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Cocaine Use and Death During Heat Waves

The heavy toll in human lives claimed by heat waves is increasingly evident in the medical literature. During the heat wave of 1980 in St Louis and Kansas City, Mo, 308 persons more than the number expected to die during that period lost their lives. At the worst of the heat wave, daily mortality in St. Louis increased to 3 times the usual rate.[1] More recently, in the summer of 1995 in Chicago, Ill, severe heat-related mortality claimed more than 700 victims, and daily mortality peaked at more than 5 times the usual rate.[2]

See also p 1795.

Heatstroke is the only syndrome that is clearly attributable to environmental heat and that has a substantial death-to-case ratio. It might therefore seem logical to ascribe heat wave–related mortality largely to heatstroke. However, an accumulating body of literature strongly suggests that this is not the case. In most heat waves, only a minority of "excess deaths" (ie, those deaths that occur above the usual number for that place and season) are attributable to heatstroke.[3] Thus, any new information on the causes of nonheatstroke excess deaths increases understanding of the effects of sustained high temperatures on a population's health.

In their article published in this issue of THE JOURNAL, Marzuk and colleagues[4] make a significant contribution by presenting substantive evidence that the risk of death from cocaine overdose increases sharply during hot weather. Working with data from the New York City Medical Examiner's Office, the investigators carefully and systematically identify deaths attributable to cocaine overdose and show these deaths to be more frequent during hot weather in New York.

The ability to use these findings in prevention will be critically dependent on whether the association between heat and deaths from cocaine overdose represents true cause and effect. The study design used by Marzuk et al shares some features in common with ecological studies (ie, based on group rather than individual information), which are highly susceptible to confounding. Temperature was measured as a single, daily number for the entire population from the weather station in Central Park. Daily data on the thermal microenvironments of individuals were obviously unavailable.

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The absence of individual data makes it difficult to evaluate possible confounders that might explain away an apparently causal association. In this study, data are insufficient to determine even whether the cocaine users with the hottest individual microenvironments were the ones at highest risk of overdose death, which is what would be expected if heat caused the mortality excess. The possibility cannot be ruled out that some factors or events closely associated in time with hot weather account for the increase in deaths from cocaine overdose.

Nevertheless, the authors have done a commendable job in seeking and presenting evidence that tends to refute the most likely alternative explanations for their findings. For example, if the overall number of persons using street drugs increased during hot weather, then an increase in cocaine-related deaths might represent only increased cocaine use, not an increased risk among a constant number of users. But if there were truly an increased number of drug users during hot weather, then overdose deaths from opiates and other drugs should increase in hot weather as well. The authors found that overdose deaths from opiates and other drugs do not increase in hot weather, implying an effect of heat that is specific to cocaine.

The authors also evaluated 2 comparison groups (homicides and motor vehicle crash fatalities) for evidence of increased cocaine use during hot weather and found none, further diminishing the likelihood that the increase in cocaine overdose deaths is due to increased cocaine use in the population at risk.

Another indication of a cause-and-effect relationship is that the 1993-1995 "cross-validation" sample shows what appears to be a gradual, essentially monotonic increase in risk of death above a threshold temperature. This finding could represent a biological gradient or dose-response effect of heat, a frequent attribute of causal relationships.

Moreover, a causal association here makes biological sense. Both cocaine and environmental heat stress have substantial metabolic and cardiovascular effects. Increased body temperature also has been implicated as a contributor to cocaine overdose death.[5] Plausibly, joint effects of cocaine and environmental heat could be additive or synergistic and result in more frequent fatal outcomes of cocaine use.

