

The Dust Settles on Water Vapor Feedback

Anthony D. Del Genio

Increasing concentrations of greenhouse gases such as carbon dioxide in the atmosphere are expected to alter global climate. The direct warming caused by greenhouse gas emissions is modest, but positive feedbacks in the climate system may amplify the effect. One such feedback is particularly important: Water vapor should almost double the sensitivity of climate to greenhouse gas increases (1). However, doubts about the sign and magnitude of the effect persist because of coarse model resolution and limited understanding of cloud processes that supply and deplete water vapor (2, 3).

To understand water vapor feedback, scientists have examined variations in current climate, for example, with the seasons or during El Niño–La Niña cycles. However, these heating variations are concentrated in specific regions, inducing shifts in the atmospheric circulation. This is quite unlike the greenhouse gas signal, which is global and has much less effect on the circulation. On page 727 of this issue, Soden *et al.* (4) study a climate perturbation that has more in common with greenhouse gas–related climate change. They demonstrate that the volcanic eruption of Mount Pinatubo in 1991 caused a positive water vapor feedback, and that a general circulation model (GCM) reproduces this global feedback accurately.

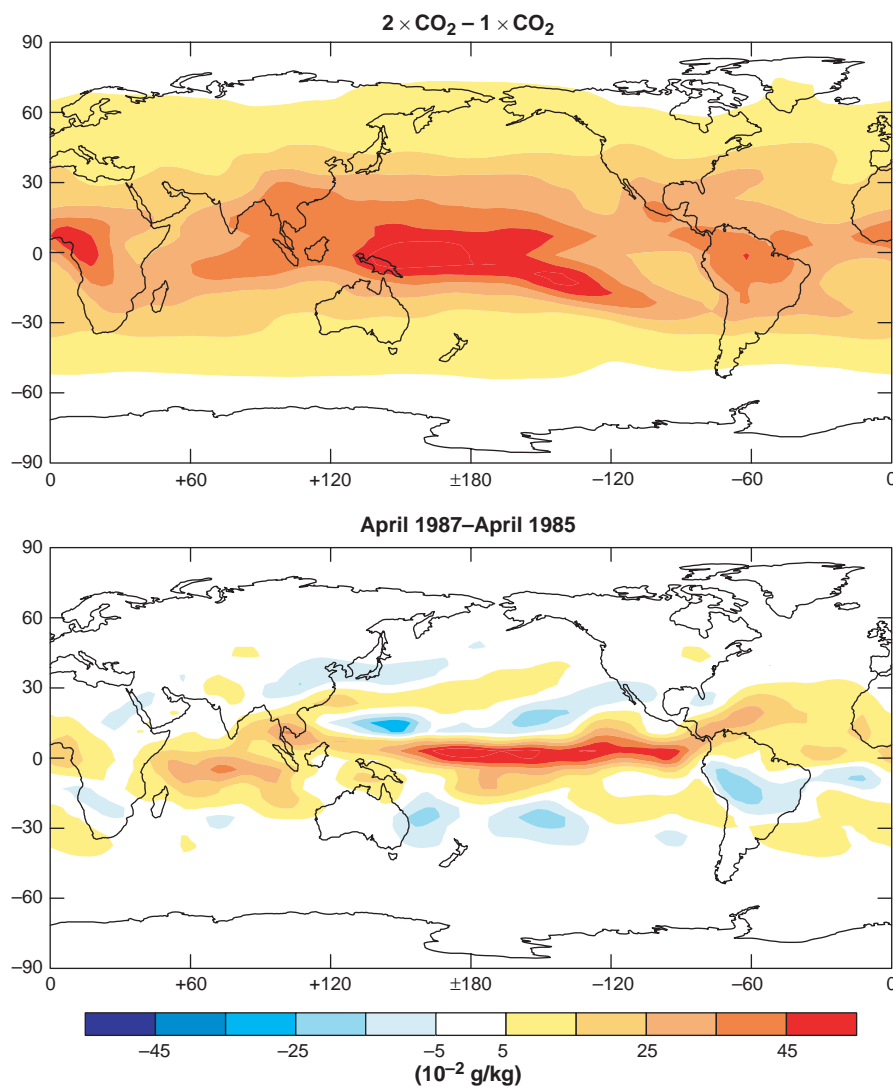
Water vapor concentrations vary widely throughout the atmosphere. The maximum (saturated) water vapor concentration in air increases sharply with temperature. Condensation, evaporation, and transport by winds also cause relative humidity (5) to vary considerably from place to place.

