

Exposure to New York City as a Risk Factor for Heart Attack Mortality

NICHOLAS CHRISTENFELD, PhD, LAURA M. GLYNN, PhD, DAVID P. PHILLIPS, PhD, AND ILAN SHRIRA, BA

Objective: If New York City (NYC) residents' unusually high rate of ischemic heart disease (IHD) results from chronic exposure to that city, there might also be an effect of acute exposure among visitors to NYC. We explored this possibility and also whether IHD is reduced among NYC residents dying away from the city. **Methods:** Using all US death certificates for 1985–1994, we examined (correcting for age, race, and sex) IHD deaths in three groups: NYC residents who died in the city, non-NYC residents visiting the city, and NYC residents traveling out of the city. **Results:** IHD deaths among NYC residents dying in the city were 155% of the expected proportion ($p < .0001$). Among visitors to the city, such deaths were 134% of the expected proportion ($p < .0001$). The proportion of IHD deaths among NYC residents dying out of the city was only 80% of the expected value ($p < .0001$). These effects are not due to nearby commuters, recent immigrants, local classification practices, or socioeconomic status, and they do not appear in other US cities. **Conclusions:** With both chronic and acute effects of exposure to NYC, these data are consistent with the hypothesis that the stress of NYC is linked to the high rate of IHD. **Key words:** heart disease, acute stress, chronic stress, New York City.

NYC = New York City; ICD-9 = International Classification of Diseases, ninth edition; IHD = ischemic heart disease; PMR = proportionate mortality ratio; SES = socioeconomic status.

INTRODUCTION

Residents of NYC have a notably higher-than-expected rate of deaths due to IHD, an effect that is not so pronounced, and often not present at all, in other major US cities (1, 2). Possible explanations for this effect include differences in diet, exercise habits, the type of people who choose to live in NYC, and exposure to stress. One way to shed light on this issue is to examine whether the heightened risk of IHD is detectable not only for residents, who suffer chronic exposure to the city, but also for visitors to NYC, who suffer acute exposure. One can also examine whether there is a reduction in IHD for NYC residents when they are away from the city. Some of the explanations for an excess of IHD in NYC do not clearly predict effects for visitors. For example, the hypothesis that NYC has a high concentration of cardiac-prone people does not imply that visitors would be at elevated risk or that residents traveling out of the city would be at reduced

risk of IHD. Similarly, explanations for the excess of IHD in NYC such as diet or exercise should require that people suffer chronic exposure to the city and so also do not predict an elevation for visitors or a reduction for those leaving NYC. However, other sorts of explanations, such as the putative stress of being in NYC, suggest that one might find effects not only for residents but also for visitors. Thus, the existence of acute effects of exposure to NYC would provide clues about the cause of the high rate of IHD in NYC and also provide information about chronic vs. acute risk factors for heart attacks.

METHODS

We examined all US deaths for the decade ending in 1994, the latest year for which US computerized death certificates are available (3). Among other things, these certificates list cause and location of death and location of the decedent's residence. Because the precise frequency and duration of visits into and out of NYC cannot be known, it is not possible to calculate a death rate for visitors. However, because the total number of people who died while visiting the city is available, the proportion of all such deaths that were attributed to IHD can be used as the indicator of whether IHD deaths are overrepresented. Consequently, to examine IHD deaths, we used a PMR (4) rather than the population's IHD rate. The PMR is the ratio (multiplied by 100) of the observed proportion of deaths due to IHD to the expected proportion of such deaths. Thus, for example, an IHD PMR of 200 indicates that twice the expected proportion of deaths were due to IHD, and a PMR of 100 indicates that the observed proportion was no different from the expected proportion.

The PMR allows one to assess the overrepresentation of a disease in a population whose precise size is not known but for which all deaths are recorded (5). PMR calculations can be performed for NYC residents who died in NYC, for NYC residents who died out of the city, and for people who died while visiting NYC. The PMR does not directly assess the odds of a NYC visitor dying of heart disease. However, if being in NYC increases the risk of IHD more than it

From the University of California, San Diego (N.C., D.P.P., I.S.), La Jolla, CA, and University of California, Irvine (L.M.G.), Irvine, CA.

Address reprint requests to: Nicholas Christenfeld, Department of Psychology, University of California, San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0109. Email: nicko@ucsd.edu

Received for publication February 26, 1999; revision received June 3, 1999.

RAPID COMMUNICATION

increases the risk of dying of other causes, then the proportion of IHD deaths should be elevated for those visiting the city.

