Breaking up a superfluid

Henk T. C. Stoof

Ultracold atoms held in a three-dimensional pattern by a web of light beams can now be switched from a superfluid to an insulating state. This achievement may be useful for performing quantum computations.

At extremely low temperatures of less than one-hundred-millionth of a degree above absolute zero (10 nanokelvin), the atoms in a rubidium gas essentially all join into a single quantum state to form a Bose–Einstein condensate. In such a condensate the atoms can flow without friction, and so the gas is a superfluid. When this superfluid is placed in an energy landscape consisting of high-energy mountains and low-energy valleys there is initially little effect (Fig. 1a). In particular, the superfluid nature of the gas does not change and the atoms still move freely from one valley to the next. But when the mountains become just a little too high the atoms suddenly lose their freedom, and each atom is trapped in a single valley (Fig. 1b). This surprising behaviour — switching a quantum gas from a superfluid to an insulating phase — has been demonstrated for the first time by Greiner *et al.*[1], as they report on page 39 of this issue.

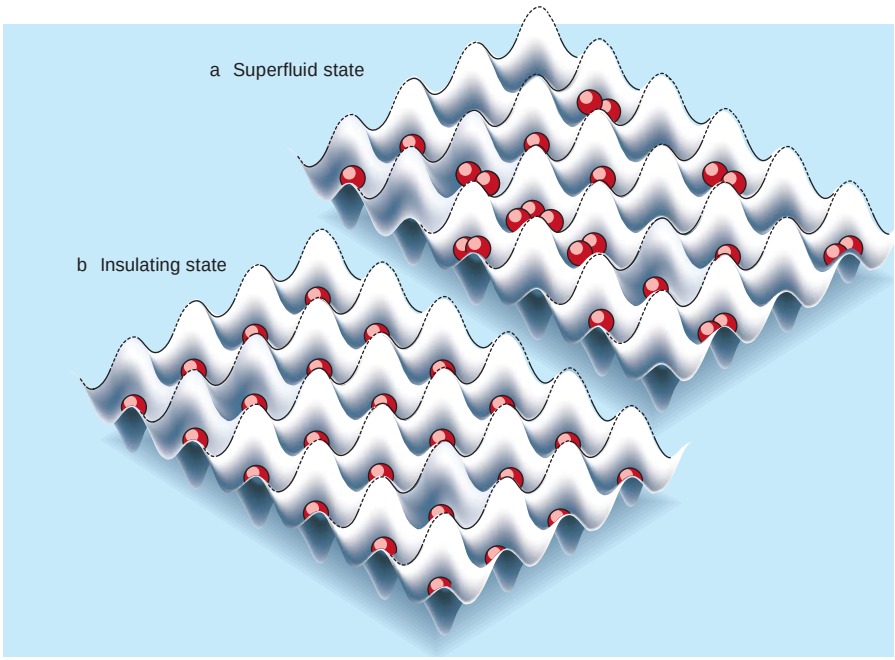

Figure 1 **A quantum phase transition in an ultracold gas. By using a web of laser beams to create an energy landscape of mountains and valleys (an optical lattice), Greiner *et al.*[1] can reversibly switch a gas of rubidium atoms from a superfluid to an insulating phase.** a, **At a temperature of 10 nanokelvin or less the rubidium atoms share the same quantum state and are in a superfluid phase, in which they can move freely between valleys.** b, **By increasing the intensity of the laser beams in the optical lattice, the researchers force the gas into an insulating phase, in which each atom is trapped in an individual valley. Such control is vital to most proposals for building a quantum computer.**

The experiment by Greiner *et al.*[1] represents a landmark in the history of Bose–Einstein condensed gases, for several reasons. The creation of a Bose–Einstein condensed gas of rubidium atoms in a magnetic trap is challenging in itself, and the first researchers[2] to do so in 1995 were recently rewarded with the 2001 Nobel prize for physics. Several laboratories around the world can now create Bose–Einstein condensates almost routinely, but Greiner *et al.*[1] also needed a periodic energy landscape for the atoms. This is most easily achieved using the electric field of a laser beam or, more precisely, several criss-crossing laser beams, which create a light-wave interference pattern known as an optical lattice.

