NEWS & VIEWS

QUANTUM PHYSICS

Indistinguishable from afar

Michael Fleischhauer

Imprinting a coherent light pulse on the spins of atoms is standard quantum sorcery. Retrieving the same light pulse from a second, distant set of atoms looks rather like black magic. But it, too, is just quantum mechanics.

In the quantum world, particles of the same kind are indistinguishable: the wavefunction that describes them is a superposition of every single particle of that kind occupying every allowed state. Strictly speaking, this means that we can't talk, for instance, about an electron on Earth without mentioning all the electrons on the Moon in the same breath. Fortunately, the existence of distant particles is for practical purposes inconsequential, and we can generally get away with ignoring them. On page 623 of this issue[1], however, Ginsberg, Garner and Hau show that two ensembles of atoms, known as Bose–Einstein condensates, can be made to reveal their indistinguishability, even when they are some distance apart. The condensates are admittedly not as far apart as Earth and the Moon, being separated by a fraction of a millimetre. But in the quantum world, that is a very large distance indeed.

To perform this trick, Ginsberg *et al.* employ a technique that was developed some years ago to 'store' a light pulse in an ensemble of atoms[2,3]. In the classical understanding of this process, an incoming laser pulse transfers its energy to a gas by inducing tiny, oscillating distributions of positive and negative electric charge — optical 'dipoles' — in the atoms. These dipoles radiate and quickly decay, so another laser is used to transfer the charge oscillations to oscillations in spin, which are more stable. When this control laser pulse is switched off, although the probe pulse disappears, its coherent information content is conserved in the spin oscillations of the atoms. If the control laser is then switched back on, this process is reversed[4]: the atoms radiate coherently, rather like an array of phased antennas, according to the phase of the original pulse imprinted in their spin oscillations. Light leaves the ensemble again as a coherent pulse with a well-defined shape and direction of propagation.

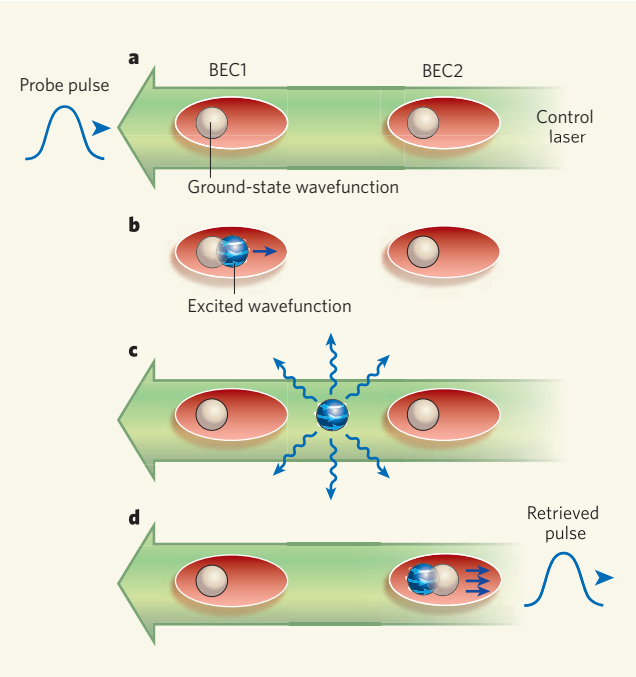

Figure 1 | Distant bounds. a, For their demonstration of quantum-mechanical indistinguishability on a macroscopic scale[1], Ginsberg, Garner and Hau use two ensembles of atoms known as Bose–Einstein condensates (BECs). With the help of a control laser, they store a probe-laser pulse in the first BEC with its atoms in their ground state. **b**, When the control laser is switched off, the probe-laser pulse imprints itself on the atoms of the condensate by exciting oscillating spin dipoles. The quantum-mechanical wavefunction of these dipoles consists of two superposed components corresponding to atoms in the ground and excited states. Momentum conservation requires that the excited-state wavefunction moves off as the excited atoms absorb their photons, but the ground-state wavefunction stays where it is. **c**, The initial pulse cannot be retrieved coherently while the spin-excited wavefunction is in transit between the two condensates; if the control laser is switched back on, only spontaneous, incoherent emission of photons occurs in all directions. **d**, If, on the other hand, the control laser is not switched back on until the excited-state wavefunction is within the second condensate, the original light pulse can be regenerated. The condensates were prepared independently so they would appear alien to each other. But they are composed of the same type of atoms in their ground state, and these are quantum-mechanically indistinguishable objects.

