Are high-T_c superconducters doped Mott insulators?

SIR—In a recent report in *Nature*, Sir Nevill Mott[1] pointed out that there are almost as many theories of high-T_c superconductivity, where T_c is the critical temperature, as there are theorists. However, he showed a preference for a phonon-mechanism theory. At the European Physical Society in April 1987, in Pisa, I suggested that high-T_c superconductors are doped Mott insulators, that is, there exist strong Coulomb correlations in the narrow band made by the hole of Cu^{2+} ions. This should lead not only to the insulating behaviour but to an antiferromagnetic ground state for the undoped La_2CuO_4 compound. My theoretical considerations received scepticism except from K. A. Müller.

Since the recent experiments at AT&T Bell Laboratories[2] and by the Berkeley group which showed no isotope effect in the superconductor $YBa_2Cu_3O_7$, the importance of correlations as a possible mechanism for superconductivity is now recognized. Subsequently, in *Nature*, Anderson and Abraham[3] excluded all theories based on a phonon mechanism and argued that experiments indicate a strong short-range interelectronic repulsion U. But the quoted theories are all in the small U limit except for Anderson's resonant valence bond (RVB) model[4] which starts from a non-magnetic insulator.

My proposal that high-T_c superconductors are doped antiferromagnetic Mott insulators is now supported by several reported magnetic properties of these compounds. But I believe that the importance of these results has been insufficiently stressed. Furthermore, starting from an antiferromagnetic Mott insulator, I present a physical picture which is different from the one developed by Anderson in the RVB model.

As far as the magnetic properties of these materials are concerned, neutron diffraction studies[5] on La_2CuO_4 have shown antiferromagnetism. Susceptibility measurements[6] performed at IBM on doped superconducting materials show a large negative Curie paramagnetic temperature, indicating antiferromagnetic correlations in these compounds. For the other family, the compound $YBa_2Cu_3O_{6.5}$, which contains only Cu^{2+} ions, shows semiconducting behaviour and also a susceptibility with a Curie–Weiss component. More experiments are needed but we expect that neutron diffraction above T_c would show directly antiferromagnetic correlations in the two families.

On the theoretical side, I recently used an approach developed for explaining the magnetic properties of a lattice of Jahn–Teller ions[7]. I applied it to Cu^{2+} in a perovskite structure and predicted an antiferromagnetic ground state for La_2CuO_4 that neutron experiments confirmed. This shows therefore that the usual approach of the lattice of Jahn–Teller ions developed in the large-U limit can be successfully applied to these new copper perovskites. In the large-U limit, the undoped materials can be either an application of Anderson's RVB model or an antiferromagnetic Mott insulator. Present experiments strongly support the second point of view.

A main difference between my approach and that of Anderson's is as follows. While doping (creating holes in the lower Hubbard sub-band) I do not allow the pre-existing pairs of the RVB model to move as they are in an antiferromagnetic state. In fact the antiferromagnetic state is destroyed but the strong interactions between spins on the copper atom will persist. At high temperatures, it leads to the Curie–Weiss behaviour with a negative Curie paramagnetic temperature; at temperatures linked with the strength of the interactions, it leads to superconductivity. The behaviour of T_c as a function of doping is different from the RVB model and so are some of the other physical properties. For example, I calculate a jump in the specific heat function of doping[8]. I predict no pairs above T_c but the properties are described through a correlated hole hopping within a lower Hubbard sub-band as discussed at length by Mott[9]. Below T_c, the antiferromagnetic interactions between carriers are directly responsible for the new type of superconductivity, which is singlet and anisotropic.

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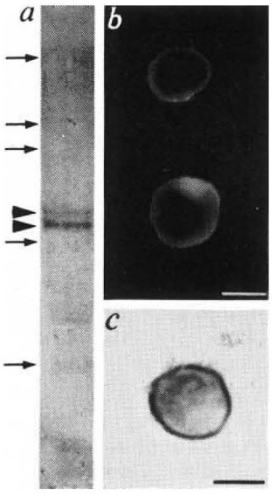
Synapsin or protein 4.1 in chromaffin cells

SIR—The role of the cytoskeleton in the control of exocytosis in secretion from adrenal chromaffin cells and neurotransmitter release from nerve terminals was discussed in our recent News and Views articles[1,2]. Commenting on these articles, Siegel[3] speculates about common mechanisms in the control of exocytosis in chromaffin cells and nerve terminals and poses the question of whether synapsin I is present in chromaffin cells.

Synapsin I is believed to be important in the nerve terminal, where its state of phosphorylation could determine the extent of actin filament crosslinking and possibly linkage of actin filaments to synaptic vesicles[4]. As Siegel[3] points out, secretory granules from chromaffin cells do contain a phosphoprotein[5] of the same relative molecular mass as synapsin I. But, unlike synapsin I, this phosphoprotein does not show clear calmodulin-dependent phosphorylation[5] and is in any case very much less abundant in chromaffin granules than is synapsin I in synaptic vesicles. Furthermore, denervation of the adrenal medulla results in almost complete disappearance of synapsin I from the gland[6], suggesting that it is present in nerve terminals in the adrenal medulla but not chromaffin cells, and immunocytochemical studies[7] fail to detect synapsin I in chromaffin cells.



Presence of a 4.1-like protein in bovine adrenal chromaffin cells. *a*, Immunoblotting with anti-red-cell 4.1 of an homogenate of chromaffin cells purified by differential plating after dissociation of adrenal medulla. Arrowheads, immunoreactive doublet; arrows, positions of relative molecular mass standards of 205,000 (205K), 116K, 94K, 67K and 43K (from top to bottom). Immunocytochemical localization of the 4.1-like protein using affinity-purified anti-red cell 4.1 by *b*, immunofluoresence or *c*, immunoperoxidase staining in chromaffin cells maintained in culture for 24 h showing intense staining at the cell periphery. Scale bars in *b* and *c*, 10 μm. The preparation and characterization of anti-red-cell 4.1 was described previously[8].

Synapsin I is functionally similar to, and shows immunological cross-reactivity with, red-cell protein 4.1 (ref. 8), which is involved in linking spectrin and actin filaments to the plasma membrane. Because of the similarity between the suggested role of the cytoskeleton in nerve terminals and chromaffin cells in restricting vesicle access to exocytotic sites before stimulation, we have examined the possibility that a 4.1-like protein is present in chromaffin cells instead of synapsin I. As shown in the figure, there is indeed an immunoreactive doublet of similar molecular mass (about 70,000) to red-cell