



Figure 2 The common ancestor of modern humans and African apes, including gorillas, bonobos and chimps (pictured), may have used a form of locomotion called 'knuckle-walking' when on the ground.

of fossil morphology in terms of function, especially locomotion. Ever since Lucy was discovered in the late 1970s, there has been debate about the locomotor repertoire of *A. afarensis*, the species to which she is assigned.

The basic facts are not in dispute. *A. afarensis* has a combination of traits that is not seen among living primates. In some respects, *A. afarensis* is quite human-like (for instance in the foot structure, non-opposable big toe, and pelvis shape). In others, it is quite ape-like (relatively long and curved fingers, relatively long arms, and funnel-shaped chest).

What can be made of these features? For some researchers, the ape-like characteristics of *A. afarensis* are non-functional retentions from the common ancestor of hominids and the African apes. Here, emphasis is put on the human-like characteristics, and *A. afarensis* is seen as a hominid that walked on two legs and got about in no other way⁹. For others, the ape-like traits are functionally important, and *A. afarensis* is interpreted as using a 'mixed' locomotor repertoire, in which a form of terrestrial bipedalism was combined with an ability to move around effectively in trees¹⁰.

Richmond and Strait add a twist to this debate. They propose that the knuckle-walking features of *A. afarensis* are non-functional retentions from the common ancestor of hominids and African apes. This seems an entirely reasonable position, given that *A. afarensis* shows many traits that are thought to be associated with bipedal locomotion. The alternative idea — that *A. afarensis* combined knuckle-walking, bipedalism and climbing — is somewhat counterintuitive, because it implies the use of two entirely different modes of terrestrial locomotion.

By the same token, however, Richmond and Strait's argument undermines the idea

that *A. afarensis* combined bipedalism with climbing, which many researchers have hitherto considered to be the best interpretation of the evidence. Can we assert that one set of ape-like characters indicates that *A. afarensis* was an able climber, while at the same time arguing that another, equally good, set of ape-like characters is indicative of nothing except the phylogenetic history of *A. afarensis*? If the knuckle-walking characters are considered to be primitive retentions, must not the same hold for the other ape-like characters?

Are our only choices to accept that *A. afarensis* was a striding biped with a large number of non-functional primitive retentions, or to have to take seriously the counterintuitive idea that the locomotor repertoire of *A. afarensis* included forms of bipedalism, climbing and knuckle-walking?

It is difficult to predict how palaeo-anthropologists will react to Richmond and Strait's study. But one thing is certain. It will encourage many researchers to reconsider their assumptions about the phylogenetic and functional implications of bone shape and size in the primates, and most especially in the early hominids.

Mark Collard and Leslie C. Aiello are in the Department of Anthropology, University College London, Gower Street, London WC1E 6BT, UK. e-mails: m.collard@ucl.ac.uk
l.aiello@ucl.ac.uk

1. Richmond, B. G. & Strait, D. S. *Nature* **404**, 382–385 (2000).
2. Ruvolo, M. *Mol. Biol. Evol.* **14**, 248–265 (1997).
3. Andrews, P. & Martin, L. B. *J. Hum. Evol.* **16**, 101–118 (1987).
4. Dainton, M. & Macho, G. A. *J. Hum. Evol.* **36**, 171–195 (1999).
5. Pilbeam, D. R. *Mol. Phyl. Evol.* **5**, 155–168 (1996).
6. Clarke, R. & Tobias, P. V. *Science* **269**, 521–524 (1995).
7. Berger, L. R. & Tobias, P. V. *J. Hum. Evol.* **30**, 343–348 (1996).
8. McHenry, H. M. & Berger, L. R. *J. Hum. Evol.* **35**, 1–22 (1998).
9. Latimer, B. & Lovejoy, C. O. *Am. J. Phys. Anthropol.* **78**, 369–386 (1989).
10. Susman, R. L., Stern, J. T. & Jungers, W. L. *Folia Primatol.* **43**, 113–156 (1984).

Quantum optics

Tricks with a single photon

Peter Zoller

Laser light can trap atoms. But can the light field of a single photon hold an atom in free space? Because it was the development of the laser as an intense light source that made optical trapping and cooling of atoms practical, this seems to be an essentially impossible task. But now a group from the Max-Planck Institute for Quantum Optics in Munich headed by G. Rempe reports an experiment¹ (on page 365 of this issue) in which a single photon stored in a microcavity produces an optical force sufficient to trap an atom. Together with the related experiments on trapping and cooling of single atoms in optical cavities by H. J. Kimble's group at Caltech², these recent advances mark a new generation of cavity quantum electrodynamics (QED) experiments^{1–5}. Such work opens up exciting new

avenues for basic research in quantum physics with single atoms and photons, and suggests fascinating future applications in quantum information processing.