Quantum mechanically, spin dipoles are a superposition of the ground state and a spin-excited state of the atoms. The simultaneous existence of both components allows phase information to be stored, and is thus crucial to the coherence of the retrieval process. If only the spin-excited state were occupied, the atomic dipoles would oscillate with random phases, and when the control laser was switched on again it would induce only the spontaneous emission of incoherent light.

It is important to recall here that it is only atoms in the spin-excited state that have actually absorbed a photon. This is significant because, although the atoms of a Bose–Einstein condensate are very cold, they can move freely, and the process of storing the light pulse in the atomic ensemble is accompanied by a transfer of photon momentum to the excited atoms. As a result, the spin-excited part of the wavefunction starts to move away from its original position with the recoil velocity of the photon, whereas the ground-state part stays where it is. In a condensate, spin oscillations live long enough for the spin-excited component to travel over distances much larger than its own spatial extent. This leads to the question: might a coherent retrieval of the initial light pulse be possible even when the two components of the wavefunction — spin-excited and ground state — have become separated in space?

Ginsberg and colleagues[1] show that this can indeed be the case — at least as long as the wavepacket of the spin-excited states does not leave the condensate. This is because, in a Bose–Einstein condensate, the wavefunction of the ground state extends over the whole condensate. There is thus always a spatial overlap between it and the spin-excited wavefunction, and the retrieval process is always coherent.

But it is in a second experiment that the authors made their most astonishing observation. They prepared two independent

Bose–Einstein condensates in two traps, each extending over about 50 μm and separated by a fraction of a millimetre. A coherent light pulse was stored in the first ensemble, in the conventional way (Fig. 1a). A wavepacket of spin-excited atoms was created, which travelled, as before, with photon recoil velocity in the direction of the second condensate (Fig. 1b). Once this 'messenger' wavepacket had left the first condensate, a coherent regeneration of the original light pulse was no longer possible (Fig. 1c) — as expected.

But a strange thing happened if the time of retrieval was chosen to be much later — late enough for the messenger wavepacket to have reached the second condensate. Under these circumstances, the initial light pulse could indeed be regenerated, with well-defined shape and direction of propagation (Fig. 1d). The two condensates had been independently prepared, and so from a naive point of view the messenger wavepacket transferred from the first condensate should be completely alien to the atoms of the second. The only way to understand the experimental observation is to consider the atoms in both condensates as indistinguishable quantum objects. As such, the ground-state wavefunction would have a component in both traps simultaneously, and could thus combine with the spin-excited messenger component, once this had passed between the condensates, to coherently retrieve from the second condensate the light pulse stored in the first.

The work of Ginsberg *et al.*[1] is a striking and intriguing demonstration of a fundamental aspect of quantum physics — indistinguishability. But it also shows that we are entering a state of unprecedented experimental control of coherent light and matter waves. That could bring very real technological benefits: applications that spring to mind include quantum-information interfaces that allow the transfer of a quantum bit encoded in a photon to a single atom, as well as ultra-sensitive rotation sensors and gravity detectors. ■

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CANCER BIOLOGY

Gone but not forgotten

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The p53 tumour-suppressor protein is a cell's principal guardian against cancer. Most cancers eliminate p53 — but it seems that its pathway remains intact, so resurrecting it might provide a cancer therapy.

