ATMOSPHERIC SCIENCE

A Matter of Humidity

How strong a part does water vapor play in global warming?

Andrew E. Dessler[1] and Steven C. Sherwood[2]

The water vapor feedback is the process whereby an initial warming of the planet, caused, for example, by an increase in atmospheric carbon dioxide, leads to an increase in the humidity of the atmosphere. Because water vapor is itself a greenhouse gas, this increase in humidity causes additional warming. The water vapor feedback has long been expected to strongly amplify climate changes because of the expectation that the atmosphere's relative humidity would remain roughly constant—meaning that the specific humidity would increase at the rate of the equilibrium vapor pressure, which rises rapidly with temperature. However, observational evidence has been harder to come by, and the effect has been controversial. Much of that controversy can now be laid to rest, thanks to new observations and better theoretical understanding.

In the 1990s, there was little observational or theoretical understanding of atmospheric humidity and how it varied with global climate. As a result, debate raged over whether the water vapor feedback would really occur, with some very influential proposals that it would not (*1*). In particular, many believed that atmospheric humidity and the water vapor feedback were controlled by processes—such as the details of cloud dynamics and microphysical processes—that are not sufficiently well understood and inadequately represented in climate models.

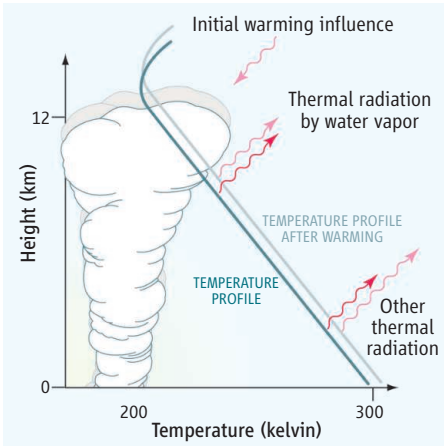
Successive reports from the Intergovernmental Panel on Climate Change (IPCC) have suggested increasing confidence in our understanding of the water vapor feedback, but they have remained cautious in defending its magnitude. However, recent advances have placed the traditional view of the water vapor feedback on a stronger footing than is widely appreciated.

The water vapor feedback mainly results from changes in humidity in the tropical upper troposphere (*2*), where temperatures are far below that of the surface and the vapor is above most of the cloud cover. The distribution of humidity in this region is well reproduced by "large-scale control" models, in which air leaves stormy regions in a saturated condition, but with negligible ice or liquid content. Water vapor is thereafter transported by the large-scale circulation, which conserves the specific humidity (the ratio of the mass of water vapor to the total mass in a unit volume of air), except during subsequent saturation events, when loss of water occurs instantaneously to prevent supersaturation. Despite the simplicity of this idea, which entirely neglects detailed microphysics and other small-scale processes, such models accurately reproduce the observed water vapor distribution for the mid and upper troposphere (*3*, *4*). One recent study (*5*) estimated the uncertainty in the water vapor feedback associated with microscale process behavior at less than 5%, as a result of the overwhelming control of humidity by the large-scale wind field.

Thus, the water vapor feedback is essentially controlled by the large-scale dynamics and the saturation specific humidity in the outflow of the tropical deep convective systems. Convective outflow temperature should, on average, warm along with the mean atmosphere, thus producing the feedback (*6*, *7*).

Given these considerations, there are good reasons to expect global climate models to accurately simulate the water vapor feedback: The large-scale wind and temperature fields that mainly control the humidity are explicitly calculated from the basic fluid equations, unlike small-scale processes that must be represented by crude parameterizations.

Although the water vapor feedback is strong in all global climate models, its magnitude varies somewhat due to differences among the models in the amount of upper tropospheric warming (and hence the increase in specific humidity) per unit of surface warming. The spread among models in the water vapor feedback is, however, largely compensated by an opposite spread in the "lapse-rate feedback," a negative feedback that occurs because a warmer atmosphere radiates more power to space, thereby reducing net surface warming. As a result, the sum of the two feedbacks is insensitive to errors in predicted warming of the upper troposphere, and to quantify the sum accurately, one only needs to know how relative humidity (the ratio of specific humidity to that in a saturated condition) changes as the climate warms. The sum of the feedbacks is also smaller than the water vapor feedback—about half the magnitude—and more consistent among climate models (*8*), because no model predicts substantial and systematic changes in relative humidity.



Schematic of the water vapor feedback. Because thermal emission to space by water vapor does not increase, more warming is needed to balance a given energy input.

Despite these advances, observational evidence is crucial to determine whether models really capture the important aspects of the water vapor feedback. Such evidence is now available from satellite observations of the response of atmospheric humidity (and its impacts on planetary radiation) to a number of climate variations. Observations during the seasonal cycle, the El Niño cycle, the sudden cooling after the 1991 eruption of Mount Pinatubo, and the gradual warming over recent decades all show atmospheric humidity changing in ways consistent with those predicted by global climate models, implying a strong and positive water vapor feedback (*9*–*13*). A strong and positive water vapor feedback is also necessary for models to explain the magnitude of past natural climate variations (*14*).

Both observations and models suggest that the magnitude of the water vapor feedback is similar to that obtained if the atmosphere held relative humidity constant everywhere. This should not be taken to mean that relative humidity will remain exactly the same everywhere. Regional variations of relative humidity are seen in all observed climate variations and in model simulations of future climate, but have a negligible net impact on the global feedback (*12*).

