their respective membrane anchors, thereby forcing the membranes close together and overcoming the energy barrier that normally prevents fusion.\(^2\) (Fig. 1). Interestingly, a similar mechanism has been suggested to explain how enveloped viruses infect cells.\(^3\) These viruses inject their genetic material using specialized membrane proteins that mediate fusion between the viral membrane and that of a target cell. After appropriate activation, viral fusion proteins change conformation and can insert a newly exposed amphiphilic fusion peptide into the target membrane.\(^4\) Subsequent conformational changes seem to allow the now doubly anchored protein to fold back on itself, pulling the fusing membranes together.\(^5\)

The SNARE complex has, until now, mainly been studied using proteins in solution, without the membranes that they ultimately control. Weber et al.\(^6\) have taken the step of successfully reconstituting SNAREs into artificial membranes, allowing these proteins to be studied in a defined environment. With synaptobrevin in one membrane and syntaxin and SNAP-25 preassembled in another, they found that these proteins could form ternary complexes and induce interactions between the membranes. The interactions were observed as slow mixing of the membrane bilayers, suggesting that the vesicles had spontaneously fused with one another. Thus, the idea that SNAREs can interconnect — and possibly fuse — membranes has been realized in a purified system, complementing results obtained using the complex biological membranes of yeast vacuole-precursor vesicles.\(^7\)

These models of SNARE function are almost certainly oversimplified. But they do account for most of the available data. The use of artificial membranes, such as those described by Weber et al., will open the way to understanding the molecular mechanisms that underlie fusion of biological membranes, while distinguishing them from nonspecific fusion caused by membrane-perturbing molecules such as annexins and amphiphilic peptides.\(^8\) Much will be learned by understanding how (and how many) ternary complexes are arranged at sites of fusion, and how regulatory proteins such as munc-18/Sec1, Rab/Ypt, and the calcium sensor synaptotagmin interact with and affect such complexes. These approaches should help us to work out how biological fusion acquires its characteristic efficiency, speed and regulation.

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allowed momentum quantization values come with the preferred directions for conduction. In other words, the electronic properties of tubes are controlled by their structural details. This remarkable prediction has recently been verified using a scanning tunnelling microscope to image the atomic structure of tubes and probe their electronic properties.

By attaching leads to nanotubes, electronic devices are created. Metallic tubes have already been made into single-electron transistors that operate at low temperatures, but ‘Tans et al. now provide the first report of a nanotube transistor based on semiconducting tubes (Fig. 1). A nanotube lies across two metallic contacts fabricated on top of a layer of SiO₂, and a voltage is applied to the conducting substrate to move carriers onto the tube. The authors find that the tube can be turned on by applying a negative bias to the substrate, which induces holes on the initially non-conducting tube. This device is thus analogous to a p-type MOSFET, with the nanotube replacing silicon as the material that hosts charge carriers. The resistance of the device can be changed by many orders of magnitude, and it operates at room temperature — a property that has eluded most other nanoscale devices.

This experiment is a first step in developing electronic devices based on semiconducting nanotubes. Other advances are likely to follow soon. Doping of the nanotubes is possible, and has been demonstrated in ensembles of tubes and more recently in single ropes (M. Bockrath, personal communication). Other devices, such as p-n junction diodes and bipolar transistors, are likely to be realized soon. And more exotic devices are possible, owing to the unusual properties of nanotubes. For example, a single defect can change the structure of a tube from the metallic to the semiconducting variety, creating a metal–semiconductor junction composed entirely of carbon atoms. Such devices would be free from problems that occur in conventional devices made from different materials, such as interdiffusion. They would also, of course, be much smaller, and therefore much faster.

The success of carbon-based electronics will depend on how rapidly the techniques for fabricating, doping and manipulating nanotubes develop. As yet these techniques are crude, and dreams of a controlled technology seem fanciful; but many groups are working hard on these issues, and their progress is impressive. By combining techniques from engineering, chemistry and biology, researchers are learning how to grow, cut, sort and chemically modify nanotubes in new and exciting ways. It is difficult to imagine carbon-based electronics competing head-on with silicon in the near future, but there may be niche applications to which it is particularly well-suited — and if carbon is indeed the atom of the twenty-first century, then history is on its side.

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Conservation biology
What killed the monk seals?
John Harwood

In May and June 1997 the bodies of over 100 Mediterranean monk seals (Monachus monachus) were found along the Cap Blanc peninsula. This short stretch of coast, which spans the border between Mauritania and the former Western Sahara, is home to the largest surviving colony of these critically endangered creatures. The species’ highly fragmented distribution extends as far east as the Black Sea, but the only other population of any size is over 4,000 km from Cap Blanc, in the eastern Mediterranean. Few ecologists were surprised when this mass mortality was attributed to infection with a previously undescribed morbillivirus, for viruses in this family have caused similar catastrophes in other species of marine mammal. The seals in the affected area are also virtually confined to two caves only about 1 km apart, so providing ideal conditions for the transmission of infection.

On page 28 of this issue, however, Hernández et al. suggest that the seals actually died because they had eaten fish contaminated with phycotoxins, which are produced during blooms of certain dinoflagellate algae. The same toxins cause paralytic shellfish poisoning in humans. Neither of the algal species implicated in the die-off had been recorded on this coast before 1994, when one of the species was responsible for the death of four people.

Identifying the cause of mass mortalities in wildlife populations is always tricky, and could have been particularly difficult in this incident (the political status of the Western Sahara has been undefined for several decades, making access difficult, and the whole area is littered with landmines). However, the Cap Blanc seals have been studied by a team from the University of Barcelona since 1993 (ref. 3), and many individuals can be recognized by their unique markings. Also, the prevailing currents ensured that seals that died close to the shore were likely to be washed up on the beaches of the peninsula. As a result, it was possible to estimate that about 70% of the local population had died, constituting about one-third of the world population.

Hernández et al. detected several phycotoxins in the dead seals, and found high concentrations of the dinoflagellate Alexandrium minutum in the coastal waters. Other workers have found evidence that the same toxins had entered the nervous system of some of the dead seals. Nevertheless, we cannot be certain that they were the cause of death, because there are no data on lethal or background levels of these toxins in seals.