an impediment to light supply would be a disadvantage for the host plant, but the effects of lichen cover on leaf surfaces have never been quantified. Affected leaves might be able to adjust to the new conditions of shading, by biochemical means and by structural alterations, such as the movement of chloroplasts to new locations within the cell[5]. For most tropical rainforest leaves, the lichen coverage increases gradually as they age. So leaves might be able to adapt to the new light conditions and retain their productive potential.

Anthony and colleagues[1] have studied lichens on the leaves of the palm *Calamus australis* in northern Queensland, Australia. Their first concern was to determine precisely how light conditions alter for a host leaf as a consequence of developing lichen cover. They found that light interception values for lichens had a mean of 48%, so the lichens effectively halved the amount of light reaching the host leaves. But did the lost light lie within the wavelengths required for photosynthesis by the host? It did — the light absorbed by the lichens had a similar spectral composition to that absorbed by the host leaf.

However, the possibility remained that compensation could occur by adjustment of a leaf's photosynthetic systems. Anthony *et al.* investigated this by examining the relationship between light intensity and carbon gain by photosynthesis in parts of the leaf either covered by or clear of lichens. They found that the two areas behaved quite differently. The part covered by lichens showed characteristics of a classic 'shade leaf', having a capacity to fix carbon effectively even at very low levels of irradiance. Respiration rates and the light compensation point (the irradiance level at which carbon fixation balances respiratory demand) remained unaltered, but the more efficient use of low light levels showed that lichen-covered parts of the leaf had acclimated to the new conditions. Checking the chlorophyll levels in different parts of the leaves revealed that the shaded portions had increased concentrations, although the ratio of the two main types of chlorophyll (*a*/*b*) remained the same. The gradual accretion of lichen cover during the lifetime of a leaf evidently allows this adjustment to take place, and local photosynthetic efficiency can thereby be maintained. Modelling the total carbon gain for entire leaves, Anthony *et al.* found that the general carbon economy of the host was not greatly affected by the presence of the epiphylls.

These findings would appear to resolve the physiological conundrum, first described for coffee plants[3], in which increased epiphyte shading resulted in no loss of productivity. But other questions remain, such as those relating to coevolution of organisms within rainforest and the consequent high biodiversity of this habitat. The leaf can

adapt to the lichen epiphyte, but has the lichen also adapted in a way that will ensure its host's survival? For example, the fact that the lichen steals only half of the available light could be the result of a trade-off between further productivity by the epiphyll and the consequent danger of reducing the productivity of the host leaf and possibly destroying it. Then there is the question of why leaves with a particularly long life-span generally have low levels of epiphylls[4]. Is the development of some chemical or structural defence mechanism by these highly persistent leaves an alternative strategy to that of appeasement? ■

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Attophysics

Ultrafast control

Philip H. Bucksbaum

The successes of two pioneering groups are now brought together to create trains of identical ultrashort laser pulses that can control what's happening inside an atom.

One attosecond is one-millionth of one-millionth of one-millionth of a second. If the next second of your life were expanded to fill the history of the Universe, then one attosecond would correspond to less than one second of that history. In one attosecond (10^{-18} s), light travels slightly further than the length of a single water molecule, and the molecule itself appears utterly frozen. The natural vibrations of a molecule take place over tens of thousands of attoseconds, and rotations take millions of attoseconds — a relative eternity. Even the rapid motion of atomic electrons is typically measured in hundreds to thousands of attoseconds; and a single oscillation of a wave of visible light lasts nearly 2,000 attoseconds.

The title, then, of the report by Baltuška *et al.*[1] on page 611 of this issue — "Attosecond control of electronic processes by intense light fields" — is impressive indeed. The authors claim to have harnessed light on an attosecond scale, and used the light to control electrons with similar precision. This

work combines the efforts of two creative research groups in optical science: the precision measurements of Theodor Hänsch's group at the Max Planck Institute for Quantum Optics, and the ultrafast-laser expertise of Ferenc Krausz's group at the Technical University of Vienna. Krausz, Hänsch and their colleagues have achieved this dizzying feat by delicately stacking together two techniques that have grown out of their research into ultrafast-laser technology: the production of subfemtosecond soft X-ray pulses[2], and the production of an all-optical-frequency standard[3].