When a laser beam is focused, an atom can experience an optical force so that it is attracted to the centre of the light focus. The same concept underlies 'optical tweezers', which are used to manipulate much more macroscopic particles than atoms with light fields. For intense laser beams, the optical force can be strong enough to overcome gravity. The stronger the light beam, the deeper is the optical potential that holds the atom. In addition, for an atom to remain bound in the laser focus, its thermal energy must be sufficiently small that it cannot escape. In fact, the atoms must be extremely cold in comparison with gases at room

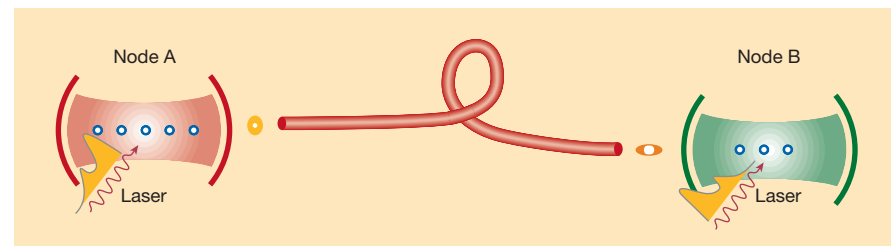


Figure 1 A quantum network based on cavity quantum electrodynamics (QED). Quantum communication channels connect the spatially separated nodes. Each node is a quantum processor that stores and processes quantum information locally. Atoms in the cavity provide the quantum memory, laser pulses play the role of quantum gates by exchanging cavity photons, and the cavities themselves become optical interconnects with a fibre linking the cavities. Exchange of information between the nodes of the network is accomplished by way of quantum channels. New techniques to confine atoms within optical cavities^{1–3} bring such quantum networks closer to reality.

temperature, and it was only the development of laser cooling techniques during the past two decades, allowing atoms to be cooled to microkelvin temperatures, that made optical trapping of atoms possible. These achievements were honoured with the award of the 1997 Nobel Prize in Physics to Chu, Cohen-Tannoudji and Phillips.

Quantum physics teaches us that the light field is quantized; that is, light comes in energy units of $E = h\nu$, where h is Planck's constant and ν is the light frequency. These light quanta are called photons. The granular quantum structure of light becomes visible only when we deal with extremely weak light fields consisting of a few photons. In this sense, a single photon represents the weakest possible light field. So how can the light intensity of a single photon be sufficient to trap an atom? The key idea is to store the photon in an optical cavity consisting of two high-quality mirrors. The smaller the volume in which we confine the photon, the larger is the energy per volume, and it is the corresponding strong electric field that is seen by the atom and determines the optical force. Trapping an atom with a single photon can thus be achieved using microcavities with extremely high-quality optical mirrors, where the distance between the mirrors is only ten or twenty times the optical wavelength. This is the basis of the Max-Planck and Caltech experiments¹⁻³.

How are atoms actually captured inside the cavity, and how do we know that it is there? The cavity mirrors are not perfect, and a single photon will be lost from the cavity after a short time. These photons must therefore be continuously replenished by a pumping laser that puts the photon back into the cavity. But a unique feature of such cavity QED experiments is that the light field leaking out of the cavity allows us to observe the motion of the atom in real time inside the cavity. So we can 'see' an atom entering the cavity, and this enables a feedback loop to change the parameters guiding the laser, which can then capture the atoms.

The latest developments in cavity QED with optical and microwave photons should be seen in a much broader context. An important new direction in modern physics has been the attempt to control processes in individual quantum systems on a quantum by quantum level. One of the long-term hopes of these endeavours is to develop the ability to assemble quantum devices as composite controllable quantum systems. Quantum optics has taken a leading role in this area, and cavity QED and laser-cooled trapped ions are its prime examples. The recent achievements in cavity QED¹⁻⁵ and ion traps⁶ now set the stage for future developments, ranging from basic studies of quantum physics all the way to possible applications.

Among the most exciting prospects are

the possibilities offered by cavity QED to build quantum information processors and to connect these devices in an optical quantum network⁷. Quantum computers require the storage of information in a set of two-level systems (called quantum bits or qubits), the processing of this information using quantum gates, and a final readout by a quantum-state measurement.

