observations: which aspects of environmental motion make the lizards adjust their choreography? What is the specific relationship between tail-flick dynamics and noise-signal distribution that leads to this particular signalling strategy? Which properties of low-level motion detectors and higher level motion integration mechanisms are required to optimise such signal detection in the presence of noise? Whatever the details that future work will uncover, the current paper demonstrates for the first time how a visual communication system is smartly adjusted to specific dynamic environmental conditions.

The study of animal communication, which bewilders the scientist with its variety and complexity, has the opportunity to achieve a new level of understanding by considering the communication signal content in the context of the neural processing necessary to enable 'secure' communication in real life, which is dynamic, noisy and short. This task requires the classically trained ethologist to communicate and collaborate with researchers in diverse other fields, such as ecologists, physicists, sensory physiologists and computational modelers. So what has this genuinely cross-disciplinary approach in stall for us as scientific community, for our sponsors, for our society? Studying motion processing mechanisms under natural operating conditions can provide essential clues to understanding how complex distributions of local motion signals can be segmented into meaningful patterns [14]. A deeper understanding of communication processes in other species will also provide new insights into the nature of human communication, its opportunities and limitations, and perhaps will even generate ideas for repairing or augmenting damaged or insufficient communication mechanisms. Comparative studies may be particularly helpful for analyzing body language in humans, a topic which has only recently seen the introduction of more rigorous quantitative methods, for instance, to investigate dynamic face perception [15]. Understanding how particular communication channels are optimized, how signal processing and signal production are shaped by external constraints, can further help to design sophisticated methods of signal extraction in a wide range of technical applications. And perhaps — blending the legend of Saint Francis, the fiction of Doolittle, and the passion of pet lovers into reality — we might eventually even be able to tap into animal communication channels and speak to the birds and the wolves.

**References**

1. Bradbury, J.W., and Vehrencamp, S.L. (1998). Principles of Animal Communication (Sunderland, MA: Sinauer Associates).
2. Pasteur, G. (1982). A Classification Review of Mimicry Systems. Annu. Rev. Ecol. System. *13*, 169–199.
3. Catchpole, C.K., Dittami, J., and Leisler, B. (1984). Differential responses to male song repertoires in female songbirds implanted with oestradiol. Nature *312*, 563–564.
4. Ghazanfar, A.A., and Hauser, M.D. (1999). The neuroethology of primate vocal communication: substrates for the evolution of speech. Trends Cogn. Sci. *3*, 377–384.
5. Marino, L., Connor, R.C., Fordyce, R.E., Herman, L.M., Hof, P.R., Lefebvre, L., Lusseau, D., McCowan, B., Nimchinsky, E.A., Pack, A.A., *et al*. (2007). Cetaceans have complex brains for complex cognition. PLoS Biol. *5*, e139.
6. David, C.T., Kennedy, J.S., and Ludlow, A.R. (1983). Finding of a sex pheromone source by gypsy moths released in the field. Nature *303*, 804–806.
7. Riley, J.R., Greggers, U., Smith, A.D., Reynolds, D.R., and Menzel, R. (2005). The flight paths of honeybees recruited by the waggle dance. Nature *435*, 205–207.
8. Salmon, M., and Hyatt, G.W. (1983). Communication. In Biology of Crustacea: Behaviour and Ecology, F.J. Vernberg and W.B. Vernberg, eds. (New York: Academic Press), pp. 1–40.
9. Endler, J.A. (1992). Signals, signal conditions, and the direction of evolution. Am. Nat. *139*, S125–S153.
10. Fleishman, L.J. (1992). The influence of the sensory system and the environment on motion patterns in the visual displays of anoline lizards and other vertebrates. Am. Nat. *139*, 36–61.
11. Peters, R.A., Hemmi, J.M., and Zeil, J. (2007). Signalling against the wind: modifying motion signal structure in response to increased noise. Curr. Biol. *17*, 1231–1234.
12. Zeil, J., and Zanker, J.M. (1997). A glimpse into crabworld. Vision Res. *37*, 3417–3426.
13. Peters, R.A., Clifford, C.W.G., and Evans, C. (2002). Measuring the structure of dynamic visual signals. Anim. Behav. *64*, 131–146.
14. Zanker, J.M. (2001). Combining local motion signals: A computational study of segmentation and transparency. In Motion Vision: Computational, Neural and Ecological Constraints, J.M. Zanker and J. Zeil, eds. (Berlin Heidelberg, New York: Springer), pp. 113–124.
15. O'Toole, A.J., Roark, D.A., and Abdi, H. (2002). Recognizing moving faces: a psychological and neural synthesis. Trends Cogn. Sci. *6*, 261–266.

