Anyons Superconduct, But Do Superconductors Have Anyons?

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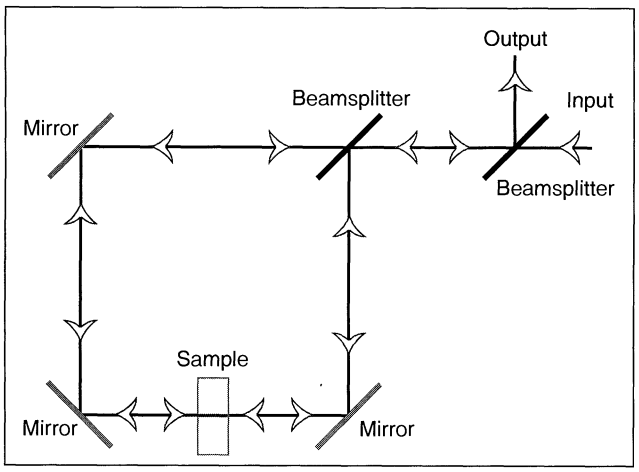
High-temperature copper-oxide superconductors achieve their remarkable properties in ways that are still mysterious. Although the topic has been intensely debated since the initial discovery of this class of layered ceramics 6 years ago, no microscopic mechanism has received a full vote of confidence. Among the more exotic proposals, that of "anyon" superconductivity (*1*) has attracted a great deal of attention. Anyons (*2*) are quantum particles that inhabit a shadow world between fermions (such as electrons) and bosons (such as photons), the two major categories known to physicists. Recent theoretical and experimental studies have begun to untangle the threads that might connect anyons to superconductivity.

Anyons can be viewed as ordinary bosons or fermions that act as if they each carry a tube of magnetic flux around with them. Wen and Zee (*3*) pointed out that these fictitious flux tubes lead to a crucial experimental test of the theory. Because of their peculiar physical attributes, anyons cause what physicists call "spontaneous symmetry breaking." In this case the symmetry being broken is that of time-reversal. Consequently, there was considerable excitement in the condensed matter physics community when two groups, Lyons *et al.* (*4*) and Weber *et al.* (*5*), reported signatures of broken time-reversal symmetry in YBaCuO superconductors.

Time-reversal symmetry is one of the fundamental principles of physics: the laws of motion of particles in mechanics and quantum mechanics do not distinguish between time going "forward" and time going "backward." To see what this means, consider an experiment in which a light beam is scattered from a collection of molecules. If we reverse the direction of propagation of the light and reverse the motion of all the particles, the results of the experiment will be exactly the same as before (with the roles of incoming and outgoing beams interchanged). Now in practice, we may not be able to reverse the motion of each molecule in (say) a gas. Imagine, however, making a movie of a gas that has its molecules moving randomly in equilibrium. If we view the movie, we will not be able to tell whether the film is being run forward or backward—in both cases we see molecules moving randomly in equilibrium and all macroscopic observables are the same. Such a system is said to be symmetric under time-reversal.

Sometimes, however, there is an observable difference when we reverse the direction of time. Consider a magnet that has a majority of its electron spins aligned in some direction. If we reverse time, the electrons spin in the opposite direction and the magnetization reverses. Thus there is an observable macroscopic difference. The laws of physics being time-reversal invariant implies that the two states of the magnet have exactly the same energy but the system has randomly selected one of the two states and is thus said to have "spontaneously broken" time-reversal (T) symmetry. Similarly, in a system that has no external magnetic field to bias the choice, anyons may occur in one of two time-reversed states of equal energy.



Rounding up anyons. Laser beams traveling in opposite directions around an optical gyroscope are used to detect time-reversal symmetry breaking. If anyons were present in the superconducting sample, the resulting phase shift could be detected in the output beam.

Imagine transmitting a beam of linearly polarized light through a (transparent) magnet. The direction of polarization of the light will, in general, be rotated during its passage through the sample. The direction of rotation will be determined by the direction of rotation of the electron spins and be independent of the direction in which the light is propagating.

Unfortunately there are spurious ways that the polarization of the light can be modified. For example, the molecules in the material might have an intrinsic handedness [broken parity (P) symmetry]: a sugar solution might contain predominantly dextrose instead of levulose. You might think that because the molecules are randomly oriented, the net effect will be zero. This is not the case however. Consider an ordinary threaded rod: the threads look right-handed from either end. As a result, the direction of rotation of polarization is always (say) "to the right" as viewed in the direction of propagation of the beam (just as the direction of rotation of a nut on a threaded rod is controlled by the direction of its motion). Hence, in a reflection rather than transmission experiment, broken P symmetry will have no effect because the rotations as the beam goes in and then comes back out will cancel. Broken T symmetry gives rotations that add (giving a net rotation angle of typically 10^2 to 10^4 microradians for a ferromagnet). This allows one in principle to distinguish P effects from T effects.

Other possible spurious effects include beam alignment errors, surface roughness, and most importantly, lack of rotational symmetry of the sample. Unfortunately, to make the sample effectively symmetric, one has to rotate the direction of the polarization continuously and do a time average over all possible polarization directions.

