evidence that the virus is linked to SIVcpz there may be calls for more use of chimpanzees in AIDS research. But according to a growing constituency, evidence of ape possession of human-like cognition and emotions should reduce our willingness to infect them, and condemn them to solitary confinement for life (which lasts for 60 years or more)[16].

The approach of Gao *et al.* may prompt fresh thinking. Fieldwork with free-living apes provided information for the evolutionary history of chimpanzee subspecies that was critical to their results. Further fieldwork linking demographic data to biomedical monitoring could be even more productive. The four chimpanzee subspecies have deep evolutionary roots, and may yield further types of SIV beyond the two already identified. These are best studied in the place where the host–virus systems evolved. Chimpanzees in captivity are mostly taken from the wild before they become sexually active, and so rarely harbour SIV. Infection can be expected at higher frequencies in wild adults[17], and such individuals would offer research opportunities that simply do not pertain in captivity, where experimenters cannot know how virus strains match to genetic lineages.

Two other ways forward are these. Field researchers intermittently find fresh corpses of individual apes, often with well-documented life-histories, which offer untapped possibilities for virological analysis. And DNA studies of faeces might allow non-invasive monitoring of virus infection.

Biomedical researchers have the prospect of collaborating with fieldworkers in a synthesis that would benefit conservation. The link between HIV-1 and SIVcpz may open a door for research that helps both humans and apes. □

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Condensed-matter physics

# Return of the itinerant electron

**David Ceperley**

On page 412 of this issue, Young *et al.*[1] demonstrate magnetic polarization in a dilute, three-dimensional electron gas at a temperature of 600 K. Years of theoretical debate connect this intriguing result to a long-sought mechanism for ferromagnetism in metals first conjectured by Bloch[2], who suspected that the 'electron gas' is susceptible to magnetic ordering at low density.

What is an electron gas and why is it interesting? The idealized homogeneous gas (also known as jellium or the one-component plasma) has a long history going back to the discovery of the electron. It was used by the early quantum pioneers to model the valence electrons in a solid; they replaced the ions by a rigid uniform background of positive charge. This is arguably the most important model in condensed-matter physics because it is routinely used as a reference state in most realistic calculations of electronic structure. The valence electrons are the outer electrons of the atom, which determine how it interacts with its environment, so understanding the properties of the electron gas is significant. In most metals, the density is high enough that the electrons move almost independently and form a 'normal Fermi liquid' of mobile electrons (Fig. 1) with equal numbers of electrons with up and down spin. But at low density the potential energy dominates the kinetic energy, and the electron 'gas' freezes into the 'Wigner crystal'[3], with the electrons localized on lattice sites. This dependence on density is the exact opposite found in systems with cores, such as atoms, where it is high not low density that favours a lattice structure.

In 1929, Bloch noticed that within the 'Hartree–Fock' approximation (that is, assuming the electrons are in independent states), the spins of the electrons spontaneously align, and he suggested this as an explanation of ferromagnetism. This spin polarization should happen at a density just lower than that found in some alkali metals, such as potassium, rubidium or caesium. But at these densities one cannot ignore the fact that electrons move in a correlated way. In the Stoner model[4] introduced in the 1930s, the electron–electron Coulomb interaction is replaced by a constant repulsive energy between opposite spin electrons, to account for this correlation. The spins only partially polarize and they do so at a lower density. Many theorists have concluded from this and other calculations that the electron gas would never be ferromagnetic[5]. Calculations at an intermediate density are difficult because very small energy differences are important and any approximation has to treat the various 'phases' of the gas with equal accuracy.

