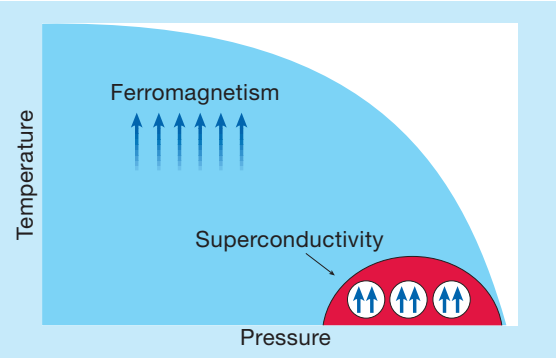

Figure 1 **Temperature–pressure phase diagram for UGe_2. Superconductivity is observed in a narrow regime just within the ferromagnetic state of an itinerant electron system. Arrows indicate relative spin orientations. (Courtesy of K. Ahilan, S. S. Saxena & G. G. Lonzarich.)**

temperature superconductors. But despite 30 years of speculation, there were still no firm examples of superconductivity having developed in association with ferromagnetic spin fluctuations.

The discovery by Saxena *et al.*[1] of superconductivity inside a ferromagnet (a uranium–germanium compound, UGe_2) finally sets the record straight. Saxena *et al.* realized that past experiments simply did not get close enough to the boundary with magnetism. Unless the spin-pairing forces are strong, tiny amounts of disorder are enough to destroy the coherent scattering effects needed for triplet pairing. The idea was to use high pressures to bring a ferromagnet to the very brink of magnetic order. The temperature at which a ferromagnet loses its ferromagnetism is the Curie point, and applying pressure to a ferromagnet depresses its Curie temperature to zero kelvin. Saxena *et al.* reasoned that when the Curie temperature goes to zero, the resulting strong magnetic fluctuations would eventually cause the system to develop the long-sought-after superconductivity.

This idea had previously been tried without success on another ferromagnet, manganese silicon. Here it turns out that a quirk of crystal symmetry — the absence of inversion symmetry — prevents triplet pairs from forming. The material chosen for this study, UGe_2, avoids this problem, and can also be prepared in crystals of high purity. The f-electron character of this ferromagnet causes its Curie temperature to be highly sensitive to external pressure, and 1.6 gigapascals (1.6 GPa or 16 kilobars), well within the range of modern high-pressure experiments, is sufficient to eliminate the magnetism.

But Saxena *et al.* report another surprise: they find that superconductivity actually develops at a lower pressure, before the ferromagnetism is eliminated, producing an unexpected situation in which superconductivity coexists with ferromagnetism. Measurements of the a.c. susceptibility confirm the observation of bulk superconductivity within the ferromagnet. Could the observed superconductivity be of the old-fashioned antiparallel, singlet variety? It's highly unlikely. The authors point out that the energy bands of spin-up and spin-down electrons inside the ferromagnet are likely to be split by an energy gap of roughly 70 millielectronvolts — equivalent to a temperature of almost 1,000 kelvin. The only way in which Cooper pairing could take place under these conditions is for the spins in the Cooper pair to be aligned in a parallel, or triplet, configuration[6].

What then is the origin of the electron pairing in UGe_2? No definitive answer can yet be given, but the discovery of pairing in the region where magnetism vanishes is a strong clue that it is likely to be magnetic. Furthermore, Saxena *et al.* show that the highest superconducting-transition temperature is obtained at a pressure where the density of electron states is at its highest, and it is here that magnetically mediated pairing is expected to be strongest. Unfortunately, the ferromagnetic phase ends abruptly at 1.6 GPa, and no trace of the superconductivity survives at higher pressures. Future experiments that combine pressure with an applied magnetic field may show how superconductivity extends into the non-magnetic phase. Moreover, experiments using the much higher pressures available with diamond anvil pressure cells may be able to induce superconductivity on the verge of magnetism in a broad variety of magnetic materials. ■

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Erratum

The technique known as protein signature analysis, developments of which were discussed by Satish K. Nair and Stephen K. Burley in the article "Functional genomics: Recognizing DNA in the library" (*Nature* **404,** 715–718; 2000), was originally described in a paper by T. W. Muir, P. E. Dawson, M. C. Fitzgerald and S. B. Kent in *Chemistry and Biology* (**3,** 817–825; 1996).

Daedalus

Spinning and leaking

Can microwaves damage living tissue — in particular the brain tissue of mobile-phone users? Mobile phones put out too little power to cause much heating; even so, they may induce more subtle non-thermal damage. Daedalus now proposes a mechanism.

A typical cell membrane is a fairly fluid bilipid layer with a few big protein molecules embedded in it. These molecules are receptors or 'antennae' by which the cell responds to specific molecules around it, or announces its internal state to the outside world. Every protein molecule, says Daedalus, is polar, and will tend to align itself with an electric field. It is also chiral, and often helical: rotate it in its socket, and it will screw inwards or outwards. Together, these facts mean that a rotating electric field could screw it into or out of the cell.

Almost any microwave source, such as a mobile-phone transmitter, will have some component of circular polarization. It will make billions of rotations every second. Any volume of brain tissue must have a few cells bearing protein molecules that happen to resonate at its rotation frequency. Even very weak coupling to the field should soon spin these molecules through the few turns needed to screw them out of the cell membrane. The cell might then simply leak to death — penicillin kills bacteria by making them leak. Or it might plug the hole with some wrong protein, creating more indirect trouble. Either way, the cell could lose the information entrusted to it, causing the memory loss complained of by mobile-phone users.

So, says Daedalus, to reduce the hazards of mobile phones, minimize the component of circularly polarized radiation in their output. But he also sees the way to a powerful new radiotherapy. If you knew the rotational resonant frequency of a particular protein in a particular type of cell, you could hit it with circularly polarized microwaves of exactly that frequency. Molecules of that protein would be spun out of their cell membranes, killing the cells or inducing them to take up some specially tailored therapeutic 'channel-blocker' which you had cunningly injected beforehand. Other proteins, and other cells nearby, would be unaffected. This elegant therapy could be directed against foreign bacteria or parasites in the body, or over- or underperforming glands, or cells succumbing to viral or neoplastic derangement. It would need much knowledge and insight to develop, but would have amazing power, specificity and freedom from side effects. **David Jones**