ducer and concentration of prostaglandin used.

The results presented here indicate that the state of hyporeactivity that develops in virus-infected mice is reversible and that by judicious selection of inducers and prostaglandins the response of hyporeactive mice could be restored to near normal levels. These results are significant in that they not only suggest that the therapeutic efficacy of inducers may be significantly enhanced by coadministration with prostaglandins but that the state of hyporeactivity that develops in virus infections or neoplastic diseases is probably mediated by a specific cellular event that is influenced in some fashion by prostaglandins. The possibility that the effect seen was due to modulation of cyclic nucleotide levels was considered. However, direct addition of dibutyryl adenosine 3′,5′-monophosphate, isoproterenol, theophylline, carbachol, or dibutyryl guanosine 3′,5′-monophosphate had no significant effect upon the responsiveness of cells from EMC- or SFV-infected mice or on the response of the intact animals themselves (*8*). These results suggest that prostaglandins restored the cellular interferon response through an unidentified process not involving cyclic nucleotides.

In further studies (*8*), mice infected with influenza A_2 or Friend leukemia viruses (*8*) developed an infection-induced state of hyporeactivity, and the interferon response of these animals was enhanced or restored by coadministration of prostaglandins with inducers. These results indicate that the restoration was a fairly general phenomenon. It was not restricted by the inducer or prostaglandin used, and the interferon response of mice infected with any of four viruses was enhanced. Enhancement was selective, however, in that the response of normal cells was generally not affected although hyporeactive cells were. At present, the mechanism by which hyporeactive cells are rendered more responsive is not fully understood, although it does not appear to be linked to overt manipulation of cellular macromolecular synthesis or increased uptake of inducer (*8*).

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Atmospheric Dust: Climatological Consequences

Idso and Brazel (*1*) conclude from various radiation measurements for a range of dust-loading events at Phoenix, Arizona, that a buildup of anthropogenically produced tropospheric aerosols must inexorably tend to warm the planet's surface. We question the conclusion for several reasons.

Some of the results raise the question of measurement error. For the dust event of 22 February 1977, figure 1C in (*1*) shows that the dust loading, as indicated by the ratio of diffuse to normal-incidence solar radiation, rose sharply at 1100 hours, remained high until 1300 hours, and did not return to the unperturbed level until 1400 hours. The infrared radiation from the sky (figure 1B) rose sharply at the same time, 1100 hours, but fell sharply to the unperturbed level by 1200 hours. On 15 April 1977, the infrared radiation from the sky lagged behind the dust loading.

Idso and Brazel's raw data do not imply a warming effect, at least in two of the three cases presented (22 February and 15 April). On those dates increased dust levels gave rise to a change in net all-wave radiation (darkened circles of figure 1) such that the net incoming radiation (incoming visible minus outgoing infrared) was reduced. This represents a local cooling effect at the ground—reduced incoming solar radiation more than outweighed the change in infrared radiation. The inference of a climatological warming rests on the argument that infrared changes are felt over the entire area ($4\pi R^2$) of the earth's surface, whereas the solar radiation, being effectively unidirectional, falls normally on the cross-sectional area (πR^2). This might be correct if the measurements were all taken with the sun in the zenith (that is, zenith angle $\theta = 0°$). They were in fact taken at zenith angles between 40° and 70°. Since incoming normal flux is proportional to $\cos\theta$, in the flat atmosphere approximation, measured solar radiation should be divided by 4 $\cos\theta$ to obtain an approximation for a global average.

A second critical aspect is the location on figure 2 in (*1*) of the point representing the present state of the atmosphere. It does not have to be moved far to the right to make it fall in a region where the curve of figure 2 is falling rather than rising, implying cooling with increasing dust. Idso and Brazel chose an initial point corresponding to clear conditions in Phoenix, but is this realistic for a real world that is about 50 percent cloud-covered, 80 percent ocean, and has a haze level that receives sizable contributions from sea salt, terpenes, and sulfates? In other words, the earth, in its entirety, may actually have an average diffuse/direct value nearer to 0.1, say, in which case addition of aerosols, according to Idso and Brazel's curve, would lead to cooling on a global scale.

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With respect to the first question raised by Herman *et al.*, the net solar and net all-wave radiation data of figure 1, A and B, were acquired at a site 4 km distant from the site of the diffuse and normal-incidence solar radiation measurements of figure 1C, and this spatial separation could conceivably create discrepancies. However, we see no discrepancy on 11 January or on 15 April. On 22 February there is an apparent discrepancy, but on closer examination we see that it is due to a faulty delineation of the dust event in the solar and all-wave radiation data. That is, in comparing all 3 days, it is evident that in our report the net solar and net all-wave radiation traces for 22 February are too flat over the midday period.

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When the proper reconstruction is done, the time boundaries for the dust event match in all parts of the figure. These adjustments also alter the magnitudes of the solar and thermal radiation perturbations for 22 February, and these alterations are incorporated into the new evaluation of the data described below.

Their second major point, concerning the factor cos θ, is correct. This adjustment should have been made in our report. Also, the original diffuse solar radiation data were not corrected for the portion of this radiation obscured by the shade band. When these changes are made, along with the changes for 22 February and some changes discussed below, our final result is such that the trace of our original figure 2 (not the individual data points) has its ordinate values reduced 60 percent; the abscissa values remain unchanged. Thus, the curve still starts at a diffuse/normal-incidence ratio of about 0.04, peaks at a ratio of about 0.1, and crosses to negative values at a ratio of 0.9. It remains conceptually unaltered.