Both of these achievements are based on the Kerr-lens mode-locked laser, the workhorse of ultrafast science, invented only a dozen years ago and now found in most of the world's ultrafast-research laboratories[4]. It produces a continuous train of 10–100-femtosecond pulses (10,000–100,000 attoseconds) in the near-infrared region of the optical spectrum, through a fortuitous nonlinear effect known as the Kerr lens (named
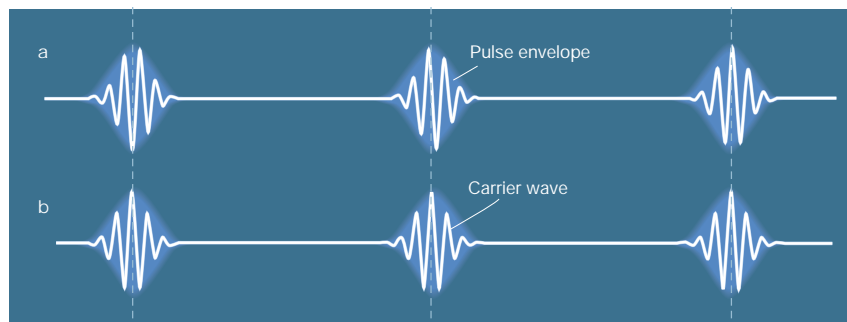

Figure 1 **Locking onto the laser frequency. a, The output of a typical ultrafast laser, based on the Kerr lens, forms a train of non-identical pulses: the optical carrier wave inside each pulse is shifted with respect to the pulse envelope, because its frequency is not an integer multiple of the pulse repetition frequency. b, If, however, the laser frequency is stabilized (using an optical comb generator**[3]**), truly identical pulses are created.**

after the Scottish physicist John Kerr, 1824–1907). The lens is created by the light itself, and forces the photons in a laser cavity to bunch up into extremely short pulses. These pulses can be thought of as electromagnetic waves confined in a single short envelope, bouncing back and forth between the end mirrors of the laser cavity. If one of the end mirrors is partly transmitting, a small portion of the pulse leaks out on each round trip, creating a train of output pulses.

But, although transmitted through the mirror on successive round trips, the pulses in the train do not look identical. The optical carrier wave appears shifted by some fraction of a cycle on every bounce (Fig. 1a). This is because the carrier frequency is not commensurate with the pulse repetition frequency — that is, the ratio of the optical frequency to the repetition frequency is not an integer. If the ratio were an integer, then the optical frequency could be simply related to the round-trip frequency.

Several years ago, Hänsch realized that if this defect could be overcome, it would be possible to build all-optical-frequency standards, which would significantly simplify and improve the precision of time measurement. His group found a way to do this, and the result, known as the 'optical comb generator', is revolutionizing the time standard. The details involve 'holy fibres', self-phase modulation and second-harmonic generation, a fascinating story which has been told in a previous issue of *Nature*[3]. Suffice to say here that Hänsch learned how to make a Kerr-lens laser in which every pulse in the train is truly identical (Fig. 1b).

Meanwhile, Krausz's group was pursuing a different application of Kerr-lens ultrafast lasers. They amplified light pulses only a few cycles long to peak powers of 100 billion watts, to create bursts of short-wavelength radiation. The generation of energetic ultrashort pulses with these lasers is not trivial. Straightforward laser amplification will destroy both the laser and its light because the intensities are high enough to rip atoms apart. This catastrophe can be averted using exotic techniques with equally exotic names: chirped-pulse amplification, hollow-fibre self-phase modulation and chirped mirror compression. This is ultrafast optics raised to a high art, and Krausz is a virtuoso.