In a typical cavity QED set up, long-lived internal states of atoms can play the role of reliable quantum memory for storing the qubits. Quantum operations on a single qubit can be performed by coupling two atomic states with laser pulses. Joint operations on two qubits require the controlled exchange of a cavity photon between two atoms, where the cavity mode plays the role of a quantum data bus. This mechanism for a two-bit gate relies directly on the strong coupling of single photons with single atoms.

Although isolated atoms represent an ideal quantum memory, optical photons propagating in fibres are a fast and reliable

way to transmit quantum information to distant nodes of a network (Fig. 1). Cavity QED provides a natural setting for the interface between atoms and photons in the system in the form of optical interconnects. The primary technical obstacle to achieving these goals is the need to develop techniques to confine atoms within cavities. The recent experiments by the Caltech and Max-Planck groups are important milestones in this direction. ■

Peter Zoller is at the Institute for Theoretical Physics, University of Innsbruck, 6020 Innsbruck, Austria.

e-mail: peter.zoller@uibk.ac.at

1. Pinkse, P. W. H., Fischer, T., Maunz, P. & Rempe, G. *Nature* **404**, 365–368 (2000).
2. Hood, C. J., Lynn, T. W., Doherty, A. C., Parkins, A. S. & Kimble, H. J. *Science* **287**, 1447–1453 (2000).
3. Ye, J., Vernooij, D. W. & Kimble, H. J. *Phys. Rev. Lett.* **83**, 4987–4990 (1999).
4. Nogues, G. *et al.* *Nature* **400**, 239–242 (1999).
5. Varcoe, B. T. H., Brattke, S., Wedinger, M. & Walther, H. *Nature* **402**, 743–746 (2000).
6. Sackett, C. *et al.* *Nature* **404**, 256–259 (2000).
7. Cirac, J. I., Zoller, P., Kimble, H. J. & Mabuchi, H. *Phys. Rev. Lett.* **78**, 3221–3224 (1997).

Neurodegenerative diseases

Parkinson's pathology in a fly

Christian Haass and Philipp J. Kahle

Human life expectancy has increased dramatically over the past century. But can we really enjoy our extra years? Diseases such as Alzheimer's and Parkinson's, characterized by the death of certain nerve-cell populations and consequent dementia or movement defects, threaten more and more elderly people in the Western world. Many of the molecules behind these diseases have been identified, but we still lack a coherent understanding of the processes involved. On page 394 of this issue¹, however, Feany and Bender offer a new way to tackle the problem. They have developed a model of Parkinson's disease in the fruitfly *Drosophila melanogaster* that reproduces many of the features of the human disorder. The hope is that, with this model, we can learn much about the disease and, perhaps, new ways to treat it.

Parkinson's disease is the second most common neurodegenerative disorder. The pathological hallmark of the disease is the accumulation of fibrous protein deposits in neuronal cytoplasm (Lewy bodies) and nerve fibres (Lewy neurites) in the brain. These deposits may interfere with normal neuronal function. Selective death of the neurons that normally secrete the neurotransmitter dopamine results in a movement disorder that is characterized by muscle rigidity and resting tremor. The neurons that are affected in patients with Parkinson's disease are found in the regions of the brain

called the substantia nigra and the locus coeruleus (and form the nigrostriatal dopamine system).

Mice, flies and worms that have been engineered to express human genes linked to Alzheimer's disease have already yielded invaluable insight into the molecular mechanisms underlying this disorder², and have even offered exciting possibilities for therapy³. Until recently, it was difficult to take the same tack for Parkinson's disease, as genes linked to its onset or progression were unknown. Three years ago, however, by studying an Italian-Greek family in which Parkinson's disease was inherited, Polymeropoulos *et al.*⁴ identified a critical mutation in the gene encoding α -synuclein. This mutation resulted in alanine being replaced by threonine at amino-acid position 53 of the protein (A53T mutation). A second mutation, resulting in the substitution of alanine with phenylalanine at position 30 (A30P mutation), was later identified in a German family by Krüger *et al.*⁵ (Fig. 1a, overleaf).

Interestingly, α -synuclein, a phosphorylated protein⁶ normally found in presynaptic neurons⁷, is the main component of Lewy bodies⁸ (Fig. 1b, overleaf). Ablation of the gene encoding α -synuclein in mice results in functional deficits of the nigrostriatal dopamine system⁹, providing further evidence for a connection of α -synuclein with Parkinson's disease. In familial Alzheimer's disease, mutations in several distinct genes