Optical lattices have also been around since the mid-1990s, and a standard three-dimensional optical lattice is made using four laser beams, so the resulting lattice is less sensitive to instabilities in the lasers. But Greiner *et al.*[1] used six laser beams to create an optical lattice with an extra twist. With the additional laser beams they can make an energy landscape in which the distance between the valleys (and mountains) in all three directions is exactly the same — it is a perfect cubic lattice. Such experimental wizardry allows the authors to demonstrate convincingly the transition of the ultracold atoms from superfluid to insulating behaviour and back again. To confirm the state of the quantum gas at any instant they simply switch off the magnetic trap and the optical lattice, and then take an image of the gas cloud after it has expanded for 15 milliseconds. Only when the gas is a superfluid do they observe a beautiful interference pattern.

For a physicist, the observation of this transition is exciting because it is solely the result of the Heisenberg uncertainty principle. Such 'quantum phase transitions' have attracted a lot of attention in recent years, because they are fundamentally different from their more familiar 'classical' counterparts that are driven by thermal rather than quantum fluctuations. For example, true quantum phase transitions occur only at a temperature of absolute zero. So, in principle, thermal fluctuations will still have a pronounced effect on the transition[3], even at temperatures as low as 10 nanokelvin. In the case of a quantum gas in an optical lattice, understanding the effect of thermal fluctuations remains an important challenge. In this respect, theorists are particularly interested in the speed of sound in the gas near the transition point.

In its most familiar form, Heisenberg's uncertainty principle says that knowing precisely the position of a particle prevents you knowing precisely its momentum, and vice versa. What you gain on one hand you lose on the other. The uncertainty principle at work in Greiner *et al.*'s experiment[1] forbids simultaneously knowing the number of atoms in a certain valley, and the phase of the condensate's 'wave function' in that same valley. Because all the atoms in a Bose–Einstein condensate occupy a single quantum state, they are described by a quantum wave function whose phase is exactly the same in each valley. As a result, when the gas cloud is in the superfluid state the number of atoms in each valley can vary considerably. In the insulating phase the situation is reversed: the number of atoms in each valley is now fixed, so the phase of the wave function changes randomly from one valley to the next.

This particular quantum phase transition was first studied theoretically by Fisher *et al.*[4] in the context of granular superconductors and Josephson-junction arrays. It

was also predicted to occur for a quantum gas in an optical lattice by Jaksch *et al.*[5]. Their proposal has several advantages, as demonstrated by Greiner *et al.*, because there are no lattice imperfections (disorder) so the physics of the quantum phase transition occurs in its most ideal form. Another advantage of using a quantum gas in an optical lattice is that the height of the mountains in the energy landscape can easily be changed by varying the intensity of the laser fields. This makes it possible to switch back and forth between superfluid and insulating behaviour. For granular superconductors and Josephson-junction arrays it is essentially impossible to achieve the same control over the energy landscape because creating a new landscape requires a new sample. So one cannot actually see the superfluid–insulator transition taking place in a single system.

A tantalizing application for the ideal array of single atoms created in the insulating phase is quantum computing. Every rubidium atom has a magnetic moment and so has two internal states that may serve as the 0 and 1 of a quantum bit. Because there are a large number of rubidium atoms in the optical lattice, they can act as a memory for a quantum computer. Moreover, if there are two such memories that can be moved relative to each other, we can even make use of the interactions between the atoms to perform a quantum computation[6]. The first step towards this exciting goal has now been taken. ■

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Ageing

The price of tumour suppression?

Gerardo Ferbeyre and Scott W. Lowe

The p53 protein works to suppress cancer, so one might think that bumping up the levels of this protein would be a good idea. But this isn't so — mice with too much p53 age prematurely.

The p53 gene is often touted as 'the most frequently mutated gene in human cancer'. As a result, a great deal is known about it. When fully functional, p53 works to suppress the development of tumours, and current thinking suggests that it does so by affecting how cells respond to damage[1]. p53 can be activated by many stresses, such as breaks in DNA, and in turn induces responses that keep cell numbers down, including cell death (apoptosis) and permanent cell-division arrest (senescence; Fig. 1a). When p53 is mutated, cells cannot respond correctly to stress and are predisposed to becoming cancerous.