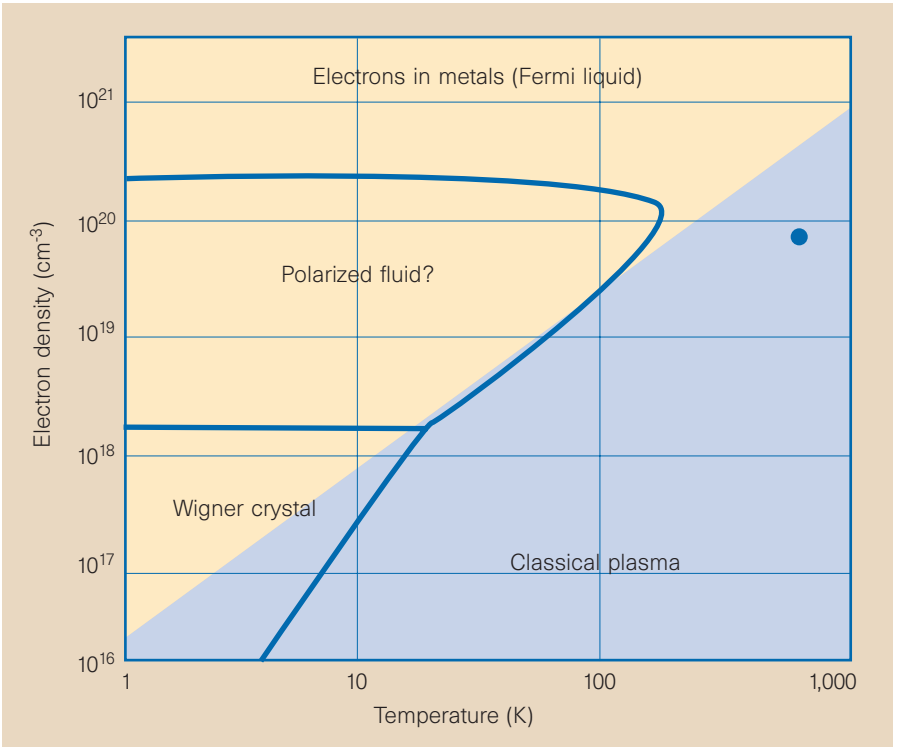


Figure 1 **Phase diagram of the electron gas. The two colours divide the classical (blue) from the quantum (yellow) regimes. The phase transition boundaries are estimates from ref. 6. The dot is the transition temperature measured by Young *et al.*[1].**

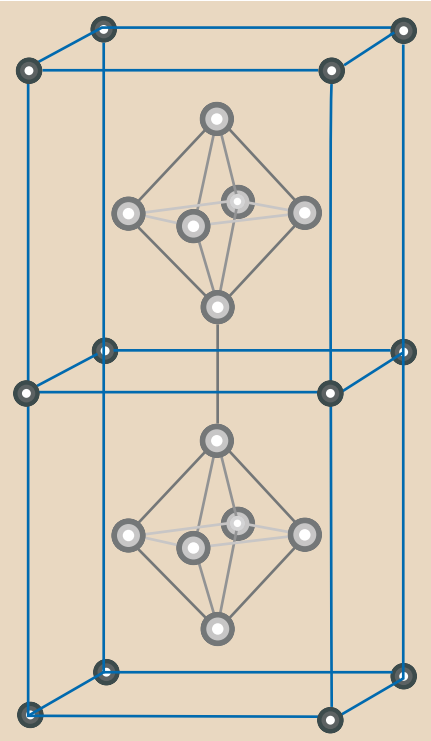

Figure 2 **Crystal structure of $CaB_6$. The calcium atoms are in black and the boron atoms in grey.**

The most reliable calculations[6] have used a computer simulation method known as quantum Monte Carlo. These calculations predict that electrons will spin-align at a density less than $2 \times 10^{20}$ cm$^{-3}$ and crystallize at a density of $2 \times 10^{18}$ cm$^{-3}$ into the body-centred cubic lattice of the Wigner crystal. The magnetic transition is predicted to be gradual with the spin polarization building up slowly as the density drops, just as in the Stoner model. Such computer calculations have only been done at zero temperature; so the estimate of the transition temperature shown in Fig. 1 is a combination of the computer results and the Stoner model. These predictions have remained untested because of the difficulty of achieving a physical realization of the electron gas at low densities.

