NATURE|Vol 438|8 December 2005


50 & 100 YEARS AGO

## 50 YEARS AGO

Over the past twenty years I have been interested in the possibility of using X-ray crystallographic methods to find the arrangement of the atoms in protein molecules and particularly in insulin. One of many possible approaches to solving this problem seems to be the crystallographic study of naturally occurring peptides such as the gramicidins and tyrocidine... These all have molecules much smaller in size than even the smallest protein molecules; some indeed are smaller than vitamin $B_{12}$, of which we have already found it possible to obtain the kind of information we require... We already have evidence that there may be a connexion between the way the peptide chain is folded in gramicidin *S* and the way it is folded in part of the molecule of insulin. But even if later we find that the connexion in chain configuration is less close than we at present suppose, we think that the atomic arrangement in these peptide molecules is itself of great interest and some importance.

**Dorothy Crowfoot Hodgkin**

From *Nature* 10 December 1955.

## 100 YEARS AGO

**The death-knell of the atom**[1]

Old Time is a-flying; the atoms are dying;
Come list to their parting oration:—
"We'll soon disappear to a heavenly sphere
On account of our disintegration.

"Our action's spontaneous in atoms uranious
Or radious, actinious or thorious:
But for others, the gleam of a heaven-sent beam
Must encourage their efforts laborious.

"For many a day we've been slipping away
While the savants still dozed in their slumbers;
Till at last came a man with gold-leaf and tin can
And detected our infinite numbers."

[1]Sung at the Chemical Laboratory dinner at University College, November 17.

From *Nature* 7 December 1905.

beam is turned off, the medium becomes opaque once again, and any photon inside it is trapped, converting into another atomic excitation (known as a dark-state polariton). The photon can be regenerated at any time within the coherence time of the ensemble, simply by turning the EIT laser beam on again.

Throughout this sequence of events, it is clearly essential to check that the photon maintains its quantum, particle-like properties. One way to do this is to put a beam-splitter in the photon's way, and verify that photon counts on the two paths after the beam-splitter are anticorrelated. Correlated counts would indicate that the incoming beam splits in two, a clear sign of classical, wave-like behaviour. The degree of photon splitting can be conveniently characterized[7] by a parameter $\alpha$, with an ideal single photon (exhibiting a purely quantum behaviour) having $\alpha = 0$, and a classical source having $\alpha > 1$. A value of $\alpha$ between 0 and 1 thus corresponds to a light beam showing a mixture of quantum and classical behaviours, or in other words, to an imperfect single photon. Chanelière and colleagues[1] obtain a value for $\alpha$ of 0.36 after a storage time of 500 nanoseconds, whereas Eisaman and colleagues[2] find a value of 0.51 under EIT conditions, but without storage (they also observe storage, but without evidence that $\alpha$ is less than 1).

Obviously, the 'quantum memories' (the ability to 'regenerate' a photon stored in an ensemble after a delay) described in these articles[1,2] are not the end of the story. First, what has to be stored and released is not a photon, but a qubit — the quantum information encoded on a photon. In the context of the DLCZ proposal, how to store and release a qubit is known in principle, and preliminary results have been obtained[8]. Another crucial issue is that it should be possible to create some entanglement between distant atomic ensembles[9], as discussed also by Chou *et al.* in this issue (page 828)[10]. The next key step will be to gradually increase the entanglement between the two remote memories — the process of 'entanglement distillation', which would be the fundamental duty of a quantum repeater[3,4]. This is a long-term goal, as many features have to be improved: counting rates (presently much too low), storage times (presently much too short), and the fidelity of the successive transfer processes in the ensembles. Although this looks more like mountain climbing than highway driving, new ways upwards keep on opening, as the present research[1,2] shows. The summit may seem far off, but it is not out of reach. ■

Philippe Grangier is at the Laboratoire Charles Fabry de l'Institut d'Optique, 91403 Orsay Cedex, France.
e-mail: philippe.grangier@iota.u-psud.fr

1. Chanelière, T. *et al. Nature* **438,** 833–836 (2005).
2. Eisaman, M. D. *et al. Nature* **438,** 837–841 (2005).
3. Bennett, C. H. *et al. Phys. Rev. Lett.* **70,** 1895 (1993).
4. Briegel, H.-J., Dür, W., Cirac, J. I. & Zoller, P. *Phys. Rev. Lett.* **81,** 5932–5935 (1998).
5. Duan, L.-M., Lukin, M., Cirac, J. I. & Zoller, P. *Nature* **414,** 413–418 (2001).
6. Felinto, D. *et al. Phys. Rev. A* **72,** 053809 (2005).
7. Grangier, P., Roger, G. & Aspect, A. *Europhys. Lett.* **1,** 173–179 (1986).
8. Matsukevich, D. N. *et al. Phys. Rev. Lett.* **95,** 040405 (2005).
9. Matsukevich, D. N. *et al.* preprint available at www.arxiv.org/quant-ph/0511012 (2005).
10. Chou, C. W. *et al. Nature* **438,** 828–832 (2005).

CANCER BIOLOGY

# Emissaries set up new sites

Patricia S. Steeg

**The capacity of tumours to spread to other organs is one of their most dangerous attributes. A study of how cancer cells settle in new places shows that they send out envoys to prepare the ground for them.**

During the process of metastasis, tumour cells move from the primary tumour to colonize another organ. But why do these mobile cells put down roots only in particular organs, or only at specific sites within an organ? The lungs and liver, for example, seem particularly popular secondary targets for tumour cells. Some studies imply that this 'preference' might occur because, as they branch out within those organs, the blood vessels become very narrow, and the blood-borne tumour cells are trapped when they enter the fine capillary beds[1]. Other work has identified proteins that are specific to the cells lining the capillaries of certain tissues as possibly promoting metastasis formation[2]. A report from Kaplan *et al.* (page 820 of this issue)[3] provides another explanation. The authors show that tumour cells can mobilize normal bone-marrow cells, causing them to migrate to particular regions and change the local environment so as to attract and support a developing metastasis.

Metastasis is a sequential process, contingent on tumour cells breaking off from the primary tumour, travelling through the bloodstream, and stopping at a distant site. At the new site, the cells establish a blood supply and can grow to form a life-threatening mass. Both stimulatory and inhibitory molecular pathways within the tumour cell regulate this