Indeed, mice that lack p53 develop normally but rapidly succumb to cancers[2], implying that, at the whole-organism level, p53 acts purely as a tumour suppressor (Fig. 1b). But on page 45 of this issue, Tyner *et al.*[3] identify an unexpected aspect of p53 biology. They show that mice engineered to have high p53 activity are resistant to tumours — but age prematurely. Their results raise the shocking possibility that ageing may be a side effect of the natural safeguards that protect us from cancer.

Tyner *et al.*'s findings[3] were serendipitous. They were trying to engineer mice with a particular mutation in p53, and instead produced animals in which a large portion at one end of the p53 gene (the 5′ end) was missing. The remaining portion, which they call the *m* version of the gene, is just the 3′ end and encodes a shortened protein. Mice with one normal copy of the p53 gene and one copy of the *m* version were resistant to tumour formation, but even relatively young animals resembled old normal mice. By contrast, mice with one non-functional p53 gene and the *m* gene were indistinguishable from mice that lack p53 altogether. So it seems that a normal p53 gene is needed for the *m* gene to have an effect, hinting that the protein encoded by the *m* gene steps up the activity of normal p53. This seems remarkable, yet is consistent with the observation[4] that short peptides encoded by the 3′ end of the p53 gene can activate normal p53.

It is not yet certain that the features of ageing are only due to the mutant p53 gene. The authors could not detect the truncated protein in the mutant mice, and at least one essential gene near the p53 gene was also deleted. Still, a second p53-mutant strain also shows signs of premature ageing[3]. Of course, the definitive experiment — showing that loss of p53 increases longevity — is impossible, because p53 and cancer are so intertwined.

Tyner *et al.*'s results might simply reflect a highly abnormal, diseased state. But they may also reveal a role for p53 in normal ageing. What might this role be? The evolutionary theory of ageing suggests that organisms invest resources to ensure reproductive success early in life, at the expense of longevity. It is not clear how this works at the molecular level. One model proposes that loss of the normal mechanisms for quelling gene expression leads to genes being expressed incorrectly and, ultimately, to ageing. So, enforced expression of the gene-silencer SIR2 extends lifespan in yeast and worms[5]. Another model holds that non-reproductive cells have a clock that monitors how often they have divided (based on the lengths of telomeres, protein–DNA complexes that cap chromosome ends and erode gradually with each cell division). Short telomeres eventually trigger senescence. Indeed, mice with critically short telomeres age prematurely[6].

In a third model, cellular damage leads to mutations or general cellular decline, ultimately producing the spectrum of age-related characteristics[7]. Support for this idea comes from the fact that radiation increases

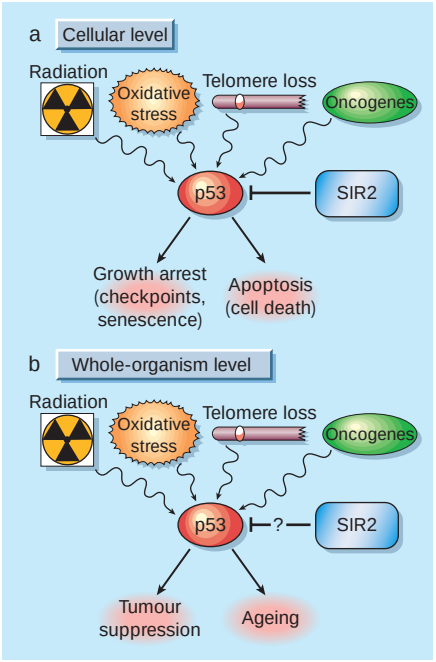

Figure 1 **The p53 system and ageing.** a, **At the cellular level several stresses can activate p53, inducing checkpoints in the cell-division cycle, permanent cell-division arrest (senescence) and cell death. Suppression of p53 activity prevents these responses, predisposing cells to becoming cancerous.** b, **At the whole-organism level, p53 activation results in a lower cancer incidence. But Tyner *et al.*[3] show that p53 can also promote ageing. The SIR2 protein, which increases longevity in experimental organisms such as yeast, can associate with p53 and suppress its activity in cultured cells (**a**). It is not known whether this process contributes to whole-organism ageing (**b**). Tyner *et al.*'s results suggest that aspects of ageing may arise as by-products of p53's tumour-suppressor activity.**