Department of Psychology, Royal Holloway University of London, Egham, Surrey TW20 0EX, UK.
E-mail: J.Zanker@rhul.ac.uk

DOI: 10.1016/j.cub.2007.07.006

# Circadian Rhythms: Rho-Related Signals in Time-Specific Light Perception

**A recent study shows that a small GTPase, LIF1, helps to coordinate the plant circadian clock with the daily light–dark cycle.**

**E. Kolmos and S.J. Davis**

Virtually all life on Earth is exposed to rhythmic environments. On a daily scale, and at most latitudes, our planet's rotation results in a diurnal light–dark cycle involving significant changes in light quality and quantity, as well as duration. Many species have been shown to have an endogenous 'metronome' which anticipates these predictable changes. This timing device is a biological clock that controls rhythmic processes in the organism, and has a period length of about 24 hours; it has thus been termed the *circadian* clock. Importantly, circadian clocks are autonomous and enable sustained rhythmicity in the absence of environmental cues. And equally as

important, the changes in the duration of the light environment lead to ever-constant light resetting of this oscillator.

Circadian periodicity is not always 24 hours. The clock regularly has to reset to be synchronized with the natural phase of the day–night cycle. Light is believed to be the most important input factor to the plant circadian clock [1] — particularly the boundaries of dawn and dusk [2,3] — adjusting the clock's phase in a process termed entrainment. The phytochrome and cryptochrome photoreceptors are believed to provide the primary initiation event of diurnal-sensing [4]. The expression of photoreceptor genes is clock-regulated, and therefore they serve as phase-specific sensors to regulate entrainment of the oscillator [5–7]. It is unclear how light perception *per se* leads to a signal-transduction cascade that ultimately 'rewinds' the clock. But progress is being made in understanding this process, as evidenced by the recent *Current Biology* paper of Kevei *et al.* [8]: they report evidence that a small, functional GTPase is critical for normal oscillator function at the dusk phase of the diurnal cycle.

The plant oscillator is an interconnected system consisting of four coupled feedback loops [9,10]. The core of this system is a loop containing two genes encoding Myb-like proteins: *CIRCADIAN CLOCK-ASSOCIATED1* (*CCA1*) and *LATE ELONGATED HYPOCOTYL* (*LHY*). The expression levels of *CCA1* and *LHY* peak in the early day, and their products repress the gene *TIMING OF CAB EXPRESSION1* (*TOC1*). *TOC1* encodes a 'pseudo response regulator' and is expressed towards the end of the day [11,12]; it feeds back on *CCA1* and *LHY* activity, either directly or *via* an interconnected evening loop that includes *GIGANTEA* (*GI*) [13]. Two additional morning loops containing the *PRR9* and *PRR7* gene pair are coupled to *CCA1* and *LHY* expression [9]. The system is thus built of multiple oscillators driving timing information specific to daily time. Most clock components have only been characterized in relation to transcriptional activity, though some studies [14,15] have indicated that post-translational events are also important. So in order to characterize further the properties of the circadian system, it should be informative to take a closer look at the post-translational processes of clock components.

Kevei *et al.* [8] identified the *LIGHT INSENSITIVE PERIOD1* (*LIP1*) gene in a screen for mutations that alter rhythmic clock-gene expression. *LIP1* turned out to encode a small GTPase belonging to the family of plant-specific Rho-related GTPases. The LIP1 protein sequence has divergent features compared to its closest homologues, and so might have a unique biochemical function; but it is a biochemically functional GTPase.

In the *lip1* mutant plant, multiple circadian output rhythms have an abnormally short period. The implication is that LIP1 acts as a repressor of clock pace — in its absence, it seems the clock runs more quickly. In addition, the regulation of period length is impaired at high fluence rates of light, where the *lip1* mutant has a wild-type period, suggesting that light signaling represses LIP1 activity under high irradiance. Thus, negative arms control clock rhythms with respect to light input.

LIP1 function was found to be required at a particular phase of the day. A phase-resetting experiment showed that the *lip1* mutant exhibits increased sensitivity to light during the first half of the night, further supporting the view that LIP1 controls light input to the clock. But interestingly the *lip1* mutant phenotype is not solely confined to abnormal responses to the light environment. Furthermore, LIP1 does not act on the mean transcriptional activity of clock genes, and *LIP1* expression is not itself clock-regulated. LIP1 function is thus distinct from previously described clock regulators and the data are consistent with LIP1 having a post-translational role in clock regulation.

