnett et al., 1994; Kinney and Ozkaynak, 1991; Thurston et al., 1992). These studies suggest that asthmatics may be especially vulnerable to O₃-induced pulmonary effects. The known effects of O₃ on acute pulmonary inflammation suggest a plausible role in exacerbation of asthma.

Summer-season hospital admissions for asthma and for total respiratory causes have been significantly associated with ambient ozone levels (Thurston et al., 1992; Burnett et al., 1994). In a multiple regression analysis of daily data from three New York state metropolitan areas, including New York City, Thurston et al. (1992) reported that mean ozone levels accounted for 12–24% of asthma admissions and 5–18% of total respiratory admissions in the New York state metropolitan areas studied. Analyzing similar data from southern Ontario, Burnett and colleagues (1994) reported that mean O₃ levels were associated with 5% of asthma, COPD, and infectious disease hospital admissions for persons of all ages, and with 15% of the admissions among children. Thurston et al. (1994) reported that 21% of total respiratory admissions in Toronto, Ontario were associated with mean O₃ levels and that the relationship persisted after high O₃ days (>120 ppb) were excluded from the analysis. Table 7-9 summarizes the risk coefficients (admissions per 100 ppb ozone per day per 1,000,000 persons) for respiratory and asthma hospitalizations from these three key studies, as summarized in U.S. EPA 1996a.

HUMAN HEALTH EFFECTS OF PARTICULATE MATTER

A large number of recent time series observational studies have reported small, statistically significant associations between particulate matter (i.e., TSP, PM₁₀) and daily mortality, suggesting that the mortality effects seen in episodes earlier this century persist at lower contemporary levels of particle exposure, at least among the most vulnerable members of society, such as the elderly and those with pre-existing cardiopulmonary disease (Katsouyani et al., 1997; Kinney et al., 1995; Pope et al., 1992; Schwartz, 1993). Cause-specific analyses usually have observed larger relative effects for deaths attributed to respiratory, and to a lesser extent cardiovascular, causes than for other causes of death. Quantitative results from studies of this kind have been

remarkably consistent, suggesting a 5–10% increase in total daily deaths associated with increases of 100 µg/m³ in daily average PM₁₀ concentration (U.S. EPA, 1996b).

A more limited body of epidemiologic evidence is available showing acute morbidity effects of daily PM exposures (Dockery and Pope, 1994). Observational time series studies similar to those addressing acute mortality have reported acutely increased hospitalizations or emergency room visits for respiratory complaints in association with PM_{2.5} and/or sulfate particles (Burnett et al., 1994; Thurston et al., 1992; Schwartz, 1994). Repeated measures studies in small cohorts of subjects have reported small but statistically significant declines in lung function and increases in lower respiratory symptoms associated with ambient PM₁₀ and sulfate concentrations (Hoek and Brunekreef, 1993; Pope and Kanner, 1993). As a group, findings from these studies of pulmonary effects reinforce the plausibility of the acute mortality results noted earlier, and suggest a possible role of acute pulmonary irritation in the mechanistic pathway leading to mortality in susceptible individuals.

Epidemiology studies correlating mortality rates and PM concentrations across metropolitan areas represent the oldest and most extensive evidence for chronic PM effects (Lave and Seskin, 1970; Evans et al., 1984). However, interpretation of early cross-sectional observational studies was seriously hindered by uncertainties regarding potential confounding by cigarette smoking, occupational exposures, and other factors (Evans et al., 1984). Confirmatory results have emerged recently from two large prospective cohort studies which, based on individual questionnaire data on smoking and other risk factors, were able to control for

TABLE 7-9

Effect estimates of daily admissions for asthma and total respiratory causes as a function of ambient ozone concentration. (Adm/100ppb/day/1,000,000 persons)