Young *et al.*[1] report ferromagnetism in a system that is plausibly modelled by the electron gas. They take calcium hexaboride ($CaB_6$, a semi-metal) and replace a few of the calcium atoms with lanthium atoms (Fig. 2). Each impurity atom adds an electron to the conduction band. By changing the impurity concentration they vary the effective electron density just as in doped silicon semiconductors. Remarkably, the authors observe spontaneous partial magnetization for a range of densities, in agreement with the predictions of the electron gas calculations. Several analogous compounds, for instance strontium hexaboride, show the same ferromagnetism. However, the transition temperatures at which ordering occurs are a factor of three higher than the computer estimates. In order to interpret the experiment is it necessary to correct for 'band effects', because the electrons are not free but move inside a cubic lattice. One important correction is that conduction electrons move in states oriented to the cubic crystal axes, producing an extra quantum number not considered in the electron-gas model. In addition, the effective mass of conduction electrons in $CaB_6$ is half their vacuum value[7] and depends on direction, and the other electrons screen the electronic charge. Such effects enhance the natural tendency of the electron gas to spin align. These corrections are so important that detailed calculations will be needed to establish the role of itinerant electrons in the observed ferromagnetism.

Such spin–spin correlations are ubiquitous in strongly correlated Fermi liquids and can dominate how these systems react to their surroundings. The clearest example is superfluid helium-3, which although not ferromagnetic, is nearly so. A more complicated and disputed example is of spin correlations in the high-temperature superconductors. Experiments with hexaborides could help the essence of the ferromagnetic electron to be revealed. □

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**Apoptosis**

# A cellular poison cupboard

**William C. Earnshaw**

Apoptotic cell death is spectacular — one moment a cell looks peaceful and happy, the next it enters a violent programme of cytoplasmic blebbing worthy of the death throes of an actor in a B-movie. One fascinating aspect of apoptosis is that virtually all cells contain latent forms of the molecules that trigger this destructive burst. A report by Susin *et al.*[1] on page 441 of this issue supports the idea that several of these key death factors normally reside in the space between the inner and outer membranes of the mitochondrion.

Apoptosis is driven by two classes of highly specialized proteases known as caspases (cysteine aspartases). When properly assembled on a molecular scaffold, 'initiator' caspases can be activated by self-cleavage. 'Effector' caspases are then activated downstream of the initiator caspases in an amplifying cascade. Mitochondria come into play because they intervene between these two classes of caspase in many apoptotic pathways. For example, active caspase-8 (an initiator caspase) can act either directly on downstream effector caspases or indirectly on mitochondria, in both cases releasing a cocktail of lethal factors that amplify the death response.

The first such mitochondrial factor to be described binds to a cytoplasmic scaffolding protein called Apaf-1. Binding of the mitochondrial factor allows Apaf-1 to form a ternary complex with, and activate, the initiator procaspase-9 (ref. 2). Active caspase-9 then turns on downstream effector caspases, unleashing the death cascade. The announcement that this mitochondrial factor was none other than cytochrome *c*, a component of the respiratory chain that is normally found in the mitochondrial intermembrane space[3], was greeted with incredulity — it was a great surprise that such a well-understood molecule could lead such a Jekyll-and-Hyde existence.

Susin and colleagues[1] now extend previous experiments to show that purified mitochondria can release another factor that induces apoptotic morphological changes in nuclei *in vitro*[4]. They have termed this factor apoptosis-inducing factor (AIF), and find that it is a widely expressed flavoprotein. Found in the mitochondrial intermembrane space, AIF has sequence homology to bacterial oxidoreductases (electron acceptors and donors). The AIF is released from mitochondria by apoptosis-inducing stimuli, allowing it to travel to, and accumulate in, the nucleus.

Susin *et al.* provide compelling evidence that AIF deserves a place on the centre stage, along with other well-known components of the apoptotic machinery. During apoptotic execution, caspases act both as executioners that cleave key proteins, and as executives that turn cell-survival pathways off and death-promoting activities on. One such death-promoting factor is a nuclease called caspase-activated DNase[5] (CAD; also known as caspase-activated nuclease and DNA fragmentation factor 40 kD). Purified, recombinant CAD can, on its own, induce the collapse and hypercondensation of the chromatin against the nuclear periphery — events diagnostic of apoptotic execution. Yet CAD is not essential for these final events of nuclear disassembly[6], indicating that other factors are involved.

Sure enough, when Susin *et al.* added recombinant AIF to isolated nuclei, they saw chromatin condensation at the nuclear