barrier control could make a better device — until now. Using palladium for the metal connecting wire and relatively wide-diameter (about 3 nm) nanotubes, Javey *et al.*[1] have achieved this feat, eliminating the Schottky barrier and observing ballistic transmission of electrons through the device. Both choices, of palladium and of wide-diameter nanotubes, raise intriguing and as yet unanswered questions. The authors note that there is no obvious reason why palladium should give a smaller barrier than, say, platinum; palladium is distinguished primarily by its ability to stick well to carbon nanotubes. So why is its electrical behaviour different? Does palladium react chemically with the carbon tube? Does it differ from platinum or gold in barrier height, or in some other way? Can we rule out the existence of a Schottky barrier so thin as to be virtually transparent to electrons[5]? And can the barrier be similarly eliminated in narrower tubes, whose electrical properties are more favourable for practical devices?

In any case, Javey and colleagues' device brings us much closer to the fundamental limits of conductance; it is capable of carrying unprecedented currents at modest voltage. The evidence is compelling that this improvement comes from effectively eliminating the Schottky barrier. And although there is as yet no answer to why the barrier disappears, this work raises hope that control of the Schottky barrier will become integral to future nanotube-device technology. ■

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Atmospheric science

African dust in Florida clouds

Owen B. Toon

Satellites and numerical models now track the intercontinental transport of airborne particles. Better knowledge of cloud physics will be necessary to gauge the effects on clouds and rainfall patterns.

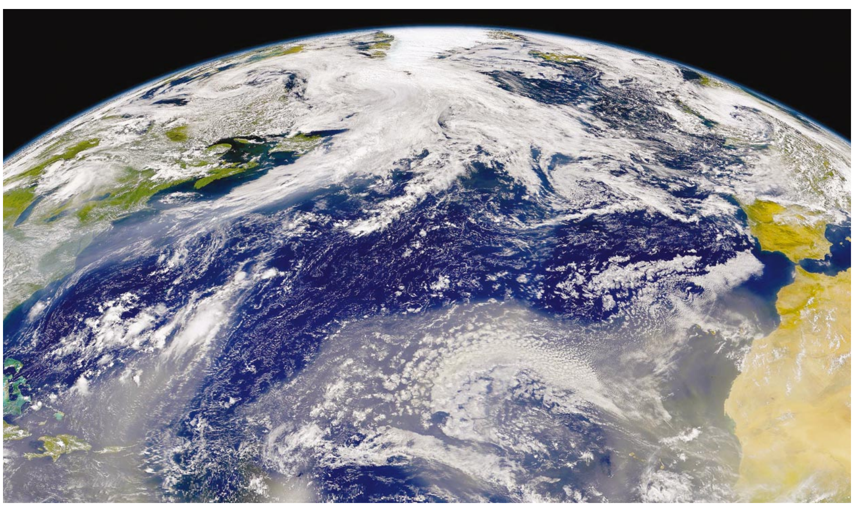
If you live near a desert you expect to get dusty every now and again. Strong winds bounce sand grains across the desert surface, which in turn blast micrometre-sized particles into the air, where they blow downwind and eventually land on you. Desert dust might not seem to be a problem in a lush tropical environment such as Florida, but as our view of Earth has improved it has become clear that dust is blowing everywhere and seeping into everything. As they report in *Geophysical Research Letters*, Sassen *et al.*[1] and DeMott *et al.*[2] show that dust from the Sahara Desert in Africa can have an impact on clouds over Florida (Fig. 1). Sassen[3] previously found that dust from Asia was affecting clouds over the western United States.

Advances in satellite remote sensing and in numerical modelling of aerosol behaviour now make possible the daily production of images and forecasts for the locations of dense dust plumes, as well as of clouds of smoke and sulphates[4]. So it was that in April 2001, awakening to visibly dirty air in Boulder, Colorado, a quick glance at the Internet[4] showed me that local pollution was not to blame, but a dust storm in China a few days earlier. Smoke plumes from far-distant fires are also commonly observed and can now be traced back to their sources, often on the other side of the world.