Reference	Location	Admission Cause	Effect Estimate
Thurston et al., 1992	New York City, New York	Total respiratory	1.4 (+/- 0.5)
Thurston et al., 1992	Buffalo, New York	Total respiratory	3.1 (+/- 1.6)
Burnett et al., 1994	Ontario, Canada	Total respiratory	1.4 (+/- 0.3)
Thurston et al., 1994	Toronto, Canada	Total respiratory	2.1 (+/- 0.8)
Thurston et al., 1992	New York City, New York	Asthma	1.2 (+/- 0.5)
Thurston et al., 1992	Buffalo, New York	Asthma	1.2 (+/- 0.5)
Thurston et al., 1994	Toronto, Canada	Asthma	1.4 (+/- 0.8)
Thurston et al., 1994	Buffalo, New York	Asthma	1.2 (+/- 0.4)

From EPA, 1996a.

major potential confounders at the individual level in the analyses (Dockery et al., 1993; Pope et al., 1994). These two recent studies are also important because they analyzed multiple, alternative PM measures, including PM_{2.5}. In a cohort of 8,111 white adults, Dockery and colleagues reported a linear exposure response of mortality risk vs. average PM_{2.5} concentrations across six U.S. cities, controlling for smoking and other risk factors (Dockery et al., 1993). The risk of death was increased by 26% for an exposure difference of 18.6 µg/m³ across cities. This mortality risk was similar to that associated with 25 pack-years of cigarette smoking. Pope and colleagues reported similar findings from a prospective follow-up of 552,138 adults from 151 metropolitan areas (Pope et al., 1994). For a subset of 50 locations where PM_{2.5} data were available, the risk of death was increased by 17% for an exposure difference of 24.5 µg/m³ across metropolitan areas.

CLIMATE CHANGE AND AIR QUALITY

As climate changes and becomes more variable, episodes of elevated O₃ and PM_{2.5} are likely to become more frequent and more severe (NRC, 1991; IPCC, 1996). There is a strong positive relationship between O₃ concentration and temperature. Relating daily maximum O₃ and temperature for three sites in Connecticut, Wackter and Bayly (1988) found that daily maximum ozone increased linearly with daily maximum temperature above 70°–80°F, and that mean maximum ozone levels may exceed the EPA standard of 120 ppb (1 hr daily avg) for temperatures over 90°F (32°C). Similar results were found for New York City (U.S. EPA, 1993). With GCMs predicting significant increases in the number of days per year >90°F (see above), the frequency of high ozone days is also likely to increase. Secondary PM_{2.5} formation also is likely to be enhanced under these conditions. Other factors that are likely to influence pollution levels in the coming century include urbanization and its influence on temperature (i.e., the urban heat island effect), changes in precursor emissions in the MEC and regions to the south and west, changes in horizontal global circulation (e.g., greater stagnation of summer time air masses), and changes in tropospheric injection rates of stratospheric ozone (NRC, 1991; Wang et al., 1998).

A comprehensive assessment of air pollution-related human health risks that might result from climate change would include all of the health effects of O₃ and PM noted in the above sections. A logical strategy would be to take meteorological outputs from alternative climate change scenarios, drive atmospheric chemistry models with these meteorological inputs along with assumptions regarding changes in precursor emissions, and thereby estimate future ground-level O₃ and PM_{2.5} concentrations at future time points. These pollution estimates would then be combined

with exposure/response functions for each health effect, derived from the available literature, to estimate the human health impacts of changing air quality. Estimates of future population numbers would also be needed as inputs to this calculation. Indices of vulnerability, based on age or socioeconomic status, might also be included.

As part of the initial year of the MEC climate change impact analysis, we began preliminary work on an air quality risk assessment of the kind outlined above. Our initial work examined O₃ and its effects on asthma and other respiratory hospital admissions. In the section that follows, we describe the methods and results of our preliminary work for this case study. Note that this is not intended to represent a comprehensive analysis of the human health impacts of climate and air quality. A full analysis would include both O₃ and PM_{2.5}, and would examine all significant health effects of these two pollutants, especially effects on mortality.

