tant implications. It is mentioned that the majority of patients received daily self-administered treatment. However, it is not clear what treatment regimen(s) was used in the remaining patients. Information regarding the treatment regimen(s) used, the duration of treatment for pulmonary and extrapulmonary/disseminated tuberculosis, and whether the drug dosage was individualized according to the body weight of the patient needs to be clarified. Conventionally, in patients with drug-sensitive tuberculosis, pyrazinamide does not give any additional benefit when administered for more than 2 months in short-course regimens (6). Therefore, it also would be interesting to know what time after the initiation of ATT did drug-induced hepatotoxicity develop in the patients in the study by Yee and colleagues (1).

Yee and colleagues (1) do not mention the associated comorbid illnesses the patients in their study had and the other medications the patients were receiving together with ATT. It also is not clear whether the human immunodeficiency virus–positive patients with tuberculosis also were receiving antiretroviral treatment concomitantly. These details are required to meaningfully interpret the published results.

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From the Authors:

We agree with Drs. Mohan and Sharma that pyrazinamide (PZA) is an essential component of modern short-course chemotherapy because it accelerates microbiological improvement in the first 2 months, allowing reduction of the total duration of therapy to 2 months (1, 2). In light of recent reports of serious toxicity with the 2-month rifampin (RIF) and PZA regimen for latent tuberculosis (TB) infection (3, 4), information regarding incidence and risk factors of adverse events related to this drug is of great interest. As noted by Drs. Mohan and Sharma, there is little published information on adverse events of first-line anti-TB drugs under operational conditions in developing countries. Whether this is because of underreporting or underrecognition is not clear.

To respond to the specific questions proposed by Drs. Mohan and Sharma, all patients received standard short-course therapy if they had drug-sensitive organisms; therapy was prolonged in patients with drug-resistant organisms (1, 2, 5). There were few patients in our study with more serious forms. As stated, only 5% received directly observed therapy; these patients received therapy three times weekly in the continuation phase at doses recommended by the World Health Organization (1). As shown in Table 1 of our article (6), drug dosage was adjusted for body weight as recommended (1, 2, 5) and was not associated with toxicity. As demonstrated in Figures 2 and 3 of our article (6), almost all adverse events occurred in the first 2 months of therapy. As we reported, few of the patients with serious adverse events had comorbid conditions; for example, none of those patients with drug-induced hepatitis had viral hepatitis B or C, one had human immunodeficiency virus (HIV) infection, and none gave a history of alcohol abuse. Patients with HIV coinfection did not receive antiretroviral viral therapy in the first 2 months of TB therapy, when most adverse events occurred.

The greater rate of adverse events in our patient population than in Drs. Mohan and Sharma's experience is not explained by greater drug dosage, use of nonstandard regimens, comorbidities, or concomitant medications. The diverse ethnic background and the older age of our patients—which is atypical for developing countries—might have been factors because they were risk factors in our study (6). We believe that PZA toxicity may be underrecognized. A careful assessment of incidence of adverse events from PZA and other anti-TB agents under operational conditions in a developing country would be of great interest.

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World Trade Center Dust and Airway Reactivity

To the Editor:

Banauch and coworkers (1) reported that a subgroup of New York City's firefighters exposed to airborne particulates during and after the World Trade Center (WTC) collapse suffered

from persistent bronchial hyperreactivity and reactive airway dysfunction syndrome (RADS). Nemery (2), commenting on the article, argued that "RADS does *not* require a clinically severe inhalation injury necessitating medical care, let alone hospitalization."

We disagree with both of these contentions and suggest that the pulmonary condition associated with the WTC not be considered as RADS. This disagreement is especially valid because the WTC pulmonary condition does not fulfill the clinical criteria of RADS, an acute asthma syndrome that develops after a single high-level exposure to an irritant gas, fume, or vapor (3). The asthma symptoms develop acutely and always within 24 hours. The exposure is of such magnitude that there is almost always a need for acute medical attention. Furthermore, a dust exposure (e.g., pulverized cement, gypsum, concrete aggregate, ceiling tiles, and wall board or high concentrations of particulate matter) has not been previously reported to cause RADS. Much of the WTC debris was of a particle size too large to be inhaled into the lungs.

Perhaps a better term to describe the WTC pulmonary condition is "nonallergic asthma." This general term also could encompass conditions such as RADS, irritant-induced asthma, and perhaps even noneosinophilic asthma (4).

An interesting observation is the high alkalinity of the dust. Perhaps there is a more effective human response to an acid inhalation challenge (e.g., from acid air pollutants). The human defense against an alkaline inhalation challenge may not be as efficient and perhaps more detrimental. A better understanding of the airway responses to acid and alkaline challenges seems to be an appropriate area for future research.