Intercontinental transport of dust has several implications, some of which have been recognized only recently. Dust scatters and absorbs sunlight, as well as infrared light radiated by the Earth, which alters the radiation budget and is a major factor in studies of climate and climate change[5]. It is difficult to regulate air-pollution standards when they are violated by dust storms occurring halfway around the world[6]. In different contexts, wind-blown dust is a significant source of minerals for oceanic plankton, and it may be a way in which biological debris is transported over intercontinental scales. On a more local scale, a dust storm in Iran caused the collapse of a military venture to rescue the US embassy hostages in 1980, dooming the re-election prospects of President Jimmy Carter, and such storms severely affected operations in the recent conflicts in Afghanistan and Iraq.

It has long been clear that rainfall is the principal mechanism for cleansing the sky of dust, but only recently has the impact of dust on clouds been appreciated[7]. Sassen *et al.*[1] and DeMott *et al.*[2] now show that such effects may be widespread and not restricted to regions near deserts.

Dust may affect clouds in two ways. All water droplets start off by forming on pre-existing particles. As the number of particles increases, for instance due to a dust storm, the number of cloud droplets may increase. If there are more cloud droplets, the droplets will be smaller because the mass of condensing water is usually fixed by air motions and ambient humidity. Smaller cloud droplets make for a greater surface area, and hence brighter clouds, a consequence that was the first indirect effect of aerosols on climate to be recognized, and that has been the object of intense study over the past decade. A less well-studied phenomenon is that smaller droplets are also much less likely to collide with each other and create precipitation. Although the net flow of water through the atmosphere may be set by the rate of evaporation from the oceans, the locations of



SEAWIFS PROJECT, NASA/GSFC, ORBIMAGE

Figure 1 **Dust-up. In this image, produced with data from the SeaWiFS satellite, Saharan dust appears as a brownish haze spread across the Atlantic Ocean from Africa to the Caribbean, mingling with various cloud systems along the way. Such cross-Atlantic transport of dust is continually observed by satellites.**

rainfall can be shifted by such induced changes in precipitation.

By acting as nuclei for triggering ice formation, dust particles can also affect clouds by causing the water droplets to freeze at higher temperatures than expected. Pure water can become supercooled to temperatures near −40 °C. But the data of Sassen *et al.*[1] and DeMott *et al.*[2] suggest that, in clouds over Florida, Saharan dust is causing water to freeze at temperatures between −5 °C and −8 °C. Ice crystals have a lower saturation vapour pressure than water droplets, so ice particles can rapidly reach large sizes in a cloud that contains both crystals and droplets. While falling, the ice particles can grow by colliding with the water droplets and induce rainfall.

Dust may thus be triggering precipitation in low-altitude clouds that otherwise would have been too warm to have produced rain, or be triggering rain at lower levels in convective clouds that otherwise would have not produced rain until reaching much higher altitudes, where it is colder. Just by acting as an abundant ice nucleus, the dust may be making high-altitude ice clouds more common. Dust may therefore inhibit precipitation by making more and smaller droplets, or enhance it by adding ice particles to warm clouds. Indeed, satellite studies over the Atlantic Ocean[8] hint that both processes may occur, with dust reducing precipitation in low-level clouds and enhancing it in high-altitude clouds.

The complexity of clouds and their interactions with aerosols provide a daunting challenge in climate prediction. Global-scale climate models, upon which we rely for climate forecasts, cannot simulate cloud physics because they lack adequate spatial resolution. Hence our climate forecasts depend upon parametrizations of clouds. Observations such as those by Sassen *et al.*[1] and DeMott *et al.*[2] provide the kind of insight that will spur the further development of such work. Several large field programmes are planned for the next few years, and their objectives include a better understanding of clouds and their interactions with aerosols. Ever-increasing computer resources mean that cloud-resolving models are becoming much more sophisticated and, finally, are beginning to incorporate fairly complete cloud microphysics and atmospheric dynamics on the scale of clouds. But we have a long way to go before we can provide coherent links between a dust storm in the Sahara, a cloud over Miami and the physical and chemical interactions of water on a dust grain within that cloud. ■

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Signal transduction

Life on Mars, cellularly speaking

Pier Paolo Di Fiore

A key molecular switch, known as the Ha-Ras protein, is active not only at a cell's outer membrane but also on intracellular membranes. This surprising discovery hints at unsuspected complexity in cellular signalling.