IMPACTS OF CLIMATE CHANGE ON GROUND-LEVEL OZONE AND RESPIRATORY HOSPITALIZATIONS

Research Questions

The aim of this research study is to analyze the potential impacts of climate change on respiratory hospitalizations due to ground-level O₃ in the MEC region in the 21st century. The rationale for this approach includes: 1) the documented impacts of current-day O₃ concentrations on respiratory hospitalizations, 2) the possibility that ground-level O₃ concentrations may increase over time in the MEC region as climate warms and precursor emissions increase, and 3) the potentially high degree of vulnerability of the MEC population, due to the current high rates of asthma in portions of the MEC region. To accomplish this aim, we combine regional photochemical air pollutant projections under scenarios of climate change with risk coefficients from previous O₃ epidemiology studies to estimate the change in O₃-attributed hospitalizations in the MEC region from 1996 to 2030 and 2100.

Data and Methods

Projections of future climate and O₃ precursor emissions are used as inputs to a global photochemistry model run at GISS. Because it is computationally very expensive to model the full range of chemical reactions in the troposphere in a GCM suitable for climate studies, we have chosen to use a subset of molecules, and include only the photochemical reactions that involve those chosen species. It is now accepted that the abundance of O₃ is most often limited by the availability of NO_x, rather than the hydrocarbons, which are also O₃ precursors (e.g. Tranier et al, 1993; National Academy of Sciences panel on Air Quality Standards,

1996). Therefore, we have not included an explicit representation of non-methane hydrocarbons (NMHCs) in our reduced chemistry scheme. We do, however, include equivalent carbon monoxide emissions from isoprene, which is thought to be the most important contribution of NMHCs (Wang et al., 1998c; Horowitz et al., 1998).

The simplified chemistry scheme is based on CH_4 , CO , NO_x ($\text{NO} + \text{NO}_2 + \text{NO}_3 + \text{HONO}$), HO_x (OH, HO_2), and O_x ($\text{O} + \text{O}(1\text{D}) + \text{O}_3$) chemistry. A contribution to carbon monoxide from isoprene has also been included, but otherwise hydrocarbons other than methane have been neglected. Methane is set to fixed values in the troposphere. Ten chemical species are transported in the model: O_x , NO_x , H_2O_2 , H_2O , CO , HNO_3 , N_2O_5 , HO_2NO_2 , CH_3OOH , and HCHO . After combining the short-lived radicals into equilibrated families, we find that all the species have long enough lifetimes (except for HO_x , whose very short lifetime keeps it in equilibrium at all times) that we can use an extremely simple explicit scheme to calculate chemical changes, using a chemical time step of one hour.

The chemical scheme includes 52 reactions. Heterogeneous hydrolysis of N_2O_5 into HNO_3 takes place on sulfate aerosols, using the reaction rate coefficients given in Dentener and Crutzen (1993). Sulfate surface areas are taken from an online calculation performed with the same GISS GCM (Koch et al., 1999), assuming a monodispersed size distribution. Photolysis rates are calculated with Fast-J, a scheme which uses only seven wavelength intervals, yet deviates only slightly from a full line-by-line calculation (M. Prather, personal communication, 1999) and compares well with other photolysis schemes (Olson et al., 1997). The model includes 14 photolysis reactions. Photolysis calculations are performed every two hours, giving us a fairly realistic diurnal simulation.

Emission inventories of NO_x and CO have been compiled for the $4^\circ \times 5^\circ$ grid used in this version (see below) of the GISS model. The use of detailed NO_x and CO emissions is critical since emissions vary widely with location. Nitrogen oxide sources are quite similar to those specified for the NASA Subsonic Assessment (SASS) aircraft project, consisting of annual emissions from fossil fuel burning, and monthly emissions from biomass burning, soils, and aircraft. CO emissions from energy use and from biomass burning are included. An additional CO source is the conversion of isoprene emissions.

To model nitrogen oxides produced by lightning, the GISS convection scheme is first used to calculate both the total lightning, and the cloud-to-ground lightning frequencies interactively in each grid box and at each time step. Then the production rate of NO_x from lightning (Price et al., 1997) is used to derive the NO_x produced, including the vertical distribution of the lightning produced NO_x (K. Pickering, personal communication, 1997). Phase trans-

formation and removal of soluble species is calculated using a wet deposition scheme as in Koch et al. (1999).

