Molecular Electronics Emerges from Molecular Magnetism

Michel Verdaguer

The history of mankind is often marked by the names of materials designed to respond to new needs of society, and thus, the present era could be called the Age of Silicon and Electronics. As the limits of silicon are approached, molecular electronics offers an alternative route to the design of materials for use not only at the macroscopic scale but at the molecular scale. Data would be stored, transmitted, and retrieved by means of molecules, as in complex biological systems. The hope is that molecular electronics will reduce device dimensions and increase speed by orders of magnitude. Chemists are therefore looking for suitable molecular systems and for simple means to switch them between two available states. Concerning the systems, molecular magnetism is a rapidly developing field (*1*) because nanomagnetic materials easily reverse their magnetic moments in a magnetic field and are already widely used in information storage. Concerning the means, light is a very convenient and powerful way to induce molecular change.

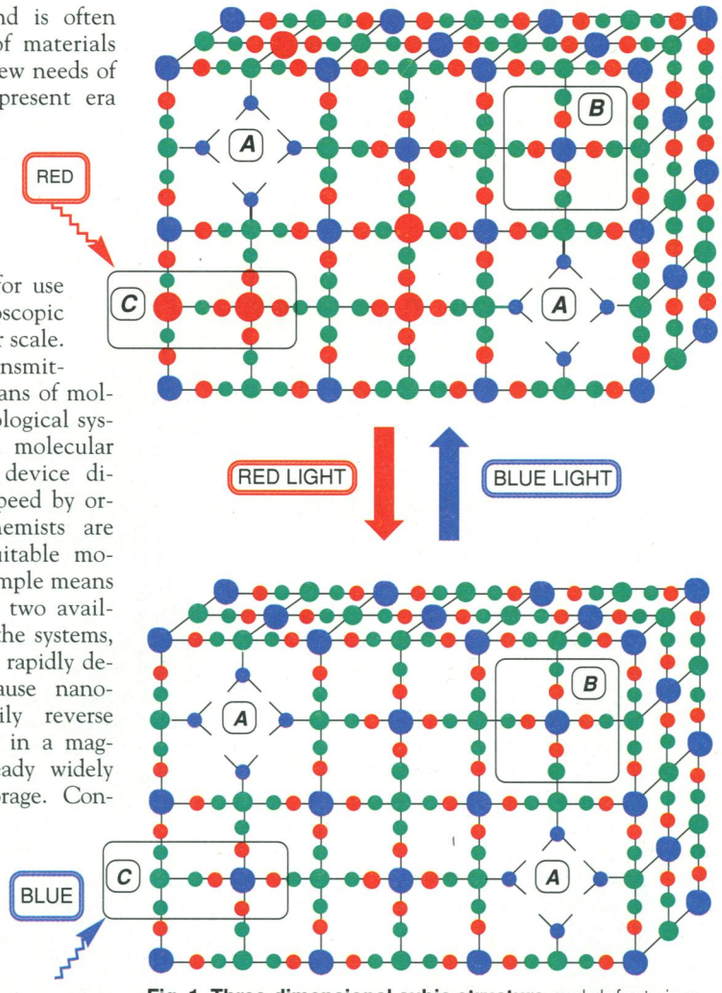

Fig. 1. Three-dimensional cubic structure and defects in a Prussian blue analog: suitable photons can switch on and off diamagnetic pairs in active magnetic analogs (see text).

Sato and co-workers (*2*) report on page 704 of this issue that they have found a way to switch the long-range magnetic properties of a simple molecule-based system derived from Prussian blue with light of different wavelengths. They demonstrate that the magnetic ordering temperature (that is, a long-range phenomenon) can be changed by photoinduced electron transfer through a molecular bridge (a molecular phenomenon), a significant contribution toward applying molecular magnetism to molecular electronics.