Water vapor absorbs heat emitted by the warm Earth surface and re-emits some of it to space. The global warming feedback is based on the fact that cold air radiates less than warm air; if water vapor increases and is distributed to higher, colder altitudes, less heat is radiated to space and thus climate warms. Soden *et al.* therefore examine both total column water vapor (the total amount of water in a column extending from the ground to the top of the atmosphere) and the relative humidity of a layer of the upper troposphere.

Water vapor responds differently to global and regional climate changes, making it difficult to separate water vapor feedback from other effects. In a representative simulation of greenhouse gas changes, atmospheric circulation and relative humidity changes very little, and water vapor primarily responds to temperature change. Water vapor increases everywhere, producing a strong positive feedback (see the figure, upper panel). In

contrast, during El Niño, heating is confined near the equator. Upward equatorial motions intensify and bring moist air up to high altitude whereas downward motions strengthen in the subtropics and bring dry air down from above. The resulting decrease in subtropical humidity (see the figure, lower panel) is sometimes cited as evidence of negative water vapor feedback (6).

The same model may thus show either decreases or increases in subtropical water vapor when subjected to different types of climate warming. Soden *et al.* clear the air by considering a better proxy for global climate change: a major volcanic eruption. Volcanic ash particles fall out within a few months of the eruption and barely affect climate, but volcanic aerosols (a concentrated sulfuric acid droplet haze)



Understanding water vapor feedback. GCMs consistently predict global increases in upper troposphere water vapor in response to a doubling of carbon dioxide (**upper panel**). The same models simulate a decrease in subtropical upper troposphere water vapor during El Niño (**lower panel**) because of strong regional circulation changes (11). Volcanic eruptions produce a water vapor signal that is opposite in sign but similar in spatial pattern to the carbon dioxide signal.

persist, blocking incoming sunlight. Stratospheric winds spread the aerosols globally, creating a climate perturbation resembling that due to greenhouse gas changes, yet opposite in sign. After a major volcanic eruption, global climate may cool for several years until the aerosols settle out of the stratosphere.

Soden *et al.* use satellite data to show that the peak cooling of ~0.5 K attributable to the Pinatubo eruption caused a ~3% decrease in column water vapor and a corresponding decrease in upper troposphere water vapor, indicating a strong positive water vapor feedback. In contrast, theories claiming that increased precipitation in a warmer climate will deplete water vapor (3) predict increased humidity in response to volcanic cooling.

Soden *et al.* also use satellite-retrieved volcanic aerosol properties in a GCM simulation of the decade of the 1990s. The model reproduces well the peak reduction in water vapor and the first several years of recovery to preeruption levels. The

amount of drying indicates that relative humidity remained almost constant after the eruption, just as it does in simulations of greenhouse gas increases. When Soden *et al.* artificially suppress the water vapor feedback on thermal radiation, the simulated Pinatubo cooling is only two-thirds of that observed.

A few cautionary notes are in order. Although the agreement shown by Soden *et al.* is good, volcanic eruptions are not perfect reverse proxies for greenhouse gas climate change. Volcanic aerosols affect incoming solar energy more than they do Earth's thermal radiation, whereas the reverse is true for greenhouse gases. Volcanic forcing decreases more from equator to pole than does greenhouse gas forcing. And both types of climate change reduce the rate at which temperature decreases with height from the surface to the upper troposphere, even though one is a global warming and the other a global cooling.