Surface dry deposition is calculated using a resistance-in-series model (Wesley and Hicks, 1977) coupled to a global, seasonally varying vegetation data set as in Chin et al. (1996). Note that the leaf area indices are therefore not connected with the GCM's land-surface component. Aerodynamic resistances are based on the model's surface heat and momentum fluxes, as in Koch et al. (1999).

The above chemistry scheme has been installed directly into the latest version of the GISS climate model, II'. This is a primitive equation model, run here with nine vertical sigma layers, centered at 959, 894, 786, 634, 468, 321, 201, 103, and 26.5 mbars, and horizontal resolution of 4×5 degrees (latitude \times longitude). The GCM's physics time step is one hour, so that changes to tracer masses from transport and chemistry are both applied every hour.

The capability of the GISS GCM to accurately model the transport of trace species has been greatly improved recently. All the chemical tracers, along with heat and moisture, are advected with a quadratic upstream scheme (Prather, 1986). (Momentum advection uses a fourth-order scheme.) Improvements to transport both within the boundary layer and across the boundary layer edge, along with convection, are especially important, since trace gas emissions at the Earth's surface come from discrete, spatially inhomogeneous sources. A primary example of the improvements can be seen in the interhemispheric exchange times of CFC-11 and 85Kr. In the earlier version of the model, the exchange times were roughly a factor of two too long, whereas in the new version, the values are within 15–25% of observations (Rind and Lerner, 1996). Improvements in the interhemispheric exchange time occurred because the alterations in both the boundary layer and convection schemes improved the tropical precipitation and wind fields. Interestingly, neither change by itself was overly effective, a result which illustrates the highly non-linear nature of GCM interactions. The GISS GCM II' has been used previously for ozone (Mickley et al., 1999) and sulfate (Koch et al., 1999) simulations.

The use of water vapor as an online, chemically active tracer is another important feature of this model. Most chemical models assume that water vapor is constant since they do not have a detailed model of the hydrological cycle, while most climate models do not have any chemistry. Thus the interaction between water vapor and climate has seldom been examined, although it is thought to be one of the key issues in climate modeling (IPCC, 1995).

Calculated O_3 and NO_2 fields are used in the GCM's computation of radiative heating and fluxes, which is performed every five hours. These fully interactive chemical constituent changes are therefore able to affect the mete-

orology in the GCM. To explore the influence of this feedback, we have also performed simulations where this feedback was not allowed, in which case the model used climatological O₃ and NO₂ distributions in its radiation calculations. In addition to O₃, the GCM calculates radiative absorption and emission from water, CO₂, N₂O, chlorofluorocarbons and methane in the longwave, and CO₂, NO₂ and O₂ in the shortwave (Hansen et al., 1983). Change in tropospheric O₃ plays an important role in the radiative heating that drives tropospheric meteorology, through their absorption in the Huggins bands in the ultraviolet, the Chappius bands in the visible, the 9.6 micron and especially the 14 micron bands in the infrared (Shine et al., 1995; van Dorland et al., 1997).

A key output from the O₃ photochemistry model is a one-year time series of estimated hourly ground-level O₃ concentrations in the 4⁰ x 5⁰ grid covering the MEC region. (Note that this large grid size, covering most of New York State, is a limitation of the model used in the current application. In future developments, a regional-scale air quality model, nested within the GCM, should enable finer geographic analyses.) The model was run for the years 1999, 2030, and 2100. From the hourly data, we computed the one-hour and eight-hour maxima for each day. The daily maxima were then averaged over each year to obtain a summary measure of O₃ levels for each target year. The difference between the means for 2030 and 1999 and for 2100 and 1999 were computed. These differences characterized changes in total O₃ exposure between the base year (1999) and the projected years (2030 and 2100).

