to identify surface proteins from viral and bacterial pathogens that share structural features with host-cell ligands. Reasoning that interactions of ligand mimics with host receptors might facilitate pathogen adhesion and access to host cells, the researchers then tested whether putative mimics could directly bind to their cognate receptors. Despite the small data set used for the comparisons, the approach identified several candidates of mimicry, highlighting the potential of melding structural and evolutionary biology not only for delineating mechanisms of pathogen mimicry, but also for identifying unknown mimics and their targets.

One example of mimicry proposed by the authors was a structural feature of the simian polyoma virus SV40 protein VP1, which resembles Gas6, a ligand of the receptor tyrosine kinase Axl (Fig. 1). The identification of Axl as a probable target of ligand mimicry hints at its importance in viral infections. Axl function has also been implicated in infections with Kaposi's sarcoma-associated herpesvirus and Ebola virus[6,7], but its precise role is unclear. This case illustrates how, although informative, the use of structural approaches as a predictive tool for studying mimicry provides only a partial view of the underlying function of the identified mimics. Furthermore, because functional mimicry can evolve in the absence of any structural similarity, many cases of convergent mimicry cannot be uncovered by structural comparisons. To move forward from structure-based screens, we will need to decipher the molecular basis of mimic functions and their contributions to pathogenesis.

Drayman and colleagues' study exemplifies how increasingly powerful tools for exploring the biology of molecules and genomes are helping researchers to uncover additional examples of molecular mimicry, some as exquisite as the classically described cases of morphological mimicry. Molecular mimicry, much like its Batesian counterpart, has major consequences for the evolution of both hosts and pathogens. Mimics pose a daunting challenge to hosts — how does a host differentiate between its native ligands and pathogen mimics, while maintaining core functions?

Research is beginning to illuminate how the rapid accumulation of protein-coding mutations, a conspicuous sign of positive selection, alters host proteins at crucial binding interfaces with pathogen mimics. See-sawing adaptations between hosts and pathogens at these protein surfaces can lead to molecular 'arms races', characterized by intense bouts of positive selection over millions of years[8]. Studying such evolutionary patterns can provide important insight into the potential for hosts to combat the challenge of mimicry[9]. Thus, the thriving alliance between evolutionary and experimental approaches[10] holds great promise for enhancing our knowledge of the evolution of pathogen mimicry. In this light, it is exciting to consider how Bates's observations of rainforest butterflies long ago might help to inform our understanding of infectious disease today. ■

Matthew F. Barber *and* **Nels C. Elde** *are in the Department of Human Genetics, University of Utah, Salt Lake City, Utah 84112, USA.*
e-mails: mbarber@genetics.utah.edu; nelde@genetics.utah.edu

1. Drayman, N. *et al. Cell Host Microbe* **14,** 63–73 (2013).
2. Elde, N. C. & Malik, H. S. *Nature Rev. Microbiol.* **7,** 787–797 (2009).
3. Doxey, A. C. & McConkey, B. J. *Virulence* **4,** 453–466 (2013).
4. Stebbins, C. E. & Galán, J. E. *Nature* **412,** 701–705 (2001).
5. Holm, L. & Sander, C. *Trends Biochem. Sci.* **20,** 478–480 (1995).
6. Liu, R. *et al. Blood* **116,** 297–305 (2010).
7. Brindley, M. A. *et al. Virology* **415,** 83–94 (2011).
8. Daugherty, M. D. & Malik, H. S. *Annu. Rev. Genet.* **46,** 677–700 (2012).
9. Elde, N. C., Child, S. J., Geballe, A. P. & Malik, H. S. *Nature* **457,** 485–489 (2009).
10. Dean, A. M. & Thornton, J. W. *Nature Rev. Genet.* **8,** 675–688 (2007).