To refine estimates of the magnitude of water vapor feedback and determine the

sign of cloud feedback, model cloud physics and dynamics must be improved with the help of observed seasonal, interannual, and decadal climate variability (7–10). The results of Soden *et al.* suggest, however, that if humankind produces a substantial climate forcing, positive water vapor feedback will cause a substantial climate response.

References and Notes

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PERSPECTIVES: TRANSCRIPTION

Of Chips and ChIPs

M. Frances Shannon and Sudha Rao

Expression profiling using cDNA or oligonucleotide microarrays allows a global description of all the genes expressed in a cell in response to specific signals or at different stages of development [reviewed in (1, 2)]. Hierarchical clustering methods then allow the allocation of genes, coregulated in time or in response to specific treatments, into expression groupings called regulons (1, 2). A host of recent papers illustrate how data obtained from microarray expression profiling, combined with technologies such as the chromatin immunoprecipitation assay (ChIP), can be harnessed to explore transcriptional regulatory networks in cells. We know that at the simplest level, transcription of genes into messenger RNAs (mRNAs) is governed by transcription factors, which bind to cis-regulatory regions of the DNA in the vicinity of the target gene. There are, however, many more complexities that now can be explored across the whole genome. Do large coregulated groups of genes share cis-regulatory elements that bind to common transcription factors? Are coregulated genes located close to each other along the

linear DNA of the chromosome, or are they simply colocalized in the nucleus? Can we efficiently map global binding sites for transcription factors and chromatin proteins along the chromosomes *in vivo*?

Systematic nonbiased searching of upstream sequences of genes in a regulon for shared protein binding motifs can help to identify common control units. Tavazoie and colleagues (3) have applied this approach to microarray expression profiling data for yeast at different stages of the cell cycle. They discovered not only known protein binding motifs that regulate genes involved in the cell cycle, but also completely new cis-regulatory elements that are the binding sites for transcription factors (4). Many of these motifs were highly selective for the regulon in which they were identified and could also be assigned to groups of genes with related functions within that regulon. A similar approach has recently been applied to the identification of pharyngeal-specific genes in the nematode *Caenorhabditis elegans* (5). Expression profiling identified a large group of genes specifically expressed in the pharynx, a subset of which were further characterized and shown to contain binding sites for the transcription factor PHA-4. When PHA-4, a forkhead-type transcription factor, is deleted in worms, the pharynx does

not form (6). The combination of experimental microarray screening with computational approaches enables the generation of meaningful biological hypotheses that can then be tested experimentally.

Analysis of yeast microarray data has also been used to investigate whether coregulated genes are located close to each other in the yeast genome. Cohen and co-workers, using the yeast cell cycle microarray expression data set, have developed chromosome correlation maps (7). These maps display the patterns of coregulated genes in linear space along the chromosomes. Such chromosome correlation maps may represent for other organisms what were visualized many years ago as chromosome puffs (regions of high gene expression) on the polytene chromosomes of *Drosophila* salivary glands. Several regions of yeast chromosomes, some as large as 20 to 30 kb, have been found to contain large groups of coregulated genes. One simple explanation for coregulation of these genes is the presence of similar cis-regulatory elements in the promoters of the coregulated genes. This turns out not to be the case, however, for certain groups of coregulated genes in yeast. Areas of the chromosome that contain groups of coregulated genes are likely to represent regions of open chromatin structure. Thus, information on changes in gene transcription through changes in chromatin structure have to be combined with knowledge of common cis-regulatory elements to generate a more global view of gene expression patterns. These findings are perhaps not surprising for those who

The authors are in the Division of Molecular Bioscience, John Curtin School of Medical Research, Australian National University, Canberra, ACT, Australia. E-mail: frances.shannon@anu.edu.au