A remarkable new experimental technique that appears to be largely free of these experimental difficulties has been developed by Spielman *et al.* (*6*). The apparatus is a modified optical gyroscope in which a laser beam circulates around an optical fiber loop along with its counter-propagating (time-reversed) partner. A time-reversal non-invariant sample inserted into the beam path introduces a detectable phase shift between the two beams which is sensitive only to T- and not P-breaking. This experiment yielded a null result with a sensitivity of a few microradians although the sensitivity of the apparatus to T-breaking domain sizes smaller than about 5 μm may be considerably poorer, particularly if the domains have a well-defined periodic structure. Very recently Lawrence *et al.* (*7*) have produced an apparatus (and results) similar to that of Lyons *et al.* (*4*) but they have isolated (and found ways to correct for) several sources of spurious errors of the types mentioned above

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and after eliminating these, they also claim a null result with similar sensitivity. Meanwhile Lyons *et al.* (*8*) have further refined their apparatus and found yet other techniques to eliminate spurious errors. They continue to find, as originally reported, a signal that is zero on average but fluctuates from point to point on the sample. They interpret this as being the result of domains whose T signal fluctuates in sign.

There are possible practical loopholes in Wen and Zee's argument associated with the difficulty of actually computing for optical photon energies the [probably very small (*9*)] magnitude of T-symmetry breaking signal which presumably involves very low energy scales (on the order of $T_c \sim 10^{-2}$ eV). There is also the possibility of partial or complete cancellation of the effect in adjacent planes owing to a kind of antiferromagnetic ordering (*10*). Although the experimental situation remains somewhat controversial, the simplest interpretation at present is that sensitive optical and other (*11, 12*) measurements have failed to unambiguously detect signatures of anyons. It is now quite clear from theoretical work that anyons superconduct, but it is by no means clear from experiment that cuprates are superconductors because of anyons.

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MAP Kinases: Charting the Regulatory Pathways

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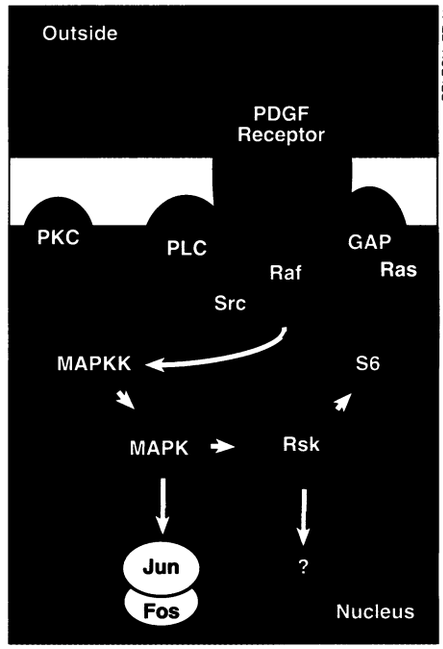
The intracellular transmission of growth factor signals is presumed to be mediated by sequentially activated protein kinases integrated into an elaborate network (*1*). Despite the identification of hundreds of distinct protein kinases, few interconnections between these kinases have been firmly established. Now at last, one of the major mitogenic signaling routes is finally coming into focus. The emerging picture is far more complex than what might have been anticipated even a few years ago (see figure).

It has long been suspected that protein kinase cascades emanating from growth factor receptors are responsible for the phosphorylation of such targets as ribosomal protein S6. Many extracellular mitogens, such as platelet-derived growth factor (PDGF), epidermal growth factor (EGF), and nerve growth factor (NGF), induce autophosphorylation of their respective receptors on tyrosine residues by activation of intrinsic catalytic kinase domains. This is ultimately translated into phosphorylation of serine and threonine residues in proteins throughout the cell. To chart the major routes for mitogenic signaling, the S6 protein has been employed as a convenient starting point for the elucidation of upstream regulatory events. Diverse stimuli can activate S6 protein kinases, in particular the 70-kD S6 kinase and a 90-kD kinase encoded by the *rsk* gene family (*1*). These S6 kinases are, in turn, activated by phosphorylation on serine and threonine residues. Multisite phosphorylation of $p90^{rsk}$ by the mitogen-activated protein (MAP) kinases, or extracellular signal–regulated protein kinases (ERKs) as they are occasionally called, is sufficient to markedly stimulate its S6 phosphotransferase activity (*2*). MAP kinases are probably the physiological activators of $p90^{rsk}$, since both are activated simultaneously in response to the same stimuli (*1*).

The MAP kinase family is composed of 40- to 46-kD isoforms that will phosphorylate myelin basic protein in vitro on a threonine residue (*3*). At least two of these isozymes, $p42^{mapk}$ and $p44^{erk1}$, require both threonine and tyrosine phosphorylation for maximal activation (*4*). The regulatory phosphoacceptor sites in murine $p42^{mapk}$ have been identified as Thr^{183} and Tyr^{185} (*5*), and these residues are conserved in $p44^{erk1}$. Both $p42^{mapk}$ and $p44^{erk1}$ can undergo autophosphorylation on tyrosine and become slightly activated (*6*). However, the kinetics of autophosphorylation and autoactivation are much too slow to account for the stimulation of these MAP kinases in vivo.

Rapid stoichiometric threonine and tyrosine phosphorylation of $p42^{mapk}$ and $p44^{erk1}$ and stimulation of their ability to phosphorylate myelin basic protein can be achieved by at least two "activating factors." These were first detected in cytosolic extracts from EGF-stimulated Swiss mouse 3T3 cells and subsequently in NGF-treated rat PC12 cells and maturing *Xenopus* oocytes (*7*). There is an excellent correlation between the stimulation of these activators and increases in activity of MAP kinases in these diverse systems. These activators do not simply accelerate MAP kinase autophosphorylation, but are bona fide MAP kinase (MAPK) kinases, since they induce the tyrosine and threonine phosphorylation of mutant $p42^{mapk}$ in which autophosphorylation has been almost completely eliminated by mutagenesis (*8, 9*).

Two human A431 cell MAPK kinases (*10*), their murine and rabbit cognates (*11*), and the *Xenopus* MAPK kinase (*7*)



Representation of mitogenic signal transduction from the PDGF receptor.

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