Atmospheric chemistry

A bad winter for Arctic ozone

Richard Stolarski

Ozone loss in the Antarctic, resulting in a seasonal 'ozone hole', is a familiar problem. But what of the Arctic? Why are the Northern polar regions less susceptible to ozone loss; and could that situation change? Two papers (by Müller *et al.*[1] in last week's *Nature* and Rex *et al.*[2] on page 835 of this issue) now report large ozone loss during the winter and early spring of 1995–96 in the Arctic. This comes after considerable losses over the previous few winters. What has been peculiar about the past few winters is that the Arctic stratosphere has stayed cold for longer than average, and this seems to have led to low ozone concentrations.

ClO concentrations in the Arctic vortex are as high as those seen in the Antarctic, more than about 1 p.p.b.v. (part per billion by volume), and when such high concentrations are exposed to sunlight, it is well established that ozone loss will take place at a high rate. It is nitric acid (HNO_3) that makes the difference: in the Antarctic, temperatures are low for long enough that particles containing nitric acid and water grow large enough to fall out of the stratosphere, leading to denitrification and dehydration. In the Arctic, temperatures are low enough for these particles to form, but not for long enough to lead to large-scale denitrification or dehydration. Because there is a lot more nitric acid in the Arctic atmosphere than in the Antarctic, ozone loss occurs in competition with recovery of ClO to chlorine nitrate ($ClONO_2$) via reaction with NO_2 released from nitric-acid photolysis. So less ozone loss can occur here than in the Antarctic, even though the Arctic atmosphere is entirely processed by heterogeneous reactions on the surface of ice and water droplets that convert inert chlorine molecules to ClO.

Yet from initial amounts of the order of 450 Dobson units (DU), Arctic ozone concentrations have been falling to about 300 DU in the spring. (Baseline ozone amounts in the Antarctic before the emergence of the 'ozone hole' were 300 DU; they now routinely fall to about 100 DU.) So why are we now seeing these large ozone losses in the Arctic? The answer appears to be longer stratospheric winters. Even though there is little denitrification, temperatures do remain cold long enough to tie up nitrogen as HNO_3 well into the sunlit period in late winter and early spring, and to cause sporadic reprocessing of the air by heterogeneous reactions in polar stratospheric clouds. The temporary lack of nitrogen allows more ozone removal; and, because absorption by ozone warms the stratosphere, these losses feed back and may keep the stratosphere cold later into the spring. In fact, Rex *et al.*[2] saw a region in the Arctic in early 1996 where some denitrification occurred and ozone loss continued unabated for more than two months.

It is easy to describe the processes leading to large ozone losses in the Arctic in recent years, but much harder to understand the quantitative details. Unusually large downward trends in ozone concentration have been observed in northern midlatitudes over the past decade and a half; but we still don't know how much of that is due to Arctic polar processes, and how much is a result of midlatitude chemistry or dynamical changes. Also, many details of the heterogeneous chemical mechanism are not established. For example, what form do the ice particles take at temperatures of 190–200 K? Nor do we understand to what extent variability in dynamics, temperature and aerosol concentration determine the year-to-year variations in Arctic ozone.

Chlorine concentrations in the troposphere have peaked and are beginning their expected slow recovery, as the Montreal Protocol, limiting the use of chlorofluorocarbons (CFCs), begins to take effect. Stratospheric chlorine concentrations should begin to decrease soon, and ozone should then begin recovering to earlier amounts. Working out the details of Arctic ozone loss is imperative if we want to recognize ozone recovery at the earliest possible date.

How do we expect this recovery to pro-

Fluid dynamics

How coffee leaves its mark

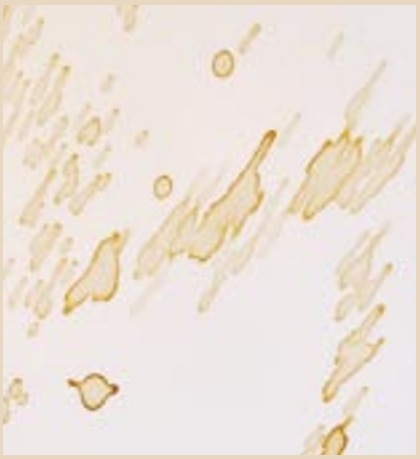
"Compared with the giants of quantum physics, we soft-matter theorists look like the dwarfs of German folk tales," says Pierre Gilles de Gennes. "We are strongly motivated by industrial purposes. We see fundamental problems emerging from practical questions." Such humility is taken to new extremes by Sidney Nagel and co-workers (Deegan, R. D. *et al.* *Nature* 389, 827–829; 1997), who find insight into the physics of soft matter by means of an eminently familiar, even mundane, phenomenon: the coffee stain.