Preparation of Prussian blue from aqueous solutions of potassium ferrocyanide and ferric salts is a routine college chemistry exercise. The substitution of Fe(III) and Fe(II) by other ions A and B leads to a family of compounds with the rock salt structure. In these compounds, molecular hexacyanometalate anions $[B(CN)_6]^{p-}$ and the metallic cations A^{q+} occupy alternate vertices of the cubes, where the A–N≡C–B distance is about 5 Å. When paramagnetic ions are used, compounds with various magnetic properties are created. In particular, the linear sequence –B–C≡N–A– leads to an interaction between the magnetic ions. In the case of Prussian blue, the only magnetic ions are the ferric ones (five unpaired electrons, spin $S = 5/2$). Their interaction through the extended diamagnetic N≡C–Fe(II)–C≡N bridge, at more than 10 Å, is weak. Below the Curie temperature T_c = 5.5 K, the system changes from a paramagnetic state, in which the spins are disordered by thermal activation energy kT, to an ordered one, in which the spins are aligned in the magnetic field in all three dimensions.

For practical applications, high Curie temperatures are necessary. In the past few years, several research teams tried to enhance this ordering temperature by changing the nature of the interaction between A and B through the cyanide bridge. Babel was the first, in 1982, to reach 90 K (above the temperature of liquid nitrogen), with a ferrimagnetic Cr(III)/Mn(II) system (*3*). The Curie temperatures continued to increase (*4*) and, recently, the room-temperature barrier was overcome, at 315 K, at the very end of 1995 with a Cr(III)/[V(II)-V(III)] compound (*5*). All of these enhancements were achieved by changing the A and B ions.

The approach proposed by Sato *et al.* is different but new and very appealing (*2*): They tune the electronic structure and the magnetic properties by applying light. Their compound appears very simple: $K_{0.2}Co_{1.4}[Fe(II)(CN)_6]$. In fact, the formula hides a complex system containing many $(NC)_5Fe(III)$-CN-$Co(II)(NC)_{5-x}(OH_2)_x$ units and vacancies filled with water (Fig. 1, A sites), some isolated diamagnetic units $[Fe(II)(CN)_6]$ (Fig. 1, B sites), and a non-negligible amount of $(NC)_5Fe(II)$-CN-$Co(III)(NC)_{5-y}(OH_2)_y$ pairs (Fig. 1, C sites, top). The surprising presence of Co(III) rather than Co(II) is most probably related to the number of large ligand field sites around the cobalt (y = 0 or 1 in the above formula). The ligand field induces a transition to low-spin Co(III), associated with an electron transfer to the stable low-spin Co(III). A similar phenomenon was already observed by Hendrickson and co-workers (*6*) in a molecular complex. The compound is dark purple: A charge-transfer excited state exists at low energy above the ground state, which can be populated by visible light. Sato *et al.* took advantage of this peculiar situation to change the nature of the sample under irradiation by provoking an internal redox reaction by a photoinduced electron transfer. Even if the exact phenomenon is still under investigation, their observations appear compatible with the process (Fig. 2).

Before the excitation, the iron and cobalt ions are low-spin and diamagnetic, and there is no interaction between them.

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The red-light excitation induces spin on the ions [with CN surroundings, Fe(III) is low-spin (S = 1/2), whereas Co(II) becomes high-spin (S = 3/2)] and a new interacting Fe(III)-Co(II) pair. The important point is that the local electron transfer, at the molecular level (Fig. 2), switches on the interaction and allows the extension of the cooperative phenomenon throughout the network. It enhances the mean number of magnetic neighbors z. The ordering temperature T_C is therefore enhanced. The increase is weak (4 K) but significant. An even more exciting observation by Sato *et al.* is that the process can be partially reversed. Changing the color of the light is enough to go back to a state that looks like the initial one and to switch off some Fe(III)–C≡N–Co(II) interactions.

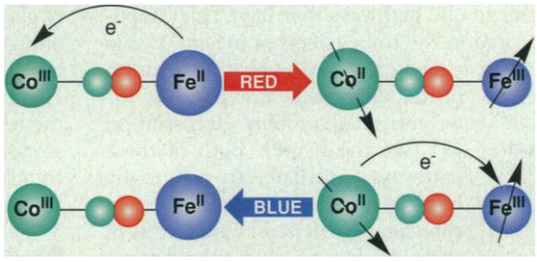

Fig. 2. Back and forth electron transfer induced by photons of different wavelengths through the molecular bridge, and the related magnetic changes.