We conducted three basic analyses using the PMR. For the first analysis, we compared the observed number of IHD deaths for NYC residents who died in the city to the expected number of such deaths. The expected number was calculated from the proportion of IHD deaths in the rest of the United States among those who died in their county of residence. To compare observed and expected figures, we standardized simultaneously on the basis of sex, age (in decades), and race (white, black, Hispanic, and other) (6). This standardization procedure was used in all analyses. To calculate expected IHD deaths, we calculated separately for each age/race/sex group the proportion of deaths in the rest of the United States that were due to IHD. We then applied this proportion to the number of members of that group who died in NYC during the study period. For example, 27% of Hispanic male octogenarian deaths were due to IHD in the rest of the United States. There were 2032 male Hispanic octogenarian deaths in NYC, so we expected 549.4 to be due to IHD. This expected number was then summed across all groups to get the standardized expected number of NYC IHD deaths. This value, divided by the total number of deaths, also gives the expected proportion of IHD deaths.

For the second analysis, we compared the observed number of IHD deaths for visitors who died in NYC to the expected number of such deaths. The expected number was calculated from the proportion of IHD deaths in the rest of the US population among those who died outside their county of residence. This comparison group controls for any differences in IHD risk for people who are likely to be traveling when they die.

For the third analysis, we compared the observed number of IHD deaths among NYC residents dying outside the city to the expected number. This expected number was calculated from the proportion of IHD deaths of NYC residents who died in the city, with an adjustment for the reduction in IHD proportions for the general population of people who died away from their county of residence. Identical analyses were conducted for IHD deaths (ICD-9 codes 410–414) as well as for heart disease in general (ICD-9 codes 390–398, 402, and 404–429). Parallel analyses were conducted for residents of the 10 largest US cities other than NYC (Los Angeles, CA; Chicago, IL; Houston, TX; Philadelphia, PA; San Diego, CA; Dallas, TX; Phoenix, AZ; Detroit, MI; San Antonio, TX; and San Jose, CA).

RESULTS

The number of IHD deaths for New Yorkers dying in NYC is significantly ($p < .0001$) higher than expected (Table 1). The excess in IHD deaths amounts to an average of 8074 cases per year, and the size of the effect is consistent with other data on the rate of IHD deaths in NYC (1, 2). Visitors to NYC experience about 262 more IHD deaths per year ($p < .0001$) than would be expected from the mortality of people who died while visiting other places. NYC residents who died out of the city show a greater reduction in the proportion of IHD deaths than do residents of other places who died out of their home counties ($p < .0001$). On average, there are 310 fewer annual IHD deaths than expected among NYC residents out of the city.

TABLE 1. Observed and Expected Deaths Due to IHD for Those Who Live in NYC, Visit NYC, and Leave NYC, 1985–1994

	Residents of NYC	Visitors to NYC	Residents Away From NYC
Number of IHD deaths			
Observed	225,333	10,621	11,318
Expected number	144,592	7,998	14,414
Observed – expected	80,740	2,623	–3,096
Total	698,311	43,775	44,475
Percentage of IHD			
Observed	32.3	24.3	25.4
Expected	20.7	18.3	32.4
IHD PMR	155.8 ^a	132.8 ^a	78.5 ^a
95% CI	155.2–156.5	130.3–135.3	77.1–80.0

^a Significantly different from 100; $p < .0001$, two tailed (4).

Alternative Explanations.

The reported effects are not due to differences in the age, race, or sex of NYC residents or visitors; the analyses control for these demographic factors. Furthermore, resident and visitor effects are detectable when analyses are restricted to whites, blacks, or Hispanics, which reduces the likelihood that the effects could be due to unusual concentrations in NYC of particular ethnic or religious groups. The findings are also unlikely to result from people coming to NYC to seek treatment for heart disease, because that cannot explain the high proportion of IHD deaths for residents. In addition, because the data are analyzed using a PMR rather than a death rate, such an explanation depends on people going to New York for treatment of heart disease more than for treatment of other diseases. In other words, if people came to NYC seeking treatment for a wide range of diseases, then the number of visitors who died might be high, but the proportion of visitors dying from IHD would not necessarily be affected.

Although ethnic differences and people seeking medical treatment in NYC are unlikely to account for the NYC patterns, six other possibilities require further analyses. First, many visitors to NYC might live in nearby suburbs and commute to NYC for work. If, for example, a heart attack is more likely at work and people commute to work in NYC, the elevated rate for visitors could then reflect a difference between being at work and being at home. Second, the effects could be due, at least in part, to an unusually high concentration of recent immigrants in NYC. Third, the effects could result from SES differences between the sort of people who live in, visit, and leave NYC and the sort who live in, visit, and leave other parts of the country. Fourth, NYC death registrars might be especially likely

to ascribe heart disease deaths to IHD (rather than to other forms of heart disease). Fifth, the amount of walking people do in NYC may overtax the cardiovascular systems of normally sedentary visitors. Sixth, the visitor effects might not be specific to NYC but might be effects of cities in general.

