should be realizable in optics using stimulated and spontaneous emission — the unavoidable presence of the latter whenever there is stimulated emission explains why perfect quantum cloning is impossible[7].

De Martini *et al.*[6] used parametric amplification in a nonlinear crystal to create photons — a technique widely used to produce entangled photon pairs. But, instead of exploiting the spontaneous process of the amplifier, De Martini *et al.* injected a photon into the crystal to stimulate the emission of two similar photons (two clones). When that happened, a third photon was also emitted in another direction. According to theory, this photon, or rather its polarization, represents the best possible approximation of the quantum NOT transformation.

The first experiments to achieve near-optimal quantum cloning have been reported recently[8–10]. De Martini *et al.* confirm these results, achieving a similar fidelity of 0.810, compared with the theoretical maximum of 0.833, or 5/6. And for the first optimal universal NOT gate, De Martini *et al.* achieve a fidelity of 0.630, close to the theoretical maximum value of 0.666, or 2/3.

Note that the optimal fidelity of 2/3 could also be achieved by simply measuring the input photon in a randomly chosen polarization basis and producing a new photon in a state of polarization orthogonal to the measurement result. As is well known, quantum measurements disturb the system, so even this procedure can't be perfect. But this crude method would achieve optimal fidelity for the NOT gate.

The universal NOT gate demonstrated by De Martini *et al.*[6], however, is much more in line with today's international effort to develop quantum logic circuits and with the ambitious goal of building a quantum computer. Whether a futuristic quantum computer will use optics to implement the qubits is unclear. But quantum repeaters for quantum cryptography[11] — the means to transmit decoding keys over large distances — will definitely use photons, and the necessary technology will involve techniques similar to the one demonstrated by De Martini and colleagues. ■

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Circadian rhythms

Finer clock control

J. D. Alvarez and Amita Sehgal

The clock that governs circadian rhythms is based on a molecular feedback loop, which has just become more complex — two more proteins have been identified as likely components of the loop.

During the 1990s, chronobiologists developed a unified model of the molecular mechanisms that underlie the circadian clock in all organisms. A typical molecular clock consists of an oscillatory feedback loop generated by a few central clock genes. But in order to understand how the clock regulates an organism's physiology and responds to the environment, it is essential to identify other genes and proteins that interact with the core components. On page 841 of this issue[1], Honma *et al.* show that the proteins Dec1 and Dec2 (so-called because they are found in differentiated embryo chondrocytes, or cartilage cells) inhibit transcription of the main clock genes. Moreover, expression of the *Dec1* gene is induced by light, suggesting that it is involved in mechanisms by which the clock senses the environment.

Circadian rhythms run in the absence of environmental cues, but the central clock nonetheless responds to such cues. For example, light resets the clock, which accounts for the phenomenon of jet lag after an intercontinental flight. At first, one's internal clock keeps time to the original light–dark cycle. It slowly adapts to the new environment and is completely reset after about a week. How light is actually translated into clock alterations is not clear.

Atmospheric science

Plumes and flumes

The fate of smoke billowing from industrial stacks, like those shown here, is a common concern — not least to those who have just hung out their washing. Writing in *Atmospheric Environment* (**36,** 4603–4615; 2002), R. W. Macdonald and colleagues describe modelling investigations of how smokestack configuration with respect to the prevailing wind affects the behaviour of smoke plumes.

As a single hot plume rises, vortices form within it and cool air is drawn in, so reducing plume buoyancy until it rises no further. But when two plumes merge, less cool air is entrained — because of the lower surface-area-to-volume ratio — and the plume is buoyant for longer, rising higher. The distance between smokestacks clearly influences plume merging. But what about the arrangement of the stacks?

Macdonald *et al.* used a water flume, studying hot-water plumes rising from two 10-cm 'stacks' by repeatedly dragging a grid of temperature probes through the plumes. Plumes in line with the overall flow in the flume quickly mixed and rose higher, as expected: the first plume shields the second from the flow, so that the latter bends less and rises into the first in a way that allows the internal vortices to mingle without destroying overall plume integrity.

Plumes from stacks aligned across the flow did not mix, and rose more slowly — mixing being hindered because the vortices at the edges of each plume tend to oppose each other. Interaction of vortices on the plume edges can also create a downwash effect, perhaps explaining the slowed plume rise.

Thus, smoke from chimneys set in line with wind direction, rather than across it, is likely to rise higher, travel further, and presumably become more dispersed and diluted before reaching the ground. **Jim Gillon**