BIOGEOCHEMISTRY

As different as night and day

An analysis of northern ecosystems shows that the effects on plant growth of rising night-time temperatures are opposite to those of increasing daytime temperatures — a finding that has implications for carbon-cycle models. SEE LETTER P.88

CHRISTOPHER STILL

An under-appreciated aspect of climate change is the fact that Earth is warming at a higher rate at night than during the day: over the past 50 years, daily minimum temperatures have increased about 40% faster than daily maximum temperatures[1]. This asymmetric warming may have important biological consequences, particularly for fundamental ecosystem metabolic processes that are strongly sensitive to temperature variations, such as photosynthesis and respiration. On page 88 of this issue, Peng *et al.*[2] document regionally significant, and in many cases opposing, effects of year-to-year (interannual) variations in daytime and night-time temperatures on plant growth and carbon cycling in land regions of the Northern Hemisphere.

Photosynthesis is driven by light and thus happens only during the day, whereas plant and microbial respiration occurs continuously. Therefore, faster night-time warming presumably affects respiration more than it affects photosynthesis, and this could have far-reaching implications for how ecosystems react to expected increases in warming in coming decades. But remarkably little research has been

PETER ESSICK/AURORA PHOTOS/ROBERT HARDING PICTURE LIBRARY

Figure 1 | Boreal forest by night. Climate change is causing nights to warm faster than days. Peng *et al.*[2] report that the metabolic effects of warmer nights oppose the effects of warmer days in certain Northern Hemisphere ecosystems, such as boreal forests.

done on how asymmetric warming influences ecological function, especially at large scales. To address this issue, Peng and colleagues have analysed satellite-derived data sets of plant greenness, which is a proxy for plant growth.

The authors found that ecosystems in cool, wet temperate and boreal regions such as northwestern North America and Japan, and those in cold regions such as Siberia and the Tibetan plateau, seem to have benefited most from daytime temperature increases over the period considered (1982–2009). By contrast, ecosystems in dry temperate regions, such as central Eurasia and western China, showed the opposite effect: increasing daytime temperatures correlated with decreasing plant greenness. These contrasting responses broadly agree with expectations for ecosystems in which plant growth is limited primarily by temperature (cool, wet climates) or moisture (warm, dry climates).

More intriguingly, Peng and colleagues found that ecosystems in many of the boreal and wet temperate regions grew less well in response to increases in night-time minimum temperatures — the opposite effect to their response to increasing daytime maximum temperatures (Fig. 1). Conversely, in many arid and semi-arid regions, such as the grasslands of China and North America, increasing night-time minimum temperatures correlated positively with plant greenness.

Peng *et al.* used a statistical approach to control for other contributing environmental variables, such as solar radiation and precipitation. This allowed them to isolate the interannual greenness responses to daytime maximum and night-time minimum temperature variations. The authors confirmed the statistical validity of their findings using other techniques, and also analysed the sensitivity of the greenness response to alternative interpolated climate data sets and at individual weather-station locations. Importantly, the different analyses all confirmed the same broad conclusions.

A strength of this study is that the researchers explored ecosystem responses to asymmetric warming using a variety of other large-scale data sets, and found similar patterns. One data set was for the net exchange of carbon between land and the atmosphere — a quantity that integrates photosynthesis and respiration, and which was inferred from a multi-year analysis[3]. Peng and co-workers found that this quantity correlated positively with daytime temperature variations for cool and wet boreal ecosystems, but negatively with night-time temperatures for these ecosystems. They also observed that the amplitudes of the seasonal cycles of carbon dioxide levels measured at Point Barrow, Alaska, and Mauna Loa, Hawaii, vary in the same way with daytime and night-time temperature variations in boreal regions, but not in temperate areas.

Peng *et al.* focused only on boreal and temperate ecosystems. The response to asymmetric warming of tropical and subtropical ecosystems, which account for most CO_2 exchange between the land and the atmosphere, is not clear and merits further investigation. Previous work[4] at a well-studied tropical forest revealed a negative correlation between tree growth and annual mean daily minimum temperatures, a response broadly similar to Peng and colleagues' findings for boreal forests. Tropical forests are thought to be vulnerable to warming[5], with some evidence[6] suggesting that they are already near high-temperature thresholds above which growth could be restricted. Future research could help to fill major gaps in our understanding of thermal tolerance and acclimation in tropical and subtropical plant species, and thus their response to warming[5,7].