The targets of LIP1 GTPase activity remain to be identified. Among the ~100 small GTPases encoded by the *Arabidopsis* genome, versions of Rab, Rho, Arf and Ran can all be detected [16]; LIP1 does not belong to any of these clades [8]. Small GTPases have notably been implicated as regulators of intracellular membrane transport processes [17]. One reason LIP1 is of particular biochemical interest is that it lacks a membrane anchor, and it may have evolved away from membrane biology to adopt a novel biochemical task [8]. It is known that different GTPase isoforms can have distinct protein targets [18]. One can speculate that LIP1 might act on the clock by direct modulation of a core-clock element. As many small GTPases are effectively 'molecular switches', it is tempting to speculate that LIP1 enacts a small feedback loop of biochemical activity within the mechanics of the clock.

Interestingly, under diurnal conditions, the *lip1* mutant was found to have low *TOC1* expression [8]. This is consistent with LIP1 having an evening function, perhaps specific to one of the four feedback loops in the circadian system. The 'second loop' connecting the expression of the evening genes *TOC1* and *GI* to the morning genes *CCA1* and *LHY* has been shown to require light-input signals [9,10]. Perhaps LIP1 has a major role in control of light input to the clock at this 'entry point'. Further experiments are needed before LIP1 can be integrated to the loop-structure of the circadian system.

The involvement of small G proteins in control of the central oscillator is emerging as an evolutionarily conserved clock-controlling step. Although LIP1 is the first example of such a protein in the *Arabidopsis* clock [8], there are indications from work on other systems that G proteins play a part in circadian biology: in particular, a diverged small GTPase-like protein apparently influences circadian rhythms in the mouse [19], and a close Rab-relative from pea was found to regulate light signals [20]. In the mouse

study [19], *earlybird* (*Ebd*) was identified as a short-period mutant in a direct screen for clock-defective animals; *Ebd* was shown to be identical to the *Rab3a* locus. Interestingly, here, as with LIP1, the *Ebd* mutant was found to have normal expression levels of core-clock genes. LIP1 and Rab3a are both GTPases suggested to be working post-translationally on as yet unknown targets.

*PRA2* from pea was isolated as a gene encoding a small GTPase that mediates photomorphogenesis [20]. The *lip1* mutant is also perturbed in light perception [8], but there are two key differences between LIP1 and PRA2. For one thing, PRA2, and not LIP1, is a typical Rab/Rho, in that it is membrane-localized [20]. Furthermore, LIP1 function in the clock can be uncoupled from photomorphogenesis. Collectively, it looks as if divergent systems have incorporated GTPases as biochemical mediators. But for LIP1, this is probably the extent of analogy, as it is degenerative within the Rab/Rho clade, and it is not obviously membrane sequestered. Understanding how LIP1 functions within the *Arabidopsis* oscillator holds great promise towards opening our eyes to the biochemical and cell-biological events of the plant circadian oscillator.

