ing for the answers in Madagascar. It is one of the many problems that can only be resolved by further fieldwork and research. And it is one of the reasons why the discoveries of Late Cretaceous dinosaurs by the Sereno team in northern Africa will continue to attract international attention.

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Stratospheric Control of Climate

Alan Robock

From 1905 to 1952, Charles Greeley Abbott directed the measurement of the solar constant and its relation to sunspots from his office in the tower of the Smithsonian Building, convinced that these solar variations held the clue to weather and climate forecasts (*1*). Since then, the lack of a convincing mechanism has hampered acceptance of such a connection. Now, as reported in this issue, Haigh (*2*) has used a climate model simulation to show a mechanism whereby the stratosphere is changed by the sun, which in turn drives the tropospheric climate.

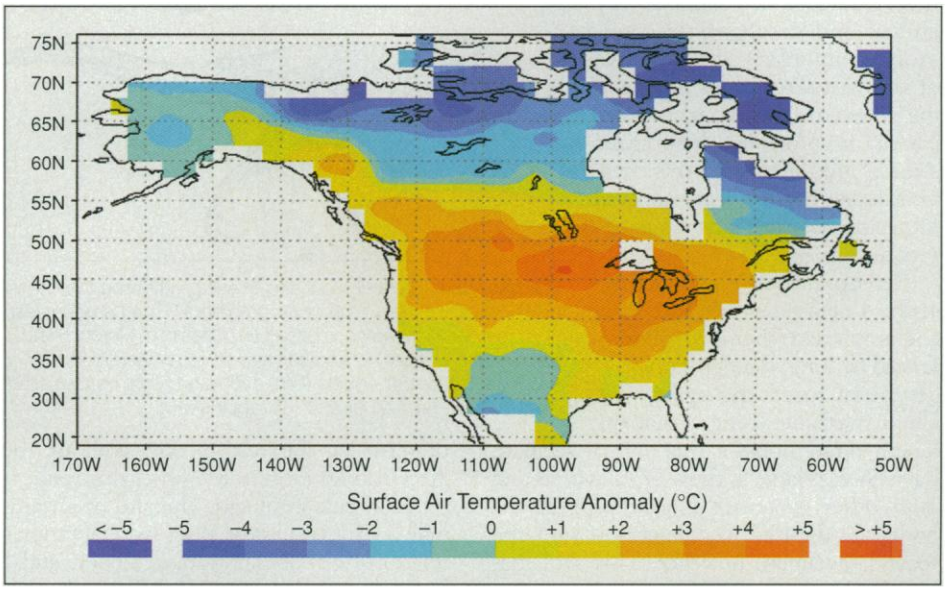
Interest in possible solar-weather variations grew after Eddy (*3*) described the Maunder Minimum in sunspots in the 1600s and suggested that it represented reduced solar insolation and was responsible for the Little Ice Age. He suggested that the envelope of the sunspot number was most representative of the solar influence on climate, but my subsequent work (*4*) showed that the decadal-scale variations produced by climate models that this implied were not representative of recent climate change of the past several centuries. Whereas all these earlier studies emphasized surface climate variations, van Loon and Labitzke, in a long series of observational papers (*5*), found climate variations at the surface and in the upper atmosphere with significant variations related to the sunspot number. They found that some of the signals were stronger when modulated by the quasi-biennial oscillation (QBO) in equatorial stratospheric wind.

Variations of the total solar irradiance over an 11-year sunspot cycle are only about 0.1%, but most of this variation is in the ultraviolet (UV) wavelengths, which are absorbed by the ozone in the stratosphere. Recognizing this, Kodera *et al.* (*6*) and Rind and Balachandran (*7*) used general circulation models (GCMs) of the climate system in which the solar variations in the UV were felt in the stratosphere, producing gradients of heating and inducing a response in the tropospheric circulation with much larger indirect effects than what would be produced by the direct effects of spectrally averaged heating. Still, the results did not correspond closely to the observed solar-related variations, and they had to use unrealistically large solar UV variations to get a significant response.