Results

The impacts of a given change in ambient O₃ concentrations on asthma and total respiratory hospital admissions have been characterized in several recent studies, as noted above (Thurston et al., 1992, 1994; Burnett et al., 1994). Table 7-9 summarizes the risk coefficients derived from these studies. The coefficients represent estimates of the average population risk of being admitted to the hospital when O₃ concentrations increase by 100 parts per billion (ppb). Note that typical daily maximum one-hour O₃ concentrations in the MEC summer range from 60 to 100 ppb, with occasional peaks extending up to 150 ppb or higher. In the initial work presented here, we calculated a weighted average risk coefficient for total respiratory admissions using the inverse squared errors of the individual coefficients as weights, yielding a mean risk coefficient of 1.5 admissions per 100 ppb ozone per day per 1,000,000 persons. Similarly for asthma, a mean risk coefficient of 1.2 was calculated. These coefficients are assumed to represent the average daily risk faced by a group of 1,000,000 persons in the MEC region per 100 ppb increase in O₃. Note that these risk coefficients have been

used as point estimates in the present analysis. An alternative method for combining risk estimates using a random effects model would incorporate variability in risk between studies. Such an approach, along with uncertainty estimates on the O₃ estimates, would enable an explicit consideration of uncertainties.

To calculate increases in hospitalizations under climate change scenarios, we multiplied the mean risk coefficient by the changes in annual mean ozone concentrations (using the 8-hour daily maxima) from 1999 to 2030 and to 2100 (in units of 100 ppb) obtained from the ozone chemistry model. Our use of 8-hour average ozone estimates along with risk coefficients based on 1-hour average regressions may lead to an underestimate of the magnitude of the projected ozone impacts. The result was then multiplied by 365 to cumulate over the year, and then multiplied by the projected MEC population in millions. For the present analysis, we have assumed that the MEC population remains constant over time. This can be considered a conservative assumption, since increases in total population, and/or shifting to the right of the age distribution, would lead to larger impacts. Underestimates may be especially great in suburban regions where rapid growth is anticipated.

The calculations described above yielded estimates, for the MEC region as a whole, and for the New York state MEC counties, of the numbers of additional total respiratory and asthma hospital admissions that might occur in 2030 and 2100 as a result of increasing ozone exposures. For the New York state counties, we further divided the total admissions by the numbers of hospitalizations in 1996 to calculate the percent increase in annual hospitalizations at the two time points.

Results are displayed in Table 7-10. In 2030, we estimate an increase in annual average 8-hour daily maximum ozone concentrations of about 12 ppb. For the MEC region as a whole, this corresponds to an increase of 995 and 819 annual hospital admissions for total respiratory causes and asthma, respectively. About 80% of these increased admissions occur in the 14-county part of the MEC region in New York State (804 and 643 for total and asthma, respectively), directly proportional to the fractional population residing there. These increases represent only a 0.6% rise in annual hospital admissions for total respiratory causes, but a somewhat larger 1.6% rise in asthma admissions. In 2100, a much larger annual average ozone increase of about 51 ppb is projected. For the MEC region as a whole, increases of 4,149 and 3,319 are projected for total respiratory and asthma admissions, respectively. For the New York state MEC region, increases of 3,552 and 2,682 are calculated for these two admissions categories. These increases represent a 2.5% rise in annual hospital admissions for total respiratory causes, and a 6.5% rise in asthma admissions.

CHALLENGES AND OPPORTUNITIES

These results suggest that in the short term (i.e., up to the year 2030), impacts of climate change on ground-level ozone concentrations are not likely to have a large impact on asthma and other respiratory hospitalizations in the MEC region. Impacts of the size we calculated (0.6% and 1.6% increases in hospital admissions) are not likely to be discernable given the interannual variability in hospital usage. By the year 2100, the impacts become more significant, especially for asthma. These preliminary results illustrate a general point, i.e., that it becomes more important for climate change impacts to be factored into policy decisions regarding ozone mitigation strategies as the planning horizon becomes more long-term. This comment is reinforced by the fact that our analysis has ignored demographic shifts that might result in even larger ozone-related public health impacts in a growing and aging MEC population.

