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Climate change

The cloud conundrum

Joyce E. Penner

One of the great uncertainties in projecting global warming is accounting for the effects of small particles in Earth's atmosphere. Progress is nonetheless being made with this fiendishly complex problem.

Generally accepted climate projections for the year 2100, compared with today, predict a global average temperature increase ranging from around 5.8 °C to a more benign, but still worrisome, 1.4 °C. Which of these futures awaits us depends, in part, on aerosols — tiny particles in the atmosphere — and their effect on clouds. Ackerman *et al.* (page 1014 of this issue)[1] conclude that the role of these particles in increasing the water content of clouds, and so cloud reflectivity, is smaller than previously thought.

As a consequence of air pollution, the mass of small particles in the atmosphere is about 40% larger than it would be in a pristine atmosphere[2]. These particles mainly reflect solar radiation, a direct effect on climate that has received considerable attention over the past 15 years. But when clouds form, these tiny particles also act as seeds for water condensation. When a cloud forms in a region containing a large number of particles, it will have higher concentrations of smaller droplets if the liquid water in the cloud is constant. The smaller droplets have a higher surface area; so the cloud is brighter and able to reflect more solar radiation, thereby cooling the climate. This is the so-called first indirect effect of aerosols on clouds.

But climate scientists have also explored the consequences of smaller droplets on precipitation. Smaller droplets are less likely to collide with each other and form precipitation. This change in 'precipitation efficiency' has been termed the second indirect effect, and has been calculated to increase both the total cloud cover and the total amount of liquid that is held within clouds. These two effects increase the total reflected solar radiation even more and lead to a conundrum. Because some climate models are highly sensitive to this change, they predict a total cooling effect that is larger than the warming by greenhouse gases: but the net effect of greenhouse-gas and aerosol changes over the past 100 years is observed to be a warming.

The conundrum may be solved in one of three ways. Some particles, especially the 'black carbon' produced by biomass and fossil-fuel burning, can absorb radiation rather than reflect it, and so counteract cooling. Perhaps this effect has been underestimated: if these particles are much more abundant than thought[3], or if they cause more absorption of solar radiation after deposition on snow[4] (which is normally highly reflective), they may be counteracting the effects of particles on precipitation efficiency to a greater extent than expected.

Or perhaps the effects of particles on 'ice clouds' — clouds high in the atmosphere — have been underestimated. Because high clouds tend to absorb more energy in the form of thermal radiation than they reflect in the form of solar radiation, an increase in ice-cloud amount and particle number would warm the climate. Almost no work has addressed the effect of air pollution on ice clouds; but if increased pollution also causes increases in ice particles within high clouds, it might balance the effect of increased air pollution on low, warm clouds.

A third possibility is that those climate models in which clouds are very sensitive to changes in particle concentrations have got it wrong. Ackerman *et al.*[1] show that this might indeed be the case, which is perhaps not unexpected. Because climate models are typically run at a resolution of only 250 km, they cannot resolve individual clouds, and must parametrize the effects of particles on clouds — that is, they invoke comparatively crude equations of cloud physics, rather than simulate real clouds.

Ackerman *et al.*[1] used a large eddy-simulating, cloud-resolving model to examine the effects of increased particle concentration on low-lying stratocumulus clouds. They picked a number of different cases that have been studied in the past and show that an increase in the number of particles does not lead to an increase of total liquid — unless, that is, the overlying air in the region is humid. The reason is that when precipitation decreases as a result of increased particle concentrations, the mixing of air from above the cloud with that beneath it increases. If the air above the cloudy layer is dry, the increased mixing leads to less liquid water in the cloud. But if the air above the cloud is humid, then the mixing resupplies the cloud with moisture. Ackerman and colleagues' new results restrict the number of regions that may be affected by the second indirect effect to a smaller percentage of the globe, and so mean that the total impact of that effect may be much smaller than previously thought.

If the first indirect effect — cloud brightness — is as large as some models predict, aerosols are still acting strongly to increase reflection of solar radiation, and so are masking the current effect of greenhouse gases to a large extent. If so, then the net radiative forcing that is the cause of the 0.6 °C average

temperature increase of the past 100 years must be small, and climate models must be much more sensitive to this small difference if they are to agree with past observations. The future, then, might be more at the upper range of climate projections. But if aerosols do not increase low-level cloud brightness as much as some models have it, then greenhouse gases may be only slightly masked by aerosol-induced cloud changes, and projections of future climate might follow the more benign path.

How fast the world needs to address the issue of greenhouse-gas warming still depends on which of these two paths is correct. We still have the conundrum. But it is now less daunting than it was, given that the results of Ackerman *et al.* mean we can rule out some of the large effects of aerosols on cloud water content. ■

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Developmental biology

Survival by self-digestion

Nathaniel Heintz

Mammals face a problem just after birth: they are no longer nourished through the placenta, but suckling has not yet begun. How do they survive? Digestion of the animal's own cells could be the answer.