The findings of Marzuk et al add yet another etiology to the list of known or suspected causes of excess mortality in populations exposed to prolonged periods of high environmental temperature. Other than heatstroke, the causes most consistently associated with excess deaths and accounting for the greatest number of them are cardiovascular disease (largely ischemic heart disease), cerebrovascular disease, and pulmonary diseases.[3]

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The statistics on these 3 disease categories are impressive. In 7 studies of excess deaths by diagnosis during heat waves occurring since 1934, deaths due to cardiovascular disease increased 7%–59% over baseline, accounting for 13%–42% of heat wave–related excess deaths. Similarly, in the same 7 heat waves, cerebrovascular disease increased 25%–104% over baseline, accounting for 6%–52% of heat wave deaths. In 6 of the same 7 heat waves, respiratory deaths increased from 0% to 84% more than the usual rate and accounted for from 0% to 14% of heat wave deaths.[6-10] Other putative causes of heat wave–associated excess deaths have arisen less regularly and were smaller in the magnitude of their apparent effects. Such associations may represent coincidence.[3]

Although statistically significant, the magnitude of increase in daily deaths on the "hot days" documented by Marzuk et al is relatively small. For days with temperatures greater than or equal to 31.1°C (88°F) the authors recorded a mean increase of only 0.58 deaths per day. In contrast, in 5 New York City heat waves occurring between 1972 and 1996, the average number of daily excess deaths was much higher, ranging from 50 to 143 per day.[11]

It would be inadvisable, however, to dismiss the findings of Marzuk et al as quantitatively insignificant based on these data. A heat wave represents a prolonged period of above-normal temperatures spanning several days or weeks. Heat wave days are by no means the same as the "hot days" defined by Marzuk et al. Considering that the effects of heat are cumulative over several days, the impact of heat on cocaine overdose deaths may be greater during a heat wave than otherwise. Since the investigators' "hot days" did not all occur during heat waves, it is to be expected that the measured effect of the heat would be smaller than during a heat wave.

High priority should now be given to resolving the remaining doubts as to whether the heat wave–associated increase in cocaine overdose deaths represents actual cause and effect. Investigators at institutions in other cities should attempt to replicate the findings of Marzuk et al when they experience local heat waves. To the extent feasible, more information on person-specific risk factors (eg, presence of a home air conditioner, dose of cocaine taken) should be gathered. Ideally, study case patients should be compared at an individual level with cocaine users who survive the heat to identify the important determinants of survival. In this way, practitioners will be able both to assess whether a new intervention is needed to prevent cocaine deaths in the heat and also to identify the risk modifiers likely to be involved in any program to reduce the hazard.

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Preparticipation Screening of Athletes

In this issue of *JAMA*, the article by Glover and Maron[1] addresses the issue of improving the sensitivity of the preparticipation examination (PPE) to detect silent, clinically important cardiovascular abnormalities that place the athlete at risk for sudden cardiac death (SCD). Fortunately, SCD is rare in high school athletes. But the appeal of being able to identify risk factors and thereby prevent SCD, coupled with the legal obligation for sports organizations and institutions to provide a safe environment for athletes points to the need to consider, wherever possible, ways to improve the sensitivity of the PPE. Glover and Maron have identified the following problems with the high school PPE process: (1) 16% of states have no approved history and physical examination forms; (2) of the 84% of states that do, questions deemed essential for detecting abnormalities were missing from half of the history forms and more than 60% of the physical examination forms; and (3) only 40% of the states had questionnaires that incorporated the majority of 1996 American Heart Association recommendations.

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Even before these new data were available, most clinicians who perform these examinations were aware that the PPE was not a perfect process. There are both quality and compliance issues. Bradford and Lyons[2] surveyed 114 public school districts in Pennsylvania and found that in one third of the cases the examinations lasted less than 5 minutes, 25% included no medical history, and 60% had no musculoskeletal

See also p 1817.

examination. In addition, the PPE is considered primarily to be an examination for the purpose of assigning financial responsibility and legal liability despite the consistent finding that this examination is the only routine primary or preventive health care received by the majority of athletes.[3-6] What this means is that most examinations are sport-referenced and sport specific; identification of the causes for disqualification and assessment of readiness to play are the essential attributes of most PPEs rather than the principles of health-referenced periodic examinations for patients in a given age range.

In addition, considerable resources are required to perform screening examinations, yet insurance reimbursement is not forthcoming and the institutions mandating PPEs seldom pro-

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