but variations of brightness with time allow astronomers to detect light echoes through reiterated observations of selected areas in the sky. A time-evolving re-brightening is exactly what Krause *et al.*[2] observed in a particular (and limited) region of the remnant of SN1572.

The light echo thus carries a sort of fossil imprint of the original supernova, and so analysis of the reflected light can unveil characteristics of it even centuries after it was first detected on Earth. Krause *et al.* obtained a spectrum of the bright-light echo of SN1572 with the Subaru 8.2-metre telescope on Mauna Kea (Hawaii) and showed that prominent silicon, sulphur and iron lines — which are typical of a normal type Ia supernova around its maximum — were present in the spectrum.

Type Ia supernovae are thought to be generated by the explosion of carbon–oxygen white dwarf stars in binary systems. As the white dwarf accretes mass from its companion star and eventually reaches the critical Chandrasekhar mass (1.4 solar masses), a thermonuclear explosion disrupts the star, releasing materials that are the product of the stellar nuclear fusion (mostly silicon and elements of the iron group). Nickel (^{56}Ni) in particular is ejected in great amounts — up to 50% of the whole white dwarf's mass. This radioactive isotope decays initially into radioactive cobalt (^{56}Co), and then into stable iron (^{56}Fe). Such a decay chain provides enough energy to keep the material ejected by the explosion hot for several months, and supports the supernova luminosity in the early phase of the transition towards the remnant stage. During this transformation, some — but not all — information about the explosion itself is lost. This implies that, in principle, the type of original supernova cannot be uncovered by studying the chemical abundances in the remnant alone.

Nevertheless, Tycho Brahe's supernova was suspected to be of type Ia well before the observations reported by Krause *et al.*[2]. The historical light curve[11], the studies of the remnant in the radio[12] and X-ray[13] wavelengths, and the discovery of a surviving candidate G-type companion star[14] (a star just like our Sun) all suggested a thermonuclear supernova rather than a core-collapse event. The fundamental contribution of Krause and colleagues' work is to transform these clues into definite proof — we are now fully confident that one of the most popular supernova remnants detected in our Galaxy was produced by an ordinary type Ia supernova that was first detected more than 400 years ago.

The technique of observing light echoes from supernovae is a remarkable observational tool with which to pigeonhole the type of supernova. It will allow astrophysicists to characterize other supernova remnants in our Galaxy and in nearby galaxies. This will hopefully clarify the relationship between supernova relics and their explosion mechanisms. Finally, it is likely that precise information about the frequency of the different supernova types in our Galaxy and its surroundings will shed light on the star-formation history and chemical evolution of the Local Group of galaxies. ■

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BEHAVIOURAL ECOLOGY

The social side of wild yeast

David C. Queller

The workhorse of cell biology, yeast, is a surprisingly cooperative organism. It uses an unusual means of identifying partners — a 'green-beard gene', which encodes a tag that must match among cooperating cells.

Everyone knows how a glass or two of beer can act as social glue, making even misanthropes amiable. Oddly, the production of beer has a similarly convivial effect on the tiny brewers that make it, cells of the yeast *Saccharomyces cerevisiae*. As alcohol content rises, the normally solitary cells begin to adhere to each other in clumps called flocs. Work on these flocs, just published by Smukalla *et al.* in *Cell*[1], shows that the yeast cells face a familiar social dilemma, but that they solve it in an exotic fashion.

In addition to its humble jobs as brewer and baker, yeast has added a high-tech career. It has become the principal laboratory model organism for studying the biology of eukaryotes — organisms such as plants and animals that have a membrane-bound nucleus. We have therefore come to understand yeast as well as any organism. Yet, although brewers have known about flocculation for centuries — sedimentation of the flocs provides an easy way to remove the yeast and keep the beer from tasting like the yeast paste Marmite — biologists have been slower off the mark. The problem is that flocculation was lost during the domestication of yeast. Smukalla *et al.*[1] therefore chose to study feral strains. They show that expression of one of five flocculation cell-adhesion genes, *FLO1*, explains much of the variation in flocculation (Fig. 1, overleaf). They establish causation by expressing *FLO1* in the domesticated strain, resurrecting flocculation, and putting *FLO1* into *Saccharomyces paradoxus*, inducing flocs even in this non-flocculating species.