All organisms must adapt to environmental stresses. One of the most beautiful examples of a response to stress is autophagy — a process first described in electron microscopic studies in the kidneys of newborn mice[1], and now known to be one of the major pathways for the degradation of long-lived proteins and cellular organelles[2,3]. It has become clear that autophagy is activated in a variety of circumstances in multicellular organisms, and thus must be regulated by diverse stimuli in different cell types. But our knowledge of the biological roles of autophagy in mammals is quite primitive. On page 1032 of this issue, however, Kuma *et al.*[4] provide a fascinating insight into the mammalian response to birth and the biological benefits of autophagy.

In cells, nutrient deprivation and other stresses elicit a complex series of orchestrated steps that result in the cells enveloping portions of their cytoplasm, delivering them to enzyme-packed organelles known as lysosomes for degradation and recycling (hence the name autophagy, or self-eating)[2,3]. Over the past decade, genetic analysis in yeasts and morphological studies in a wide variety of species have defined the molecular mechanisms of autophagy and shown its induction under many developmental and pathological situations. The logic for the pathway that has been revealed by the genetic and molecular dissection is very pleasing — in response to nutrient deprivation, the autophagy pathway scavenges intracellular substituents to supply vital components until conditions improve.

To investigate the importance of this pathway in mammals, the Mizushima laboratory has previously[5] made use of a fusion of green fluorescent protein (GFP) and LC3, the mammalian equivalent of Atg8 — an essential protein in the yeast autophagy pathway. With this fusion protein they could reveal the formation of autophagosomes, vesicular structures that are hallmarks of autophagy. Having introduced GFP–LC3 into young mice, the authors were able to map the induction of autophagy in response to food withdrawal in many tissues, by studying the formation of small fluorescent vesicles carrying the fluorescent fusion protein.

Now, the same group (Kuma *et al.*[4]) have turned their attention to the developmental roles of autophagy. To do so, they first used GFP–LC3 to study the formation of autophagosomes in newborn mice. They observed massive induction of autophagy in the heart muscle, the diaphragm, the lungs and the skin — all tissues that undergo sudden increases in energy expenditure or environmental exposure immediately following birth. The induction of autophagy was immediate and transient, reaching maximal levels only 3 to 6 hours after birth and declining to basal levels within a day or two. To explain this phenomenon, Kuma *et al.* postulated that the induction of autophagy following birth is required to provide energy before nursing begins.

To test this attractive idea, the authors generated mice that lack Atg5 — another protein required during an early step in the autophagy programme[6]. The Atg5-knockout animals were born in normal mendelian ratios but died during the first day after birth. Measurements of autophagy with the GFP–LC3 assay demonstrated that, as expected, the formation of autophagosomes was blocked in the mutant mice. The Atg5-deficient mice also died earlier than wild-type mice when not being suckled; their survival could be extended by hand feeding; and plasma and tissue amino-acid concentrations in non-suckling mutant mice were normal at birth but very much reduced relative to controls 10 hours after delivery. Furthermore, measurements of energy status in the knockout pups indicated that energy production was severely depressed but could be restored by re-feeding.

These data show that autophagy is required to produce amino acids and to maintain energy levels in neonatal mice. Taken together with the severely low glucose and lipid levels that are generally evident in newborn mice, the death of the Atg5-knockout mice after birth provides strong support for the hypothesis that autophagy is essential for survival during the unique period experienced by mammals as they make the transition from transplacental nutrition to milk.

The idea that autophagy is important to provide nutrients for neonatal survival is very attractive. It provides a genetic link between the incisive studies of mechanisms of autophagy in individual cells, and studies documenting the induction of autophagy in tissues of fasting animals[2,3]. It demonstrates that our understanding of the biological roles of autophagy in mammals will ultimately require precise genetic dissection of this pathway *in vivo*. And it raises several issues for further investigation.

For instance, under what other conditions can autophagy contribute to viability in mammals and other vertebrates? What is the mechanism through which autophagy, which is understood as an intracellular catabolic pathway (one that breaks complex molecules down), contributes to the maintenance of plasma amino-acid levels? Which tissues contribute to this process? How important is this mechanism in other organisms in maintaining energy levels during development or fasting? What is the anatomical basis for the suckling defect seen in Atg5-knockout mice, and how does that contribute to neonatal death? In the studies presented here, Kuma *et al.*[4] make a powerful case that our understanding of the unique features of mammalian development can be enriched by genetic dissection of this beautiful and fundamental biological pathway. ■

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