Readers could be forgiven for assuming that the physics of an evaporating coffee droplet would be buried in the annals of a previous century. But how liquid droplets flow, spread and dissipate is imperfectly understood. The statics of the situation are explained by Thomas Young's famous condition for wetting (1805), based on the balance of surface tensions at the droplet edge. But what of the dynamics?

The dynamical situation is often complicated by the fact that the edge of the droplet (the contact line) gets 'pinned' at points on the surface, generally as a result of surface heterogeneities. This is what underlies the work of Nagel *et al.*, who propose an explanation for why coffee stains have pronounced boundaries, where most of the material is deposited. This material is uniformly distributed in the initial drop, so why does it get concentrated around the edges?

If the edge of the evaporating droplet is pinned in place, evaporation cannot simply shrink the droplet's lateral dimensions. Instead, there must be a net flow towards the edge to replenish liquid lost by evaporation while keeping the contact line in place. Suspended material is carried along with this flow, as shown by video microscopy of polymer microspheres in a drying droplet.

The idea can be made quantitative by calculating the evaporative flux from the surface of a circular droplet as a function of radial distance. It turns out that, in a dilute solution, the flux and flow velocity become infinite at the contact line. That implies complete transfer of the solute to the perimeter by the time the droplet dries out, leaving a precisely sharp ring stain. In practice, this is modified in a concentrated solute, which leaves a finite ring width and thus the sort of graduated stains seen here.



R. D. DEEGAN *ET AL.*

Industrial processes that might benefit from these ideas are the drying of ink droplets in printing and of surface coatings applied as liquids; and perhaps the efforts of amateur water-colourists, to which de Gennes has also likened soft-matter theorists: "spending their Sunday afternoons in the park, and capturing a few simple scenes."

Philip Ball

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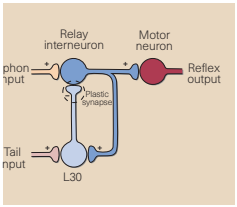

Figure 1 **A cloud in the Arctic stratosphere. Ozone losses occur in the air that has been chemically processed by such clouds.**

ceed in the Arctic? The answer depends on what we expect to happen to the temperature, which is closely related to why the past few stratospheric winters have been so cold. There are three broad possibilities:

(1) The temperature was low and the winter longer mostly because there was less ozone to absorb solar radiation. (That is, an initial effect was amplified by the feedback mentioned above.) This would imply that as ozone recovers, the temperature will recover with it. The timescale for ozone recovery would be similar to that expected in 'normal' winters.

(2) The temperature was low as part of a climatological trend independent of ozone — perhaps via CO_2 molecules radiating atmospheric heat to space. This would imply that ozone will not recover rapidly, and may remain low for another decade or more. It may be that ozone concentrations after a cold winter with 2 p.p.b.v. of chlorine are as low as those for a warm winter with 3 p.p.b.v. of chlorine.

(3) The temperature was low because of climatological variability unrelated to ozone or a climate trend. This would imply that ozone recovery will occur on the timescale of 'normal' winters, but with considerable year-to-year variability which will make it difficult to deduce when the recovery begins.

The answer is probably some combination of the above. A great improvement in our knowledge of past interannual variability and trends is necessary if we are to predict the recovery of Arctic ozone as man-made, ozone-destroying chemicals decline, and understand how other sources of stratospheric pollution, such as supersonic aircraft, may influence Arctic ozone. □

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1. Müller, R. *et al. Nature* **389,** 709–712 (1997).
2. Rex, M. *et al. Nature* **389,** 835–838 (1997).

Developmental neurobiology

Neurotrophins moving forward

John V. Heymach Jr and Barbara A. Barres

The changing size, form and connectivity of our brains — during both development and evolution — are thought to depend on trophic feedback between neurons and the target cells that they innervate[1]. An attractively simple model for how this trophic feedback works is the neurotrophic hypothesis[2]. The idea is that the number of neurons is matched to the number of target cells that they innervate by a competition for limiting amounts of target-derived peptide factors. These factors, called neurotrophins, promote survival of the presynaptic neurons by retrograde (target to neuron) signalling (Fig. 1). Thus, it now comes as a surprise to learn, in the report by Altar *et al.*[3] on page 856 of this issue, that the same neurotrophins can also act by anterograde (neuron to target) signalling.