Flocculation is a true cooperative trait that poses the classical social dilemma of how to sustain cooperation in the face of cheaters. Smukalla *et al.* show that cells on the inside of flocs are protected against damage from chemicals, including alcohol, partly because of physical shielding provided by cells on the outside. But those gains come at a cost; cells that express *FLO1* grow more slowly, even if they are prevented from flocculating. So, is it possible for cheaters to gain the protection of flocs without paying the cost?

The answer seems to be no, according to Smukalla *et al.*, because the ticket for admission to the floc is the ability to make the adhesion protein. It is just as if the bonhomie of the beer hall extended only to those drinking the same brand of beer or, on St Patrick's Day, only to those drinking green beer. In behavioural ecology, such tokens of inclusion are named not for green beer but for green beards. The name is fanciful because, until recently, such genes were purely imaginary. It is well known that genes for helping can spread by benefiting relatives, who share the gene with a specifiable probability. The alternative is to identify and help actual bearers of the gene, whether they are relatives or not[2]. Richard Dawkins[3] argued that such green-beard genes were unlikely to occur because they would need to cause three traits: a label, such as a green beard; recognition of others with the label; and nicer behaviour towards those with the label.

Green-beard systems have, however, begun to leave the realm of the imaginary. The initial examples involved nasty behaviour towards those lacking the gene. In the fire ant *Solenopsis invicta*, a linked set of alleles, including one encoding an olfactory receptor, causes workers to kill queens that lack the green-beard allele[4]. Microbes are proving a richer source of

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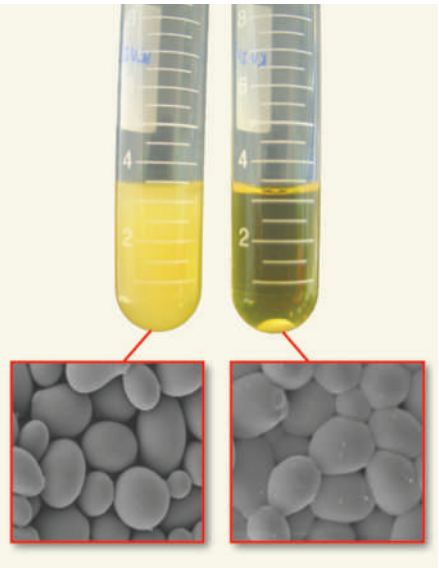
green-beard genes. Many bacteria act rather like fire ants, using linked poison-antidote genes to kill members of the same species that don't possess the antidote gene[5]. The social amoeba *Dictyostelium discoideum* provided the first single-gene example and the first altruistic one. Its *csaA* gene codes for a homophilic adhesion protein, one that grabs on to the same protein on other cells, so excluding strains that do not express the protein from the benefits of later altruism within the group[6].

The yeast *FLO1* green-beard gene adds a new level of interest because it is highly variable, and because the variation — in the number of repeats of a 100-base sequence — exerts a major influence on protein-binding strength. Conceivably, such variation could lead to multiple recognition tags (green beards, red beards, yellow beards and so on), although this possibility remains to be tested. Alternatively, the variation might be the product of highly complex adaptive dynamics, which have been observed in simulation models as various green-beard cooperators and cheaters rise and fall in frequency[7].

The discovery of *FLO1* also extends the reach of single-gene green beards. It is easy to see how homophilic adhesion molecules such as the product of *csaA* could recognize each other. In fact, it was predicted that they could be green beards[8]. But *FLO1* makes a heterophilic adhesion protein, one that binds not to itself but to oligosaccharides on the walls of other cells. Cells without the adhesion protein can still be bound by those that have it, but the binding is weaker than proper two-way binding, so such cells usually remain outside flocs. Even worse, the flocless cells that do get into flocs are exploited — they tend to end up in the outer layer, where they can be damaged in protecting the floc cells inside.