R. F. BUKATY/AP

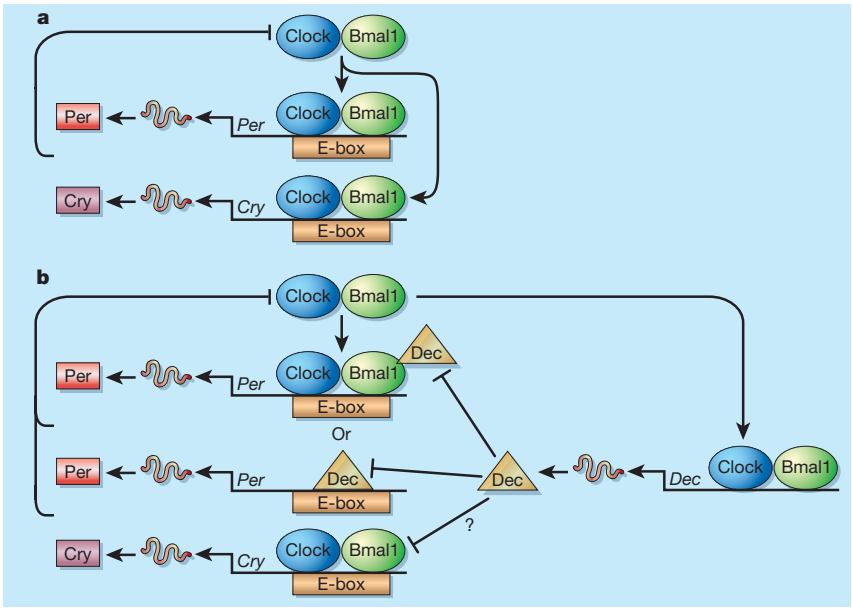

Figure 1 **The circadian clock and its regulation.** a, **The molecular basis of the clock is an oscillatory feedback loop consisting of positive and negative components. The positive components are the proteins Clock and Bmal1, which activate transcription of the negative-component genes, *Period* (*Per*) and *Cryptochrome* (*Cry*), by binding DNA sequences called E-boxes. The Per and Cry proteins repress the activity of Clock and Bmal1. Protein turnover allows the loop to restart.** b, **The Dec proteins, as described by Honma *et al.*[1], repress *Per* transcription by interfering with Clock/Bmal1 activity. The mechanism underlying this interference is not clear — Decs may bind to the E-box or to Bmal1, or to both. It is not known if they repress other genes, such as *Cry*. Transcription of *Dec* genes is reportedly activated by Clock and Bmal1.**

In mammals, the central circadian clock resides in a subset of neurons called the suprachiasmatic nucleus (SCN), which lies in the hypothalamus of the brain[2]. It is based on a cyclical feedback loop that includes two proteins, called Period (Per) and Cryptochrome (Cry)[3]. These are negative components because, when their levels are high, both proteins — but Cry in particular — repress transcription of the genes for the two proteins, resulting in decreased production of Per and Cry. The degradation of Per and Cry over time causes their levels to fall, resulting in relief of the repression and thus restarting the cycle. Both Per and Cry block the action of two positive clock components, Clock and Bmal1, which activate transcription of the *Per* and *Cry* genes. The overall result is oscillatory expression of clock genes and proteins in the SCN (Fig. 1a). Clock-gene expression also oscillates in tissues other than the SCN, so circadian clocks may occur in most organs of the body. These peripheral oscillators may tailor circadian responses to various physiological conditions, such as hunger and hormone release[4].

Honma *et al.*[1] propose that Dec1 and Dec2 are components of the core feedback loop (Fig. 1b). These proteins belong to the basic helix–loop–helix (bHLH) family, members of which dimerize with other family members and affect gene transcription by binding to specific DNA sequences called E-boxes[5]. Clock and Bmal1 are also bHLH proteins and activate transcription by binding E-boxes in the *Per* and *Cry* genes[6]. In contrast, the Decs repress transcription. Structurally, they are related to proteins found in the fruitfly *Drosophila* — Hairy and Enhancer of split — that function in neural development[7]. Honma *et al.* show that *Dec* expression is cyclic in the SCN and in other brain areas, and that Dec1 and Dec2 can strongly repress Clock/Bmal1 activation of the mouse *Per1* gene (one of three forms of *Period*). Moreover, they report that oscillatory expression of the *Dec* genes occurs in peripheral tissues.

The discovery of cyclic *Dec* transcription in the SCN is compelling. But do these genes function in the core feedback loop? Caution is needed here, because microarray experiments have identified hundreds of genes that show cyclic behaviour in the SCN[8,9]; presumably, not all of these are components of the central clock mechanism. A finding that favours a central role for the Decs is that only a small subset of genes cycle both in the SCN and in peripheral tissues, as the *Dec* genes do.

The repressive effect of the Decs on circadian transcription suggests that they are negative clock components. The oscillations in *Dec1* and *Dec2* RNA in the SCN lag slightly behind those of *Per1*, which correlates with the repressive effect on *Per1* transcription. If the Decs are indeed repressors of Clock/Bmal1-mediated transcription of *Per1*, then they would seem to be redundant given that Per and Cry — most notably Cry[10] — have the same function. Perhaps there are extra mechanisms to ensure timely repression of the positive components of the feedback loop. Alternatively, the Decs may regulate the amount of *Per1* synthesized rather than block its expression at a specific time of day. Because *Dec1* expression is upregulated following a light pulse in the middle of the night, it may be involved in the clock's response to light. Notably, *Per1* expression responds similarly to a light pulse. If *Dec1* represses *Per1* expression, it is unclear why both genes would be induced by light. Possibly, *Dec1* limits the *Per1* response and thereby the overall response of the clock to light.

Honma *et al.*[1] have produced some intriguing results. But we're left with many questions. Do the Decs merely repress Clock/Bmal1 activation of *Per1* (and perhaps other Clock/Bmal1 targets), or do they have another function within the clock? Also, how do these proteins accomplish gene repression? The Decs can interact directly with Bmal1, so these proteins may form a large complex on DNA that represses transcription. But the Decs can also bind E-boxes directly, and may inhibit Clock/Bmal1 binding to DNA. Finally, do the Decs function in peripheral clocks, as their oscillatory expression in peripheral organs would suggest? Use of gene-knockout techniques in mice may be needed to answer these questions — work that is probably already under way. ■

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correction

In Russell F. Doolittle's article "The grand assault", commenting on the genome sequence of *Plasmodium falciparum* (*Nature* **419,** 493–494; 2002), the size estimates for the *Dictyostelium discoideum* genome given in Fig. 1 were for its largest chromosome and not the entire genome — which, at around 11,000 genes and 32 megabases, is about four times larger. Labelling on the *x* axis runs as a log scale 1–10,000.