The neurotrophins are a family of homologous proteins, including nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF) and neurotrophin-3 (NT-3). The importance of retrograde signalling by neurotrophins during normal development is well established. For instance, when axons are severed or target cells ablated, the presynaptic cells generally atrophy and die. But these consequences can be prevented if exogenous neurotrophins are applied.

What is less well recognized is that, when axons are severed, anterograde degeneration also occurs — postsynaptic cells lose spines and dendrites, atrophy and even die. For example, the survival of some neurons in the chick optic tectum, a visual centre in the midbrain, depends on trophic signals that are supplied by their presynaptic (afferent) inputs from retinal ganglion cells[4]. Neurotrophins are attractive candidates for these signals because their pattern of immunoreactivity in axon processes indicates that they may be transported anterogradely[5–7]. Moreover, exogenous BDNF can prevent the death of neurons that have had their afferents removed[8]. And when NT-3 is injected into the chick eye, it is taken up by retinal ganglion cells, transported anterogradely, and released in the optic tectum where it supports the survival of tectal neurons[9]. But these experiments have fallen short of answering whether endogenous neurotrophins are normally involved in anterograde signalling.

The study by Altar *et al.*[3] now provides direct proof that endogenous BDNF is anterogradely transported, and that the amounts transported are large enough to alter the phenotype of target cells. Using a highly specific antiserum to detect BDNF protein, they showed that it is widely distributed in nerve terminals. Remarkably, BDNF is even found in regions of the brain that lack BDNF messenger RNA, such as the striatum. Because the presynaptic cells that innervate the striatum produce both BDNF mRNA and protein, these results strongly suggested that the cortical cells anterogradely transport BDNF to the striatum.

To test this possibility directly, colchicine (a drug that disrupts microtubules) was used to inhibit axonal transport. The immunoreactivity of BDNF markedly increased in the cell bodies of the cortical neurons, and it decreased in the striatum. Furthermore, cortical ablation — which eliminates the striatal afferents — eliminated the axonal BDNF immunoreactivity in the striatum, and decreased the amount of BDNF there by 66 per cent. (Much of the residual BDNF was shown to derive from other afferent pathways.) In contrast, ablation of the striatal neurons with an excitotoxin did not decrease the levels of BDNF in the striatum. So, the bulk of striatal BDNF must be anterogradely transported from the cortex. Because almost all of the BDNF in the striatum is present in the terminals of afferent neurons, and

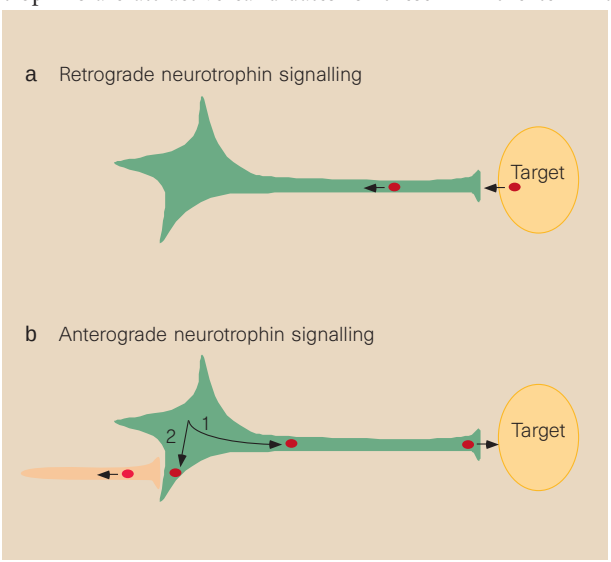

Figure 1 **Retrograde and anterograde signalling.** a, **Target-derived neurotrophin (red circle) is taken up by an innervating (afferent) neuron, and retrogradely transported back to the cell body, where it affects survival and differentiation. The target may be neuronal or non-neuronal.** b, **The neuron may direct its neurotrophin down the axon for anterograde, neuron-to-target signalling as shown by Altar *et al.*[3] (1), or to its dendrites or soma (2), for uptake and retrograde transport by an afferent (presynaptic) neuron.**

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