The last major point of Herman *et al.* concerns the initial state of the earth's atmosphere. Before considering the earth as a whole, however, we must consider our data acquisition sites. For instance, the dust event on 11 January (figure 1C) was preceded by an initial diffuse/normal-incidence ratio of about 0.05, while on 22 February and 15 April the initial ratio was about 0.1. Since our other data allowed us to determine separately the dependence of solar and thermal radiation perturbations on this ratio from its base value of 0.04 to well past 0.1, we were able to deduce what changes in both solar and thermal radiation should have transpired on 22 February and 15 April in going from 0.04 to the initial value of 0.1. These increments were algebraically added to the measured radiation perturbations in deducing the revised form of figure 2 described above.

Finally, considering the earth itself, we did not deal in our report with clouds, nor do we know what effects variable dust concentrations beneath a cloud layer would have on the net radiation balance at the earth's surface. Assuming a null effect, we would have to admit a further 50 percent reduction in our calculated net climatological radiation balance. Again, however, it remains conceptually the same. We cannot make a precise estimate of the mean value of the diffuse/normal-incidence solar radiation ratio of the nonovercast portion of the world. However, this is a crucial point, for if the mean earth ratio were coincident with the value at which we observe the maximum alteration in net radiation, either addition or subtraction of particulates from the atmosphere would tend to initiate a cooling trend. Thus, the last point of Herman *et al.* is very well taken and indicates a need to experimentally determine this ratio in many different environments. We feel, however, that this ratio is less than 0.1, for we have measured much less than that at Phoenix, which has significant natural and anthropogenic aerosol pollution. Until a considerable body of new data indicates differently, we hold to our original assessment of the situation, that increased particulate pollution of earth's troposphere must tend to warm the planet's suface.

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Subfornical Organ: A Dipsogenic Site of Action of Angiotensin II

Several years ago it was proposed that intracranially injected material may not act at the site of injection but may, in fact, spread via the ventricles (*1*) or the cerebral vasculature (*2*) to then act at some distant locus. We have indicated that one target for the spread of dipsogenic agents is the subfornical organ (SFO) (*3*), a position supported in part because localized SFO lesions reduced drinking induced by intrahypothalamic angiotensin II (AII) (*4*). The report by Buggy *et al.* (*5*) attributed to us the view that "the subfornical organ (SFO) contains the exclusive receptors for angiotensin-induced drinking" (*5*, p. 72). Rather, we stated, "It is tempting to speculate that the central dipsogenic receptors for circulating angiotensin II are within the SFO" (*4*, p. 1174). Despite their inaccurate portrayal of our position, we believe that there is sufficient interest in determining central AII sites of action to warrant discussion of their findings and interpretations.

We consider first the SFO lesions made by Buggy *et al.* (*5*), who report that intracranial AII-induced drinking recovered after 70 to 100 percent destruction of SFO. The SFO consists of rostral, central, and caudal zones (*6*), each of which possesses ultrastructurally identifiable neuronal perikarya. Because detailed description of the extent of the lesions is not provided, it is difficult to determine to what "70 to 100 percent destruction," used to denote SFO lesions (*5*), refers. It is also difficult to ascertain what adjacent tissues (for example, choroid plexus basal laminae) were damaged by these lesions. Sparing of portions of the SFO might be responsible for the drinking after lesions observed by these authors, a point previously indicated (*4*, *7*). In our study (*4*), animals were classified in terms of neural damage rather than in terms of behavioral performance (*5*). We have found that animals sustaining less than thorough SFO lesions are not reduced in AII-induced drinking (*4*, *7*). The absence of permanent deficits in AII drinking after SFO lesions (*5*) may be attributable, in part, to the methods of producing or of evaluating lesions, or both (*8*).

It was suggested (*5*) that blockade of the interventricular foramen ipsilateral to intracranial injections of AII, and not ablation of the SFO, produced deficits in elicited drinking consequent to SFO lesions. While reduced drinking could occur if the ipsilateral interventricular foramen were completely occluded, Buggy *et al.* have not demonstrated the complete occlusion of the foramen. In their radioactive tracing experiments, in fact, the blockage was not complete (*5*). Thus, as compared to recovered lesioned animals, 11 percent of the 100- or 500-ng intraventricular dose of AII passed into subarachnoid space, presumably via ventricular diffusion (*9*). The threshold for lateral ventricular AII-induced drinking is less than 1 ng of AII (*10*). Since these authors classified animals in terms of the reduction in elicited drinking and not in terms of extent of SFO damage, the relative contribution of ventricular occlusion versus SFO damage to the observed deficits in AII drinking remains uncertain.

We think there is some question as to the definition of recovery (*5*) used. In the Pittsburgh data, there is a persistent deficit in elicited drinking at 8 days after the lesion. Although a slight increase relative to the initial deficit exists, this ignores the persistent deficit. Since control water intake values (for example, injection of vehicle after the lesion) are not reported, the drinking observed may reflect recovery of response to AII per se, neurological recovery from surgical trauma, or changes in performance to repeated injections of AII (*2*). The failure of complete recovery of 1.8 percent saline intake is especially noteworthy, as it is suggested that AII acts specifically to

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