So what are the physiological mechanisms that drive large-scale correlations between temperature variations and ecosystem metabolism? The commonly discussed mechanisms involve biochemical responses to temperature, but with some interesting twists. For example, the positive correlation found between night-time minimum temperatures and greenness in semi-arid grasslands is puzzling, but might be related to greater night-time plant respiration that stimulates increased daytime photosynthesis[8]. Increases in night-time respiration have also been invoked in a pioneering study[9] of nocturnal warming that documented different plant responses in grassland: the dominant grass species declined in response to increases in night-time temperature during spring, whereas other plant species that use a different photosynthetic pathway increased in number.

A research agenda to investigate these mechanisms further should include manipulative field and mesocosm experiments (in which small parts of a natural ecosystem are enclosed and warmed). Experimental warming studies are lacking for many ecosystems. Even fewer night-time warming experiments have been conducted so far, with most being in shrublands[10] or grasslands and croplands[8]; warming experiments that truly impose asymmetry between day and night warming are rare[11]. There is a particularly urgent need for warming studies in forests, which dominate the global carbon cycle and climate feedbacks.

However, there are substantial technological challenges to conducting such experiments in large-statured ecosystems. Forest mesocosm experiments would require exceedingly complex and expensive facilities. Despite these limitations, Peng and colleagues' results argue strongly for an increased focus on the differing ecological impacts of night-time and daytime temperatures, to improve our ability to understand and predict how warming will affect Earth's ecosystems. ■

Christopher Still *is in the Department of Forest Ecosystems and Society, Oregon State University, Corvallis, Oregon 97331, USA.*
e-mail: chris.still@oregonstate.edu

1. Solomon, S. *et al.* (eds) *Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change* (Cambridge Univ. Press, 2007).
2. Peng, S. *et al. Nature* **501,** 88–92 (2013).
3. Chevallier, F. *et al. J. Geophys. Res.* **115,** D21307 (2010).
4. Clark, D. A., Piper, S. C., Keeling, C. D. & Clark, D. B. *Proc. Natl Acad. Sci. USA* **100,** 5852–5857 (2003).
5. Corlett, R. T. *Trends Ecol. Evol.* **26,** 606–613 (2011).
6. Doughty, C. E. & Goulden, M. L. *J. Geophys. Res.* **113,** G00B07 (2008).
7. Zhang, J.-L., Poorter, L., Hao, G.-Y. & Cao, K.-F. *Ann. Bot.* **110,** 1027–1033 (2012).
8. Wan, S., Xia, J., Liu, W. & Niu, S. *Ecology* **90,** 2700–2710 (2009).
9. Alward, R. D., Detling, J. K. & Milchunas, D. G. *Science* **283,** 229–231 (1999).
10. Beier, C. *et al. Sci. Total Environ.* **407,** 692–697 (2008).
11. Phillips, C. L., Gregg, J. W. & Wilson, J. K. *Glob. Change Biol.* **17,** 3263–3273 (2011).

CELL BIOLOGY

Recycling in sight

Vision requires the continuous recycling of photobleached pigments. An atypical form of a degradative pathway called autophagy seems to participate in this process in retinal pigment epithelial cells.

PATRICIA BOYA & PATRICE CODOGNO

Vision begins with the absorption of photons by light-sensitive photoreceptor cells in the retina. When photons arrive, chromophore molecules in the photoreceptors undergo conformational changes and trigger the phototransduction cascade, which converts light into electrical impulses that travel to the brain and are transformed into the images that we see. To sustain vision, the chromophore 11-*cis*-retinal must return to its original conformation through a process known as the visual cycle. Writing in *Cell*, Kim *et al.*[1] demonstrate that one process that contributes to the proper functioning of the visual cycle is an atypical form of autophagy — the 'self-eating' pathway through which cells recycle their components by degrading them in cellular organelles called lysosomes.

The visual cycle involves tight regulation of