It is important to emphasize the limited scope of the preliminary analyses presented here. Because we did not have access to an appropriate aerosol model, we have ignored possible effects of climate changes on fine particle concentrations and the wide-ranging health impacts that could result from such effects. We have restricted attention to only two of the known health effects of O₃, hospitalizations for asthma and total respiratory causes. Mortality effects could be readily incorporated into future extensions of these analyses. Also, potential interactions between air pollution and heat stress effects have been ignored. Finally, the economic costs of air pollution-related health impacts have not been assessed in the present work.

Although equity issues have been ignored in the impact analysis presented here, it is important to recall that current health status varies tremendously across the MEC region (see e.g., Figure 7-2). Given these disparities in health status, it appears likely that the public health impacts of air pollution in the MEC region will disproportionately affect those who are most vulnerable, including the very old, the

very young, and those with pre-existing health impairment such as asthma.

INTEGRATION ACROSS SECTORS

There are several areas of potential integration between the health sector and other MEC analysis sectors. One key area is that of peak energy demand during summer hot spells. The hot humid conditions that are most likely to adversely impact human health are the same conditions that place the largest stress on the energy supply infrastructure of the MEC region. Recent examples of capacity problems in the MEC include a three-day power outage in the summer of 1999 that impacted a wide area in northern Manhattan. Among other impacts, significant freezer sample losses were experienced in biomedical laboratories of the Columbia Presbyterian Medical Center. The additional capacity that will be needed to supply peak energy demands under a warming and more variable climate will likely result in increased emissions of air pollution in the MEC regions, including ozone precursors.

Another area of integration is between wetlands and vector-borne diseases. As alluded to in the earlier discussion, mosquito-borne diseases have assumed an increasingly prominent stature among potential public health threats in the MEC region. Attitudes and policies directed towards wetlands are likely to have important effects on vector population dynamics. Conversely, policies adopted in response to outbreaks of mosquito-borne diseases like West Nile virus have the potential, unless managed carefully, to cause significant harm to regional wetlands.

INFORMATION AND RESEARCH NEEDS

Further research is warranted in several areas. First, additional scenarios of ozone health impacts could be developed that include alternative assumptions about 1) the demo-

TABLE 7-10

Projected increases in hospital admissions resulting from increased ground-level O₃ concentrations in 2030 and 2100 associated with climate change

Region	Hospital Admissions Category	2030			2100		
		O ₃ Increase	New Hospital Admissions	Percent Change in Admissions	O ₃ Increase	New Hospital Admissions	Percent Change in Admissions
MEC	Total Respiratory	12.15 ppb	995	*	50.65 ppb	4,149	*
	Asthma		819	*		3,319	*
NY State Counties	Total Respiratory		804	+0.6%		3,552	+2.5%
	Asthma		643	+1.6%		2,682	+6.5%

*Unable to calculate due to the unavailability of hospital admissions statistics for NJ.

graphic makeup of the MEC region in future years, and 2) differential risk coefficients for different demographic groups. Uncertainties about these factors may represent the largest sources of uncertainty in predicting future health impacts of ozone. The analysis should be extended to PM and to other health outcomes. Another promising research direction is to analyze the independent and interactive health impacts of heat stress in conjunction with air pollution should be considered. The availability of spatially detailed surface temperature maps obtained from satellite imagery offers the potential to carry out epidemiologic studies, and risk assessments, using small geographic units. Research that seeks to model interactions across sectors, such as those discussed above, is a final area that warrants further work.

POLICY RECOMMENDATIONS

The wide range of possible impacts of climate change and variability on human health in the MEC region provide a strong rationale for incorporating climate change models into future policy decisions regarding mitigation of heat stress, vector- and water-borne diseases, and air pollution in the MEC region. We developed a model for analyzing climate change impacts on air pollution-related health effects—using O₃ effects on hospitalizations as a case study. This analytical framework can be extended to include additional pollutants and health outcomes, potentially providing a comprehensive assessment of such effects. Our analysis suggests that climate change impacts should be included as one of the considerations in developing long-range strategies directed towards ground-level ozone mitigation in the MEC region.

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