To evaluate whether the documented effects hold only for commuters, we examined IHD deaths for "long-distance" visitors, that is, those who visited NYC from states outside the northeast, and for NYC residents who visited states outside the northeast. In both cases, the effects on IHD remain highly significant. For visitors to NYC from more distant states, the observed proportion of IHD deaths was significantly higher than expected (PMR = 150.0, $p < .0001$). For New Yorkers visiting distant states, the observed proportion was significantly lower than expected (PMR = 81.7, $p < .0001$). These findings suggest that the visitor effects are not due simply to commuters.

To explore the possibility that the observed effects are due to an unusually high concentration of recent immigrants in NYC, we performed the same three basic analyses but excluded everyone whose death certificate indicated a foreign, or unknown, place of birth. This changed none of the results: The PMR for US-born residents of NYC was 150.1 ($p < .0001$); for visitors, it was 128.5 ($p < .0001$); and for NYC residents dying out of the city, it was 77.1 ($p < .0001$). The observed patterns cannot be attributed to recent immigrants in NYC.

Death certificates do not provide direct information on SES, and although some states report the decedent's occupation, New York does not. However, starting in 1989, total years of education is reported, and this is useful marker of SES (7). Accordingly, to check whether the NYC effects could be due to SES differences between groups, the analyses were standardized on the basis of years of education (as well as age, race, and sex) for the 6 years that this information was available. All three findings remain largely unchanged: Residents had an IHD PMR of 159.2 ($p < .0001$); visitors, 140.0 ($p < .0001$); and NYC residents dying out of the city, 72.0 ($p < .0001$). Furthermore, all three effects are present when analyses are restricted to college graduates, high school graduates, or those without a high school degree. Differences in SES between those living in or visiting NYC and other parts of the United States do not seem to be capable of explaining the differences in the proportion of deaths due to IHD.

To test whether the NYC effects result from geographic differences in methods of classifying heart disease deaths, we examined all forms of heart disease, not just IHD. This analysis reveals the same three

effects as the IHD analysis. NYC residents dying there had an elevated PMR for heart disease (PMR = 123.5, $p < .0001$), visitors to NYC also had an elevated PMR for heart disease (PMR = 110.5, $p < .0001$), and NYC residents who died out of the city had reduced proportions of heart disease (PMR = 88.4, $p < .0001$). These findings suggest that the NYC effects cannot be explained by NYC registrars preferring IHD as a classification category.

Perhaps visitors to NYC walk more than they normally do, and this unusual physical strain is enough to increase the number of deaths attributable to IHD. This explanation does not suggest why NYC residents should show an elevated PMR (and might even predict the opposite), but it might explain the elevated IHD PMR for visitors to NYC. However, if the visitor effect is due to walking, a similar visitor effect should be apparent in San Francisco, CA, another city where walking is common. This is not the case. Visitors to San Francisco actually show a slightly depressed, rather than elevated, IHD PMR (89.3, $p < .0001$).

To examine whether the elevated IHD level for NYC visitors and reduced level for those leaving NYC are more general phenomena of major urban areas, we repeated the analyses for the 10 largest cities in the United States after NYC. For these cities, taken as a group, there is very little elevation in the PMR for heart disease among residents (PMR = 102.1, $p < .0001$). Given the very small effect of chronic exposure to these cities, one would not expect a large effect of acute exposure. Consistent with this expectation, visitors to these cities show only a very slightly elevated heart disease proportion (PMR = 101.1, $p < .005$). The excess amounts to about 7 deaths per city per year. There is a slight effect for those leaving these cities, although in the direction opposite that observed in NYC (PMR = 107.0, $p < .0001$). This excess averages 29 excess heart disease deaths per city per year. Thus, none of the NYC effects is particularly apparent in other large cities: Residents of other big cities do not display particularly high levels of heart disease, visitors to these cities do not show notably elevated heart disease levels, and people leaving these cities actually show a slight increase, rather than decrease, in the proportion of heart disease deaths.

DISCUSSION

People who live in NYC are unusually likely to die of IHD, and we have documented a similar effect for those visiting the city as well as a reduction in IHD for NYC residents away from the city. The acute effects are not as large as the chronic effects, although, with an impact on hundreds of people each year, the effects are

RAPID COMMUNICATION

well beyond anything plausibly obtainable by chance. These mortality effects seem to be confined to NYC: Other major cities do not show the marked excess for residents, nor do they show the acute effects for visitors.

Elevated IHD proportions for residents and visitors to NYC could conceivably be attributed to local drinking water or air quality. However, although air quality has been linked to heart disease (8, 9), NYC does not have unusually high levels of air pollution (or low levels of water quality) (10, 11), and pollution explanations cannot account for other large cities with pollution at least as severe not showing high IHD proportions for either residents or visitors.