K. VERSTREPEN

Figure 1 | Yeast of a feather floc together. In the yeast *Saccharomyces cerevisiae*, the *FLO1* gene, whose expression has been lost in the domesticated strain (left), causes cells to adhere together in flocs that form a protective sediment (right). As Smukalla *et al.* report[1], *FLO1* is a green-beard gene that benefits copies of itself rather than kin as such.

The discovery of a green beard in an organism as mundane as yeast suggests that such genes might prove to be quite common, at least in microbes. More generally, microbial sociality itself is probably more common than lab studies might indicate. In model systems, domestication can easily lead to loss of social behaviours[9]. Another example is the bacterium *Bacillus subtilis*, in which multicellular fruiting bodies were seen only when wild strains were studied[10]. Much like the entrepreneur who rescued Marmite from the discards of the brewery, scientists can find value in the traits discarded by the domesticators. ■

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NEUROSCIENCE

Along memory lane

Yukiko Goda

Memories are encoded by efficient signalling between neurons. The myosin V proteins help this process by shuttling receptors and membranes to make synaptic junctions better detectors of incoming signals.

Synaptic junctions transmit information between neurons. The efficiency with which they do this is affected by how frequently they are activated, a cellular equivalent of experience. For example, repeated activation yields a lasting increase in the efficiency of synaptic transmission — a process called long-term potentiation (LTP) — which is thought to underlie memory formation. LTP depends both on enhanced insertion of receptors for the neurotransmitter glutamate at spines (the sites of synapses) and on spine growth. How neurons coordinate these two processes to trigger LTP has remained a mystery. Writing in *Cell*, Wang, Ehlers and colleagues[1] demonstrate that the motor protein myosin Vb is one missing link.

Dedicated to receiving signals, spines are tiny protrusions on the dendritic processes of neurons. They contain two types of glutamate receptor: AMPA receptors, which mediate most glutamate-dependent synaptic transmission; and NMDA receptors. On induction of LTP, activated NMDA receptors mediate a rapid influx of calcium ions (Ca^{2+}) into spines, which initiates a cascade of signalling events, culminating in the insertion of extra AMPA receptors into the spines, and spine growth.

Previously, Ehlers and colleagues had shown[2,3] that, during LTP, organelles known as recycling endosomes, which are normally found in dendritic regions near the base of spines, provide both AMPA receptors and membrane material for spine growth. In their latest work, the authors asked[1] what molecule directs the transport of recycling endosomes from dendrites into spines in response to Ca^{2+} signals.

Within the cell, motor proteins move organelles along tracks of cytoskeletal structures called microtubules and actin filaments. Dendrites are rich in microtubules, whereas spines are mostly filled with actin. So the ideal candidate for Wang and colleagues' search would be a Ca^{2+}-dependent protein that could hijack recycling endosomes from microtubules in dendrites and move them along actin within spines.

Myosin V proteins seem to fit the bill. In many cell types, these actin-based motor proteins specialize in organelle transport, their structures are regulated by Ca^{2+}, and they can counteract organelle movements along microtubules[4]. Furthermore, one of the three myosin V isoforms, myosin Vb, binds to recycling endosomes through the Rab11 protein on these organelles and another protein known as Rab11-FIP2, which interacts with Rab11 (ref. 4). In neurons, myosin-Vb activity affects surface expression and clustering of AMPA receptors[5].

Wang *et al.*[1] find that myosin Vb is indeed the protein mediating AMPA-receptor and membrane trafficking to spines. Visualizing the cellular movement of this protein by tagging it with a fluorescent molecule, they show that, under resting conditions, it is mostly present in spines. However, on induction of

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