Now Haigh (*2*) has conducted a simulation that produces realistic tropospheric response to solar variations, by recognizing that stratospheric ozone also varies with the sun and by including ozone variations in her model forcing. In these perpetual January GCM simulations, the stratosphere is heated because there is excess solar UV radiation, and there is more ozone to absorb this radiation. These combined effects produce changes in the tropical Hadley cell and in mid-latitude storm tracks. The resulting changes are still not quite as large as the observed changes, and she has not tested the relation to QBO variations, as this model keeps the stratospheric winds in the easterly phase. More detailed calculations should be done with a climate model that goes through the seasonal cycle and has more resolution in the stratosphere.

These results resemble the recent work of Kodera (*8*) and Graf *et al.* (*9*), which show a large winter dynamic response of the climate system to explosive volcanic eruptions, another response produced by stratospheric heating. In the case of volcanic aerosols, the direct heating of the stratosphere attributable to long-wave absorption by the volcanic aerosols produces a temperature gradient anomaly and a circulation response seen as winter warming of the Northern Hemisphere continents. The Graf *et al.* simulations were also conducted with a perpetual January GCM and are now being repeated with a more detailed GCM going through the entire seasonal cycle.

A large body of work (*10*) suggests that surface temperature and precipitation anomalies can be predicted for months to a year in advance by taking account of the state of the sea surface temperature (SST) patterns in the tropical Pacific Ocean, which are most dramatic during an El Niño. During the large 1982–1983 El Niño, large surface temperature variations over North America, however, have recently been shown to be a result of the circulation anomalies induced by the 1982 El Chichón volcanic eruption, and not the SST-induced changes. By comparing climate



Surface air temperature anomalies over North America for 1982–1983. The observations are obtained from Schemm *et al.* (*17*). The contour interval is 1°C. (A version of this figure that includes observations and simulations for 1982–1983 and 1986–1987 is available on the World Wide Web at http://www.meto.umd.edu/~alan/fig10c.GIF)

The author is in the Department of Meteorology, University of Maryland, College Park, MD 20742, USA. E-mail: alan@atmos.umd.edu

model simulations from the Atmospheric Model Intercomparison Project (*11*), in which GCMs were forced only with SST variations, with the observations, Mao and Robock (*12*) showed that although the winter pattern of 1986–1987 was well simulated by an average of the eight best simulations during a period without volcanic aerosols, the 1982–1983 pattern resembles the winter warming found in the volcanic GCM simulations and in observations (*13*) and does not resemble the El Niño pattern (see figure). Kirchner and Graf (*14*) have shown that it is possible to distinguish the signals of SST and volcanic forcings and that both are important in Northern Hemisphere winter climate variations.

We are entering a new period for stratosphere-troposphere exchange (*15*), with enhanced emphasis on the stratosphere by the World Climate Research Programme "Stratospheric Processes and Their Role in Climate" project (*16*). The problems are fascinating, challenging, and complex and involve understanding the simultaneous interactions of atmospheric dynamics, radiative physics, aerosols, and chemistry. Volcanic eruptions also affect ozone concentrations by providing aerosol surfaces for heterogeneous chemical reactions with anthropogenic chlorine compounds. GCMs are just now becoming available with enhanced stratospheric resolution and explicit consideration of the radiative effects of aerosols and the chemical variations induced by changing UV radiation, anthropogenic chemicals, and aerosols. With these new tools and our enhanced awareness of the potentially large impacts of stratospheric variations on the tropospheric climate, we will soon have a much richer understanding of seasonal and interannual climate variations and their causes and improved predictive skill to enhance that already existing from SST variations.