The finding that NYC that is associated with IHD in both residents and visitors is consistent with the notion that stress might be involved, although other explanations are still certainly possible. Both chronic and acute stress have been linked to heart disease: People with highly stressful jobs show an elevated heart disease rate (12–16), and those who are experiencing anger (17), job deadlines (18), or earthquakes (19) are more likely to have a heart attack. The data do not reveal whether the mechanism linking being in NYC and suffering fatal IHD is the same for visitors and residents. It is possible that stress, in the long run, takes its toll by accelerating the atherosclerotic process. In the short run, stress associated with NYC may be enough to trigger a heart attack among visitors who already have heart disease, and leaving the city may postpone, briefly, a heart attack among residents who are at high risk. If stress is in part responsible for our findings, the data do not indicate whether it is the fast pace, late nights, excitement, fear, or some other aspect of NYC that is critical. NYC has by far the highest population density of any region of the United States (20), and density has been linked to a variety of negative health outcomes (21–23). With other sorts of data, such as the activities of the decedent immediately before IHD death, it should be possible to shed more light on the particular aspects of NYC that trigger fatal ischemic events and also on why NYC is so different from other cities.

This work was supported in part by a grant (D.P.P.) from the Marian E. Smith Foundation.

REFERENCES

1. National Center for Health Statistics. Vital statistics of the United States, 1994. Vol II, Pt A. Washington DC: US Government Printing Office; 1994.
2. McNutt L, Strogatz DS, Coles FB, Fehrs LJ. Is the high ischemic heart disease rate in New York State just an urban effect? Public Health Rep 1994;109:567–70.
3. National Center for Health Statistics [database online]. Mortality detail file (1985–1994). Hyattsville (MD): US Department of Health and Human Services; 1987–1997.
4. Mausner JS, Kramer S. Epidemiology: an introductory text. Philadelphia: WB Saunders; 1985.
5. Phillips DP, Welty WR, Smith MM. Elevated suicide levels associated with legalized gambling. Suicide Life Threat Behav 1997;27:373–8.
6. Bland M. An introduction to medical statistics. 2nd ed. Oxford, UK: Oxford University Press, 1995.
7. Winkleby MA, Fortmann SP, Barrett DC. Social class disparities in risk factors for disease: eight-year prevalence patterns by level of education. Prev Med 1990;19:1–12.
8. Schwartz J. Air pollution and hospital admissions for heart disease in eight US counties. Epidemiology 1999;10:17–22.
9. Morris RD, Naumova EN, Munasinghe RL. Ambient air pollution and hospitalization for congestive heart failure among elderly people in seven large US cities. Am J Public Health 1995;85:1361–5.
10. New York City Department of City Planning. Annual report on social indicators. NYC DCP 97–12. New York: Department of City Planning; 1996.
11. Office of Air Quality Planning and Standards. National air pollution emission trends, 1900–1994. Washington DC: US Environmental Protection Agency; 1995.
12. Schnall PL, Landsbergis PA, Baker D. Job strain and cardiovascular disease. Annu Rev Public Health 1994;15:381–411.
13. Theorell T, Karasek RA. Current issues relating to psychosocial job strain and cardiovascular disease research. J Occup Health Psychol 1996;1:9–26.
14. Schwartz JE, Pickering TG, Landsbergis PA. Work-related stress and blood pressure: current theoretical models and considerations from a behavioral medicine perspective. J Occup Health Psychol 1996;1:287–310.
15. Bosma H, Peter R, Siegrist J, Marmot M. Two alternative job stress models and the risk of coronary heart disease. Am J Public Health 1998;88:68–74.
16. Theorell T, Tsutsumi A, Hallquist J, Reuterwall C, Hogstedt C, Fredlund P, Emlund N, Johnson JV. Decision latitude, job strain, and myocardial infarction: a study of working men in Stockholm. The SHEEP Study Group. Stockholm Heart Epidemiology Program. Am J Public Health 1998;88:382–8.
17. Mittleman MA, Maclure M, Sherwood JB, Mulry RP, Tofler GH, Jacobs SC, Friedman R, Benson H, Muller JE. Triggering of acute myocardial infarction onset by episodes of anger. Determinants of Myocardial Infarction Onset Study Investigators. Circulation 1995;92:1720–5.
18. Mittleman MA, Malone MA, Maclure M, Sherwood JB, Muller JE. Workplace stress as a trigger of acute myocardial infarction [abstract]. Circulation 1998;97:P15.
19. Leor J, Poole WK, Kloner RA. Sudden cardiac death triggered by an earthquake. N Engl J Med 1996;334:413–9.
20. Wright JW. New York Times almanac. New York: Penguin Press; 1998.
21. Laird JT. Mental health and population density. J Psychol 1973; 85:171–7.
22. Funkenstein DH. Physiology of fear and anger. Sci Am 1955; 192:74–80.
23. Jain U. The psychological consequences of crowding. New Delhi: Sage; 1987.