When the high-powered light is focused into atoms in a vapour, the atoms react in a number of ways. Some of them ionize rapidly, creating a shower of energetic electrons through what is called 'above-threshold ionization'[5]. Other atoms absorb laser light and re-radiate at very high harmonics (or multiples) of the laser frequency[6]. This high-harmonic radiation constitutes a unique source of 'soft' X-rays, with pulse durations that can be less than a femtosecond[2].

Krausz's group and others have studied the mechanism for high-harmonic genera-

tion. Theories suggest that the high harmonics are due to coherent radiation created by the recollisions of ionized electrons with their parent atoms[7,8]. If so (and experiments seem to support this), then the soft X-rays should arrive in attosecond bursts, occurring on every cycle of the laser field. Thus, the pattern of X-ray bursts matches the pattern of the optical carrier wave within the pulse envelope — and this is where the work of Hänsch's group and of Krausz's group now comes together[1].

When Krausz's laser is stabilized by Hänsch's technique, each 100-billion-watt pulse from the amplifier looks identical. This means that electrons ripped from the atoms in the focus of the laser beam follow identical paths, recollide with their parent atoms at identical stages of the pulse, and produce optical and electron spectra that record this violent motion. All previous experiments have been forced to average over non-identical laser pulses, and so many special characteristics of the atomic-scale process were washed out. In particular, the harmonics spectrum is now revealed to be not always 'harmonic' (an integer multiple of the pump frequency) after all. Rather, the spectrum is modulated by the coherent interference between successive X-ray bursts initiated at

different parts of successive cycles of the driving light field.

The phase-locking to produce identical pulses is successful down to a small fraction of an optical period, amounting to a temporal smearing of less than 200 attoseconds. Now, 200 attoseconds is not one attosecond, so the title of Baltuška and colleagues' paper[1] is perhaps rushing things a bit. Nonetheless, this is a remarkable achievement. This level of precision can capture the motion of photo-excited electrons as they move around their parent ion. The success of Hänsch, Krausz and their colleagues in creating frequency-locked high-field laser pulses marks the beginning of the era of 'attophysics' — the study of physical processes on the attosecond timescale. ■

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Cancer

More than skin deep

Diana Bolotin and Elaine Fuchs

Activation of the nuclear factor NF-κB has been linked with many human cancers. But in tumours of some tissues this molecule is inactive. New work examines how turning off NF-κB promotes cancer in one tissue, the skin.

What do cell death, cell proliferation, immune responses and tumour development have in common? The answer is that they are just a few of the many biological processes in mammals that are regulated by proteins of the nuclear factor NF-κB family[1,2]. Widely expressed throughout an organism, the NF-κB subunits are also exquisitely regulated in terms of their ability to enter the cell nucleus and activate key genes. However, although much is known about these proteins, many issues remain unexplored. For instance, studies of the immune system and several cell lines have indicated a role for NF-κB in promoting cell proliferation and protecting against programmed cell death (apoptosis)[2,3]. So it is puzzling that, in the skin, these proteins seem to oppose proliferation[4,5]. On page 639 of this issue[6], Dajee *et al.* address the paradox that inhibition of NF-κB in the skin can lead to increased cell proliferation — and cancer. Their findings may be important not only for understanding how skin tumours develop, but also for exploring

the design and use of NF-κB inhibitors in treating cancer.

Our cells regulate the activity of NF-κB proteins — which exist as dimers — largely by controlling their intracellular localization, using the IκB molecule[7]. When bound to IκB, NF-κB proteins are excluded from the nucleus and so are prevented from activating their target genes. However, certain stimuli can induce an enzyme complex, the IκB kinase, to modify IκB, targeting it for degradation. The NF-κB dimer is thereby released and moves into the nucleus, resulting in the expression of genes with functions appropriate to the stimuli.

For many years, the function of NF-κB in the organism seemed relatively straightforward, fitting a profile for the protein as the cell's protector against apoptosis and an enhancer of proliferation. So, in several organs and tissues, high levels of NF-κB can prevent apoptosis by activating genes for anti-apoptotic proteins such as Bcl-xL and cellular inhibitors of apoptosis[2]. And in the immune system, where these proteins have