**References**

1. McClung, C.R. (2006). Plant circadian rhythms. Plant Cell *18*, 792–803.
2. McWatters, H.G., Bastow, R.M., Hall, A., and Millar, A.J. (2000). The *ELF3* zeitnehmer regulates light signalling to the circadian clock. Nature *408*, 716–720.
3. Millar, A.J., and Kay, S.A. (1996). Integration of circadian and phototransduction pathways in the network controlling *CAB* gene transcription in Arabidopsis. Proc. Natl. Acad. Sci. USA *93*, 15491–15496.
4. Valverde, F., Mouradov, A., Soppe, W., Ravenscroft, D., Samach, A., and Coupland, G. (2004). Photoreceptor regulation of CONSTANS protein in photoperiodic flowering. Science *303*, 1003–1006.
5. Bognar, L.K., Hall, A., Adam, E., Thain, S.C., Nagy, F., and Millar, A.J. (1999). The circadian clock controls the expression pattern of the circadian input photoreceptor, phytochrome B. Proc. Natl. Acad. Sci. USA *96*, 14652–14657.
6. Dodd, A.N., Salathia, N., Hall, A., Kevei, E., Toth, R., Nagy, F., Hibberd, J.M., Millar, A.J., and Webb, A.A. (2005). Plant circadian clocks increase photosynthesis, growth, survival, and competitive advantage. Science *309*, 630–633.
7. Toth, R., Kevei, E., Hall, A., Millar, A.J., Nagy, F., and Kozma-Bognar, L. (2001). Circadian clock-regulated expression of phytochrome and cryptochrome genes in Arabidopsis. Plant Physiol. *127*, 1607–1616.
8. Kevei, E., Gyula, P., Feher, B., Toth, R., Viczian, A., Kircher, S., Rea, D., Dorjgotov, D., Millar, A.J., Kozma-Bognar, L., *et al*. (2007). Regulation of the *Arabidopsis thaliana* circadian clock by the small GTPase LIP1. Curr. Biol. *17*, 1456–1464.
9. Locke, J.C.W., Kozma-Bognar, L., Gould, P.D., Feher, B., Kevei, E., Nagy, F., Turner, M.S., Hall, A., and Millar, A.J. (2006). Experimental validation of a predicted feedback loop in the multi-oscillator clock of *Arabidopsis thaliana*. Mol. Syst. Biol. *2*, 59.
10. Zeilinger, M.N., Farre, E.M., Taylor, S.R., Kay, S.A., and Doyle, F.J. (2006). A novel computational model of the circadian clock in *Arabidopsis* that incorporates *PRR7* and *PRR9*. Mol. Syst. Biol. *2*, 58.
11. Alabadi, D., Oyama, T., Yanovsky, M.J., Harmon, F.G., Mas, P., and Kay, S.A. (2001). Reciprocal regulation between *TOC1* and *LHY/CCA1* within the Arabidopsis circadian clock. Science *293*, 880–883.
12. Ding, Z., Doyle, M.R., Amasino, R.M., and Davis, S.J. (2007). A complex genetic interaction between *Arabidopsis thaliana* *TOC1* and *CCA1/LHY* in driving the circadian clock and in output regulation. Genetics *176*, 1501–1510.
13. Locke, J.C., Southern, M.M., Kozma-Bognar, L., Hibberd, V., Brown, P.E., Turner, M.S., and Millar, A.J. (2005). Extension of a genetic network model by iterative experimentation and mathematical analysis. Mol. Syst. Biol. *1*, 2005.0013.
14. Daniel, X., Sugano, S., and Tobin, E.M. (2004). CK2 phosphorylation of CCA1 is necessary for its circadian oscillator function in Arabidopsis. Proc. Natl. Acad. Sci. USA *101*, 3292–3297.
15. Mas, P., Kim, W.Y., Somers, D.E., and Kay, S.A. (2003). Targeted degradation of TOC1 by ZTL modulates circadian function in *Arabidopsis thaliana*. Nature *426*, 567–570.
16. Vernoud, V., Horton, A.C., Yang, Z., and Nielsen, E. (2003). Analysis of the small GTPase gene superfamily of Arabidopsis. Plant Physiol. *131*, 1191–1208.
17. Grosshans, B.L., Ortiz, D., and Novick, P. (2006). Rabs and their effectors: achieving specificity in membrane traffic. Proc. Natl. Acad. Sci. USA *103*, 11821–11827.
18. Pfeffer, S.R. (2005). Structural clues to Rab GTPase functional diversity. J. Biol. Chem. *280*, 15485–15488.
19. Kapfhamer, D., Valladares, O., Sun, Y., Nolan, P.M., Rux, J.J., Arnold, S.E., Veasey, S.C., and Bucan, M. (2002). Mutations in Rab3a alter circadian period and homeostatic response to sleep loss in the mouse. Nature Genetics *32*, 290–295.
20. Kang, J.G., Yun, J., Kim, D.H., Chung, K.S., Fujioka, S., Kim, J.I., Dae, H.W., Yoshida, S., Takatsuto, S., Song, P.S., *et al*. (2001). Light and brassinosteroid signals are integrated via a dark-induced small G protein in etiolated seedling growth. Cell *105*, 625–636.

Max Planck Institute for Plant Breeding Research, Carl-von-Linné-Weg 10, D-50829 Cologne, Germany.
E-mail: davis@mpiz-koeln.mpg.de

DOI: 10.1016/j.cub.2007.07.031

# Social Evolution: The Decline and Fall of Genetic Kin Recognition

**Animals should benefit from the ability to recognise their kin, yet curiously this faculty is often absent. New theory confirms that genetic kin recognition is inherently unstable, explaining its rarity.**

**Andy Gardner and Stuart A. West**

Cooperation abounds in the natural world, and biologists are faced with the difficulty of reconciling this fact with the principle of the 'survival of the fittest'. A fundamental step in our understanding of cooperation was provided by W.D. Hamilton's theory of inclusive fitness [1]. This reveals that altruistic behaviour, where an individual pays a direct fitness cost in order to enhance the fitness of others, can be favoured by selection if individuals tend to promote the reproductive success of their genetic relatives. This raises the question of how altruists ensure that their selfless behaviour is directed primarily towards their kin. One possibility is genetic kin recognition, where individuals identify close kin on the basis of physical similarity because relatives look more similar than unrelated individuals [1,2]. Despite the apparent incentive for such kin recognition, however, there is relatively poor empirical support for this mechanism in nature. A new theoretical study of genetic kin recognition by François Rousset and Denis Roze [3] reveals that, left