Your cells are constantly talking to one another. This dialogue relies predominantly on the emission of signals by one cell and their detection by receptors at the outer (plasma) membrane of another; the receptors then send a message inwards towards the nucleus. Inside the cell, proteins of the Ras family are key binary switches that are turned from 'off' to 'on' by these receptor-transduced incoming stimuli, and propagate signals further downstream[1]. A long-held tenet is that Ras proteins work only at the plasma membrane. But we are now invited to think differently: on page 694 of this issue, Bivona and co-workers[2] confirm that one Ras protein, Ha-Ras, is also activated on the Golgi apparatus inside the cell, and they show that it is not only biologically active there, but is the sole effective form of Ras in certain settings. The Golgi is involved in protein secretion, and thereby controls how signals get out of the cell. The discovery that — through Ras — it also regulates how they get in is, from a signalling viewpoint, as unexpected as life on Mars.

Previous observations had in fact already challenged the plasma-membrane-centric view of Ras. For instance, newly synthesized Ras proteins are transiently present on intracellular membranes (those of the Golgi apparatus and another cellular compartment, the endoplasmic reticulum, or ER), where the proteins are modified. Moreover, after all modifications have occurred, K-Ras, another family member, is found mainly at the plasma membrane, but Ha-Ras is present at both this and internal membranes[3]. Finally, active receptors in the plasma membrane can stimulate Ha-Ras on intracellular membranes[4]. These findings raised the question of how a signal can reach this Ha-Ras from surface receptors — and whether the process is biologically relevant.

Now we have answers to these questions. Bivona *et al.*[2] have uncovered a previously unknown signalling pathway that causes both the activation of Ha-Ras on the Golgi apparatus and its deactivation at the plasma membrane (Fig. 1). Ras proteins are 'on' when they are bound to the small molecule guanosine triphosphate (GTP), and 'off' when bound to guanosine diphosphate (GDP). The critical event in the pathway described by Bivona *et al.* is an increase in the intracellular level of calcium ions, which causes a Ras guanine-nucleotide-exchange factor (GEF), known as RasGRP1, to move to the Golgi, and a Ras GTPase-activating protein (GAP), CAPRI, to move to the plasma membrane.

GEFs activate Ras, by catalysing the replacement of bound GDP with GTP. GAPs, by contrast, turn Ras off, by enhancing the breakdown of GTP to GDP. So these findings hint at the exciting possibility that the relative levels of RasGRP1 and CAPRI determine whether Ras signals emanate primarily from the plasma membrane or from intracellular membranes (Fig. 1). The duration of the calcium increases may also affect the final outcome. Bivona *et al.* predict that, in cells displaying persistent intracellular calcium increases, Ras signalling occurs mainly from the Golgi. But when the rises are short-lived, signalling might be mostly from the plasma membrane.

The idea that the plasma membrane is not the only signalling compartment in the cell is not new[5,6]. In particular, cellular compartments called endosomes have been proposed to have this role. The problem is that endosomes originate from the plasma membrane, making it difficult to distinguish their role in signalling from that of the plasma membrane. By contrast, Bivona and co-workers provide evidence for several biologically important roles for Golgi-activated Ras: it induces neuronal cells (PC12 cells) to differentiate and fibroblast cells to become malignant, and it makes rat intestinal epithelial cells resistant to ionizing radiation. Moreover, signalling through the T-cell antigen receptor — a key protein in the immune system — activates Ras only on the Golgi, although it is also present at the plasma membrane.

The study of signalling has traditionally involved asking 'what?' and 'when?'; it is now increasingly becoming a matter of 'where?'. But we should also ask 'why?'. Why should