The phenomenon observed by Sato *et al.* occurs in too low a temperature range (15 to 19 K) and is too slow (a few minutes) for practical applications. It demonstrates, nevertheless, that the tuning of long-range magnetic ordering is possible through a molecular excitation induced by photons. It is one of the necessary steps toward the design of molecule-based magneto-optical devices. Another step was announced recently by the same authors: They displaced electrochemically the T_C of the Mallah's compound (*4*) from 240 to 270 K, which is near room temperature (*7*).

The Prussian blues are far from perfect models. They are not truly molecular, but molecular-based (*8*), at the border between molecular and solid-state chemistry. They present many vacancies and defects and are often mixtures. It is amazing that such shortcomings of the oldest molecule-based inorganic system, which lies between molecular and solid-state chemistries, are the origin of the present discovery. The work of Sato *et al.* shows that we have still many things to learn from old systems, once we look at them anew.

References and Notes

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Diabetes Complications: Why Is Glucose Potentially Toxic?

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The discovery that insulin can save the life of a patient with diabetic ketoacidosis (acidic blood) or severe hyperglycemia (high blood sugar) was a landmark in the history of medicine. Despite insulin treatment, however, most diabetic patients eventually experience one or more of the long-term complications of the disease. These complications arise from chronic hyperglycemia, which causes damage to small- and large-caliber blood vessels and peripheral nerves, greatly increasing the risk of heart attack, stroke, blindness, amputation, and kidney failure. Exactly how hyperglycemia causes these complications has been debated for years, but the recent National Institutes of Health–sponsored diabetes control and complications trial (DCCT) clearly implicates glucose as a potentially toxic molecule (*1*). With today's technology, the blood glucose concentrations of diabetics cannot be completely normalized, a situation that has spurred the search for a way to prevent the toxic effects of glucose in the hyperglycemic patient. To accomplish this goal, the mechanisms of glucose-induced tissue damage must be understood and interrupted. In this issue of *Science*, an academic-industry collaboration reports progress in this direction. Ishii and co-workers (*2*) provide new support for the hypothesis that activation of the β_2 isoform of protein kinase C (PKC) in vascular tissue is a key step in the cascade of events through which glucose triggers diabetic complications (*2*).

PKC is a member of a ubiquitous family of enzymes that phosphorylate serine and threonine residues of intracellular proteins involved in signal transduction (*3*). In vascular smooth muscle, PKC activity influences contractility, mediates cell signaling initiated by hormones such as vasopressin and angiotensin II, and alters expression of specific genes (*4*). In endothelial cells, PKC influences permeability, presumably through effects on carrier-mediated transendothelial transport (*5*). Several isoforms of PKC are present in the microvasculature of the brain (*6*), where carrier-mediated transport of nutrients (glucose) and hormones (insulin) meet the brain's energy requirements and convey important physiologic signals (*7*).

The PKC enzyme system increases in activity in vascular smooth muscle and endothelial cells after in vitro exposure to hyperglycemia and in animal models of diabetes (*4*), but the presence of 12 isoforms of PKC has complicated the dissection of the activation. With the use of new molecular tools and specific antibodies, however, the β_2 isoform has been identified as potentially important in the vascular response to hyperglycemia. To test this hypothesis, Ishii *et al.* used a new, specific inhibitor of PKC β isoforms and showed that this inhibitor could normalize the elevated PKC activity in retina and kidney of diabetic rats. This effect is paralleled by normalization of the rate at which the kidney filters blood and of retinal blood flow, both of which are altered in diabetic animals and people. These results provide strong evidence that the β isoform is the key to the glucose-induced activation of PKC in vascular tissues and that this activation has pathophysiologic consequences.

Does this mean that some of the long-term complications of diabetes can be prevented by pharmacologic inhibition of PKC? The new findings are encouraging, but their clinical application is not quite yet

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