Once fully established, many cancers rely for their maintenance on the persistent activation of certain cancer-promoting genes (oncogenes)[1,2]. Drug developers hope to exploit this dependency, as it makes tumours vulnerable to inhibitors of the associated oncogenic proteins. Indeed, several such inhibitors have already proven to be effective cancer treatments. Three papers[3–5] now confirm an analogous idea for a tumour-suppressor protein that limits cancer growth*. The work reveals that persistent inactivation of the p53 tumour-suppressor pathway is similarly required for tumour maintenance, and thus opens new therapeutic avenues against cancer.

Cancer is the outcome of many mutations, each endowing tumour cells with more of the biological requirements for the full-blown disease. With so many genetic aberrations present in each cancer, it is difficult to distinguish the primary 'driver' events that trigger the cancer from secondary 'passenger' alterations. Moreover, even if the driver mutations are known, understanding how these lesions interact to induce cancer is a formidable task. Which mutations are crucial to the maintenance of an established malignancy, for instance? And which are required only during the initial genesis and progression of the cancer? Identification of the key 'targets' required for tumour maintenance has been a major challenge in drug development[6].

The work by Ventura *et al.*[3] and Xue *et al.*[4], on pages 661 and 656 of this issue respectively, and by Martins *et al.*[5], published in *Cell*, concentrates on the p53 protein. This tumour suppressor is one of the heavyweights of cancer biology, as direct inactivation of the gene that encodes it is the most common mutation in human cancer, and the pathway that it controls is probably compromised to some degree in all human cancers. This pathway normally helps the cell to respond to DNA damage, such as that caused by radiation or carcinogenic chemicals. DNA damage activates p53, which in turn induces the expression of proteins that halt the cell-division cycle to allow for repair. Activation of p53 can also initiate programmes of cell death (apoptosis) or permanent growth arrest (senescence) if the DNA damage is persistent and severe.

The latest studies describe three distinct genetic approaches to producing mice that lack p53 function, either by inactivating the gene encoding p53, or by interfering with production of the protein. Mice lacking p53 are highly prone to spontaneous and carcinogen-induced tumours. The new twist here is that these mice are engineered so that the dormant p53 gene can be reawakened in tumours by treating the animals with a particular chemical. Importantly, despite the different technical approaches and tumour types in the three studies, the reinstatement of p53 expression led universally to a prompt and impressive regression of established, *in situ* tumours.

The mere synthesis of p53 in a cell is not in itself sufficient to suppress tumours — the protein must also be stabilized and switched on, something that does not generally occur in normal cells. All three studies[3–5] found that restoring expression of p53 caused tumour regression. This indicates that there is some feature of a cancer that is sufficient to activate p53, and that this reinstatement of physiological p53 function is enough to halt tumour growth. Although the re-establishment of p53 expression in tumours may be even more effective when combined with stimuli that stabilize and/or activate p53 function (such as radiotherapy), these papers show that, even without such stimuli, tumour cells harbour the signals that can trigger the destructive power of p53 if it is present. We believe that this general conclusion — that established tumours remain persistently vulnerable to p53 tumour-suppressor function — is the most significant finding of the work.

How p53 carries out its anticancer function seems to differ according to the tumour type and its context. For example, reinstating p53 function in p53-deficient lymphomas (blood cancers) rapidly induces apoptosis[3,5]. By contrast, p53 reactivation in two types of solid tumour (soft tissue sarcoma and hepatocellular carcinoma) induces a potent growth arrest featuring hallmarks of cellular senescence[3,4]. Senescence is involved in suppressing the early steps of cancer development in several tissues (reviewed in ref. 7), and Ventura *et al.*[3] and Xue *et al.*[4] establish that ongoing resistance to senescence is also needed to maintain established tumours. It is not clear which features of a cancer determine whether its response to p53 activation is apoptosis or senescence. But both outcomes are associated with tumour regression and so could be of therapeutic benefit.

The tumour regression seen in the sarcomas and hepatocellular carcinomas was associated with senescence without apoptosis — but if there is no cell death, how does the tumour get smaller? Xue *et al.*[4] report an unexpected cause

*This article and the *Nature* papers concerned[3,4] were published online on 24 January 2007.