We are still left with the consideration that the exposure to the dust and debris from the WTC collapse caused asthma symptoms in only a susceptible subgroup of individuals. Although preexisting asthma in remission and asymptomatic airway hyperresponsiveness may explain some cases, there may be other factors characterizing greater host susceptibility (5, 6). The role of host susceptibility in nonallergic asthma syndromes is another area of necessary research.

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From the Authors:

We thank Drs. Truncale and Brooks for their interest in our article. Reactive airway dysfunction syndrome (RADS) (1) is a term first coined by Dr. Brooks and used to describe subjects with acute asthma syndrome that develops after a single high-level exposure to an irritant gas, fume, or vapor (2). Asthma symptoms develop acutely and always within 24 hours. We acknowledge that exposure to high concentrations of particulate matter has not been previously reported to cause RADS, but that does not mean it should not be included in this syndrome, because it definitely does meet the clinical criteria for this syndrome. In addition, the alkaline nature of World Trade Center (WTC) dust is now well documented; therefore, this dust definitely qualifies as an irritating substance (3).

In the long run, whether we choose to call this asthma, nonallergic asthma, or RADS is not important. What is important is that we acknowledge that particulate matter can cause this problem and that hyperreactivity was for the most part persistent in the severely exposed.

Dr. Truncale's point about the size of WTC dust particles is partially correct. Yes, the majority of dust particles were greater than 10 microns in diameter, but the dust burden was so high on Day 1 that substantial numbers of particles in the respirable range (< 3 microns in diameter) were present. In fact, we have demonstrated recovery of particles in the induced sputum of firefighters 6 months after exposure (4). Furthermore, the size distribution and content of these particles was very similar to WTC dust. This finding clearly indicates that WTC dust particles were inhaled into the small airways.

We agree that the role of host susceptibility in nonallergic asthma syndromes is a necessary area for future research. Why did asthma develop in some but not all WTC rescue workers? Clearly, the answer lies in understanding environmental–genetic interactions, and our cohort is large enough to study both the nature of these interactions and the effect of treatment. This is one of many reasons why long-term medical monitoring for those exposed to WTC dust is a necessary and important project.

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From the Authors:

Drs. Truncale and Brooks do not dispute the conclusions of Banauch and coworkers (1) and of my editorial (2) that a sizable proportion of firefighters had persistent symptomatic bronchial hyperresponsiveness as a result of exposure that occurred because of their work. They take issue with calling this condition "reactive airways dysfunction syndrome" (RADS).

Brooks and coworkers (3) proposed strict criteria for RADS. Regarding exposure, the sole criterion consisted of a single exposure to "a gas, smoke, fume, or vapor which was present in very high concentrations and had irritant qualities to its nature." The aerosol to which the firefighters were exposed fits this definition. Admittedly, much of the World Trade Center debris was too large to reach the lungs, but a fraction of the aerosol was undoubtedly respirable. Although an analysis of settled dust gives the impression that this respirable fraction was very small (4), the total amount of particulates inhaled during actual operations on the day of the collapse was presumably substantial. That dust exposure has not been previously reported to cause RADS is contradicted in the chapter on RADS, coauthored by Dr. Brooks, in the most authoritative textbook on occupational asthma: "The exposure is irritant in nature and generally a vapor or gas, but on occasion a high-level smoke or dust exposure may be responsible" (5). Banauch and coworkers (1) have confirmed this, perhaps especially for dust of high alkalinity.

The other criticism against our use of the term RADS is that the firefighters did not experience a clinically severe acute inhalation injury. However, it has never been established that this is a necessary condition for development of RADS. Truncale and Brooks imply this when they state that there is *almost* (my emphasis) always a need for acute medical attention. Furthermore, as pointed out in my editorial (2), two existing cohort studies on RADS suggest that the initial clinical condition need not be very serious to be followed by RADS. The findings of Banauch and coworkers strongly support this view (1). This is important not only for clinicians, but also for those studying the role of irritants in the pathogenesis of asthma.

This essentially semantic discussion shows that RADS is not a very good term. The phrase is too general—any asthma is characterized by dysfunction of (reactive) airways—and it fails to indicate the traumatic origin of the condition. "Nonallergic asthma" is not better, and I prefer the concept of irritant-induced asthma (6) because it points more clearly to the cause.

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Erratum: Inflammation, Thrombogenicity, Histamine, and Diesel Particles in Hamsters

To the Editor:

We wish to make a correction to our recently published article (1). The total cell count in the bronchoalveolar lavage was expressed as 10^3/ml instead of 10^4/ml. Therefore, the values reported in the text of the Results section and Table E1 of the online supplement should be multiplied by a factor 10. This correction does not change the conclusions of our study. We apologize for this error.

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