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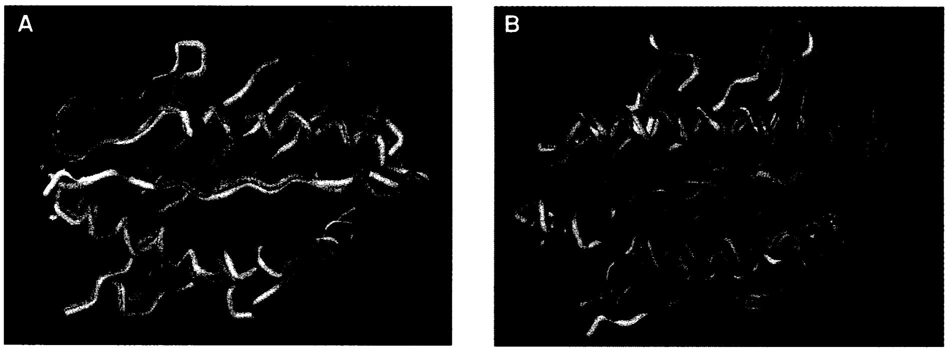
Another Twist to MHC-Peptide Recognition

Ian A. Wilson

The immune system cuts foreign invaders into little pieces, which are then displayed on the surface of cells. There, bound to one of two proteins—major histocompatibility complex (MHC) class I or class II—these foreign peptides trigger the defensive reactions of the host. MHC class I receives its peptide pieces inside the cell, derived from an intruder that has taken up residence there; but class II is filled with molecular bits of invaders from outside the cell. Before being filled with their real cargo, the binding groove of MHC class II molecules is occupied by a "dummy" peptide, CLIP. Inside a vesicle that contains the proteolyzed remains of foreign invaders from outside the cell, the acidic conditions promote dissociation of this place-holding molecule and insertion of a foreign one. A report in this week's issue of *Science* yields clues about how pH may control replacement of CLIP with foreign peptides (*1*).

Structural analyses of single peptides bound to class II molecules from human (*2*, *3*) and mouse (*1*) have allowed us to see exactly what the peptide looks like as it sits in its MHC groove (*4*). In MHC class II molecules, peptide antigens—13 to 25 residues long (*5*)—are embedded in the MHC binding groove but spill out over the ends of the binding site (*1–3*). The central nine residues are forced into a supertwisted ribbon structure that closely resembles a polyproline II–type conformation. On the walls of the groove, the "peptide-like" side chains of four conserved Asp and Gln residues of human (DR) and mouse (I-E) class II molecules hydrogen bond similarly with each peptide antigen backbone in a pseudo–β-sheet–like interaction that causes the peptide to writhe and twist along the groove into the regular, shallow polyproline II spiral. Even though the binding sites are open at either end, this backbone interaction imposes



Foreign peptides in their places: Signals for immune attack. (**A**) Class II MHC human and murine molecules with their bound peptides. Superposition of I-E^k (*3*) (white and yellow), HLA-DR1 (*1*) [1dlh (blue and cyan)], and HLA-DR3 (*2*) (magenta). The I-E^k peptides are covalently linked to the MHC molecule in a construction that allows coexpression (*16*). (**B**) Class I MHC molecules and their bound peptides. The molecules superimposed include five peptide complexes of HLA-A0201 (*17*) [1hhg (red), 1hhh (green), ihhi (blue), 1hhj (cyan), and 1hhk (magenta)], HLA-Aw68 (*18*) [1tmc (yellow)], and three complexes of murine H-2K^b (*19, 20*) [kbo (white), kbs (orange), and kbv (red)]. [Figure by R. Stanfield; coordinates provided by D. Fremont (I-E^k), T. Jardetzky, P. Ghosh, and D. Wiley (HLA-DR1 and HLA-DR3), and the published class I structures (*17–20*)]

The author is in the Department of Molecular Biology, Scripps Research Institute, 10666 North Torrey Pines Road, La Jolla, CA 92037, USA. E-mail: wilson@scripps.edu