authors' previous discovery that Cdc42 can be detected on Golgi compartments[7]. So the Golgi may host a common target for the activities of two distinct small GTPases — Arf and Cdc42.

What, then, might be the function of Cdc42 in vesicle trafficking? Wu *et al.*[1] tackle this problem by following the effects of mutant Cdc42 on the transport of vesicular stomatitis virus G glycoprotein, which is commonly used in vesicle-trafficking studies. Modest expression of so-called 'dominant-negative' Cdc42 mutants[6] has shown that such mutants interfere with the movement of this viral protein from late Golgi compartments to the basal membrane in epithelial cells. The mutants also affect the ability of the endosomal pathway to maintain the top-to-bottom polarity of epithelial cells.

Wu *et al.*, by contrast, express mutant Cdc42 at higher levels that may stoichiometrically bind the endogenous COPI pool. They find that constitutively GTP-bound Cdc42 (mutant Q61L) and a constitutively GDP-bound form (in which serine 17 is mutated to asparagine) block transport of the viral protein from the ER to Golgi. Given that neither Arf nor COPI is found on ER membranes or is involved in protein export from the ER, it seems that a block in the recycling of proteins from the Golgi to the ER has an indirect, inhibitory effect on movement in the opposite direction, as one cannot proceed without the other. Other results support the idea that the different Cdc42 mutants link to COPI function by triggering a partial collapse of the Golgi into the ER, a COPI-sensitive step. Usually, the recycling of proteins from the Golgi to the ER means that there is a lag in the processing of glycoproteins by enzymes found only in late Golgi compartments. When the Cdc42 mutants are used, this lag does not occur, suggesting that the ER and Golgi compartments are mixed together.

One particular Cdc42 mutant, in which phenylalanine 28 is mutated to leucine, can cause growth transformation of NIH 3T3 cells[8]. As this mutant interferes with the recruitment of COPI to membranes[5], Wu *et al.* reason that the association of Cdc42 with γCOP may be linked to growth transformation. Indeed, they find that a derivative of this mutant that can no longer bind to γCOP or affect the interaction of the coatomer with the Golgi (or perhaps endosomal compartments) is also unable to cause growth transformation. So interaction with γCOP must be necessary for growth transformation by Cdc42. However, Cdc42 also contains a unique insert sequence that is required for transformation, and Cdc42 lacking this insert still retains the ability to bind γCOP and promote vesicle trafficking. This insert may bind to yet another effector, required together with γCOP to mediate transformation by Cdc42.

A cautionary note must be sounded by the fact that Wu *et al.* used high levels of exogenous, mutated Cdc42 protein. Nonetheless, their results beg the question of how the binding of endogenous Cdc42 to COPI might achieve cellular transformation. Dominant-negative Cdc42 disrupts cell polarity by interfering with vesicle trafficking through the Golgi[6]. So perhaps this loss of cell polarity, in response to dysfunctional control of COPI function by Cdc42, is an important step in the transformation process. One consequence might be the deregulation of the signalling pathways that maintain cells in a dormant state when they are in contact with other cells. The transport of signalling proteins that are normally required for cell maintenance may also become uncontrolled in response to altered trafficking pathways to and from the Golgi, supporting cell proliferation[9].

It is clear that cells must integrate forwards and reverse vesicle transport, together with the regulation of their cytoskeletons, to control their polarity (or migration) and proliferative behaviour. It seems that when a protein family plays together, cell behaviour stays together. ■

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Errata

In the article "Electrons in the looking glass" (Eric Heller *Nature* **403,** 489–491; 2000), the false impression was given that Madhavan *et al.* (*Science* **280,** 567–569; 1998) were the first group to publish STM evidence of Kondo resonances. In fact, Li *et al.* (*Phys. Rev. Lett.* **80,** 2893–2896; 1998) published their results a month earlier. Both groups acknowledge being aware of the other's results before publication.

In Alan P. Boss's article "Three's a crowd" (*Nature* **405,** 405–407; 2000), the source L1551 IRS5 was said to be a member of a triple system, like T Tauri. The text should have been corrected to state that L1551 IRS5 is known to be a binary protostar, whereas T Tauri itself may be one of a system of four stars.

Daedalus

Eye contact

A modern state needs to identify its citizens constantly. Many of them are a potential threat — benefit fraudsters, terrorists, drug pushers, bogus asylum-seekers, élitists, smokers. The citizens even need to identify themselves, both to the authorities and, in their financial dealings, to each other. Yet identity cards are easily stolen, fingerprinting is messy, DNA sampling is intrusive, and none of them work at a distance. Daedalus now has a new idea.

Portraits taken with small cheap cameras are often spoiled by 'red-eye'. The light from the flash enters the eyes of the subject, is reflected from the retinal blood-vessels, and makes his or her pupils appear red in the photograph. What a splendid way, says Daedalus, of obtaining the blood spectrum of a distant subject! DREADCO opticians are devising a camera to maximize the red-eye effect, and to record its spectrum over all the wavelengths that can enter the eye, from near ultraviolet to near infrared. One team is combining a frequency-swept flash with time-resolved charge-coupled-device imaging; another favours a broad-band flash source and a dispersive or Fourier-transform spectrometric detector. Meanwhile, the company's biochemists are exploring the individuality of a blood spectrum.

For a start, it should encode detailed blood-group data: not merely A, B and O, but all the dozens of lesser blood antigens. The plasma polysaccharides and proteins will also tell their story, partly hereditary and partly medical. Indeed, direct indices of criminality, such as metabolites of alcohol, cocaine or nicotine, should show up usefully in the blood spectrum. While not as specific as DNA analysis, red-eye spectrophotometry should still be a powerful identifier. The blood spectrum of every citizen will rapidly be acquired from normal passport or identification-card photography. The resulting database will transform surveillance.

Motorists in speeding cars, rioters in the street, burglars entering protected premises, all will be literally identified in a flash. In daylight the flash might not even be noticed. Counter-measures are possible; but a modern data-dictatorship is used to outflanking them. Just as the police stop any car without a licence plate, and the British government, in its plans to intercept the whole nation's e-mail, will demand that we decrypt for it any message it can't manage to decrypt for itself, so the authorities will arrest anyone wearing dark green glasses. **David Jones**