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Thus, although there continues to be some uncertainty about its exact magnitude, the water vapor feedback is virtually certain to be strongly positive, with most evidence supporting a magnitude of 1.5 to 2.0 W/m^2/K, sufficient to roughly double the warming that would otherwise occur. To date, observational records are too short to pin down the exact size of the water vapor feedback in response to long-term warming from anthropogenic greenhouse gases. However, it seems unlikely that the water vapor feedback in response to long-term warming would behave differently from that observed in response to shorter-time scale climate variations. There remain many uncertainties in our simulations of the climate, but evidence for the water vapor feedback—and the large future climate warming it implies—is now strong.

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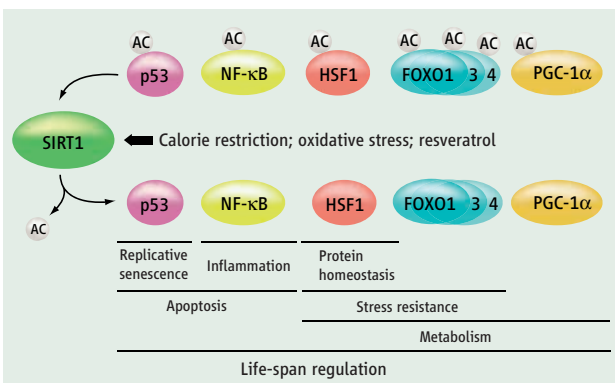
CELL BIOLOGY

Stress Response and Aging

Transcriptional regulators that respond to stress also influence life span.

Laura R. Saunders and Eric Verdin

Exposure to a variety of mild stressors, including calorie restriction, thermal stress, or hyperbaric oxygen, induces an adaptive biological response that increases eukaryotic life span (*1*). There are also a variety of mutations associated with both increased resistance to stress and increased longevity, such as those associated with altered insulin/IGF1 (insulin-like growth factor 1) signaling in the nematode *Caenorhabditis elegans* (*2*). Adaptive responses to stressors are mediated by transcription factors that regulate both stress response and life span. On page 1063 of this issue, Westerheide *et al.* (*3*) connect two additional transcriptional regulators to stress responses and longevity. The results support the idea that low levels of stressors influence life span and provide additional potential molecular targets that can be further manipulated experimentally or therapeutically.



Handling stress. SIRT1 is a deacetylase that is activated by a variety of stressors and targets transcriptional regulators including p53, NF-κB, HSF1, FOXO1, 3, and 4, and PGC-1α. These factors then control adaptive responses that modulate life span. AC, acetyl group.

Westerheide *et al.* demonstrate that the activity of a transcription factor called heat shock factor 1 (HSF1) is regulated by the enzyme sirtuin 1 (SIRT1). HSF1 exists as a monomer in unstressed mammalian cells. In response to a variety of stresses—including heat shock, hypoxia, misfolded proteins, free radicals, and adenosine triphosphate depletion—HSF1 trimerizes, translocates to the nucleus, becomes phosphorylated, and binds to regulatory elements (promoters) of genes that encode heat shock proteins (*4*). Heat shock proteins such as Hsp70 serve as chaperones and proteases that resolve damaged, misfolded, and aggregated proteins.

SIRT1, a mammalian ortholog of the yeast transcriptional regulator Sir2, is a stress-activated nicotinamide adenine dinucleotide (NAD^+)–dependent protein deacetylase that regulates cell survival, replicative senescence, inflammation, and metabolism through the deacetylation of histones (the major protein components of chromatin) and other cellular factors including the transcription factors p53, NF-κB, and FOXO1, 3, and 4, and the transcriptional regulator PGC-1α (*5*) (see the figure). Calorie restriction extends life span in part by increasing SIRT1 expression, and in yeast, worms, and fruit flies, the lack of Sir2 abrogates the effects of calorie restriction on life span (*6*). Similarly, mice lacking SIRT1 do not show some of the beneficial effects of calorie restriction related to longevity (*7*, *8*). The enzymatic activity of SIRT1 is activated by resveratrol, a polyphenol produced by plants under stress. Resveratrol extends the life-span of yeast, worms, and flies only when Sir2 is present (*9*).

Westerheide *et al.* show that in mammalian cells, SIRT1 directly deacetylates HSF1 and thereby regulates the heat shock response. The effect of SIRT1 on HSF1 appears to be dynamically regulated. In response to stresses, including heat shock, HSF1 is acetylated by the histone acetyltransferase p300, a modification that is thought to function as an "off" signal by triggering the dissociation of HSF1 from its target gene. Inhibiting SIRT1 expression via small interfering RNA prevents HSF1 from binding to the *hsp70* promoter and suppresses transcription of the gene when cells are exposed to heat shock. Conversely, Westerheide *et al.* observed that SIRT1 activation by resveratrol or SIRT1 overexpression in cells decreases HSF1 acetylation, prolongs HSF1 binding to target promoters, and enhances the heat shock response.

HSF1 is acetylated on at least nine lysine residues. One acetylated residue, Lys^{80}, controls HSF binding to DNA. Thus, acetylation of HSF1 at Lys^{80} may cause the release of

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