light-emitting mechanism known as delayed fluorescence[6]. These strategies overcome the problem for devices based on conventional fluorescent molecules, but Ai *et al.* now report an innovative alternative method: they use organic radical molecules that exploit a different light-emitting mechanism, thereby enabling an IQE of almost 100%.

So what are organic radicals? Most organic molecules have an even number of electrons, in which each electron pairs up with another one, forming what is known as a closed-shell state. Organic radicals, however, have an odd number of electrons, and have one or more unpaired electrons in 'open-shell' states. Such radicals are highly reactive and therefore chemically unstable, and are typically generated transiently during chemical reactions. But the reactivity of radicals can be suppressed by modifying their molecular structures, and some are stable enough to be handled under air at room temperature.

In the context of light emission, it has long been thought that almost all stable radicals are non-emissive and inhibit emission from other sources. Nevertheless, stable light-emitting radicals have been available[7,8] since 2006, raising the possibility that they could be used in lighting materials and devices. Importantly, it was proposed[9] that ROLEDs would have high IQEs because, owing to the radicals' open-shell electronic states, they don't exhibit the energy-loss pathways that cause problems in conventional OLEDs.

The first ROLED was reported[10] in 2015 by researchers from one of the groups that contributed to the current paper, and it had an EQE of 2.4%. A year later, the same group showed experimentally[3] that it should be possible for ROLEDs to achieve an IQE of 100% — a milestone in the history of this LED class. Ai *et al.* now report another key step in the evolution of ROLEDs: they have developed two stable radicals that emit brightly in the deep-red and infrared regions of the spectrum, and they use them in devices that not only achieve almost 100% IQE, but also have an excellent EQE of 27%. This is the highest EQE among all LEDs that emit similar colours, and is largely a consequence of the efficiency with which electrons are converted into light on the radicals.

The high efficiency of Ai and colleagues' device is impressive, but ROLEDs in general currently emit light in only a limited range of colours. This is because just a small number of stable light-emitting radicals have been reported, and only those that have a particular type of chemical structure (known as an electron-donating group) deliver high EQEs when used in ROLEDs. Moreover, the electronic characteristics of light-emitting radicals suggest that these molecules will not be good at emitting blue (high-energy) light. A crucial next step will be to establish molecular design principles that enable organic radicals to be tuned to produce a wide range of colours — Ai and co-workers' radicals are not the first to emit deep-red and infrared light, and so have not extended the colour range.

Nonetheless, Ai and co-workers have demonstrated an innovative method for increasing the EQE of OLEDs, which could not have been achieved through simple developments of conventional fluorescent OLEDs. The authors' method also increases the number of radicals that can be used in ROLEDs. Given that they were discovered only a few years ago, there is probably plenty of potential for even further improvement — a challenge that offers great opportunities for materials scientists. In this field, radical progress truly promises a bright future. ■

Tetsuro Kusamoto *and* **Hiroshi Nishihara** *are in the Department of Chemistry, School of Science, University of Tokyo, Hongo, Bunkyo-ku, Tokyo 113-0033, Japan.*
e-mail: kusamoto@chem.s.u-tokyo.ac.jp

1. Ai, X. *et al. Nature* **563**, 536–540 (2018).
2. Tang, C. W. & VanSlyke, S. A. *Appl. Phys. Lett.* **51**, 913–915 (1987).
3. Obolda, A., Ai, X., Zhang, M. & Li, F. *ACS Appl. Mater. Interfaces* **8**, 35472–35478 (2016).
4. Rothberg, L. J. & Lovinger, A. J. *J. Mater. Res.* **11**, 3174–3187 (1996).
5. Baldo, M. A. *et al. Nature* **395**, 151–154 (1998).
6. Uoyama, H., Goushi, K., Shizu, K., Nomura, H. & Adachi, C. *Nature* **492**, 234–238 (2012).
7. Gamero, V. *et al. Tetrahedron Lett.* **47**, 2305–2309 (2006).
8. Heckmann, A. *et al. J. Phys. Chem. C* **113**, 20958–20966 (2009).
9. Hattori, Y., Kusamoto, T. & Nishihara, H. *Angew. Chem. Int. Edn* **53**, 11845–11848 (2014).
10. Peng, Q., Obolda, A., Zhang, M. & Li, F. *Angew. Chem. Int. Edn* **54**, 7091–7095 (2015).

BIOPHYSICS

Membranes stick to one dimension

A nanometre-scale mechanism has been proposed to explain how bacteria improve their grip on human cells. The findings have implications for drug discovery, and might inspire biomimetic applications such as adhesives.

JOHN R. DUTCHER

Biological membranes serve as the barrier between cells and their surrounding environment, and regulate the transfer of ions and small molecules into and out of cells. Because of their central role in proper cellular operation, membranes are a target for many disease-causing microorganisms[1]. Writing in *Nature Communications*, Charles-Orszag *et al.*[2] propose a previously unknown mechanism by which one such pathogenic bacterium, *Neisseria meningitidis* (also known as meningococcus), rearranges the outer plasma membrane of host cells to improve its adhesion to the cells. Achieving improved cell adhesion is a key step in host infection, which in humans can lead to septic shock and meningitis[3].

A key challenge for *N. meningitidis* is how to stick to and colonize the endothelial cells that line blood vessels without being swept away by flowing blood. The interaction between the bacterial and endothelial-cell surfaces is not strong enough to withstand the forces exerted by blood flow[4], and so *N. meningitidis* uses extremely thin (6-nanometre-diameter) protein filaments called type IV pili (T4P) to increase its grip on the cell membrane. T4P can be extended and retracted through the cell wall in a variety of bacteria, and have crucial roles in the microbes' life cycle, allowing them to stick to and move across surfaces and to infect or damage other cells[5].

The interaction between *N. meningitidis* cells and endothelial cells results in the formation of protrusions on the endothelial-cell membrane[4], and it has been shown that proteins in T4P are essential for protrusion formation[6], and that they interact with specific receptors in the endothelial cells[7]. However, the molecular mechanism underlying the interaction of T4P with host cells was not understood. Charles-Orszag and co-workers now shed light on this mechanism by combining *in vivo* and *in vitro* studies with a simple theoretical model.

The theoretical model is one of the strengths of the new study, and describes a previously unknown mechanism for wetting (the spreading of a deformable substance such as a liquid on the surface of another substance[8]). Wetting is key to many aspects of everyday life, from the spreading of ink on paper to the beading of water droplets on spider webs or freshly waxed cars, and it typically occurs in two dimensions. However, in the case of a very thin fibre in contact with a soft membrane, the membrane cannot wrap around (wet) the fibre, because too much energy is required to accommodate the large curvature around the fibre's cross-section.

Charles-Orszag *et al.* use their model to show that it can be energetically more favourable for a narrow tube to be drawn out from the membrane, along the fibre, than wrapped around it (Fig. 1). This mechanism

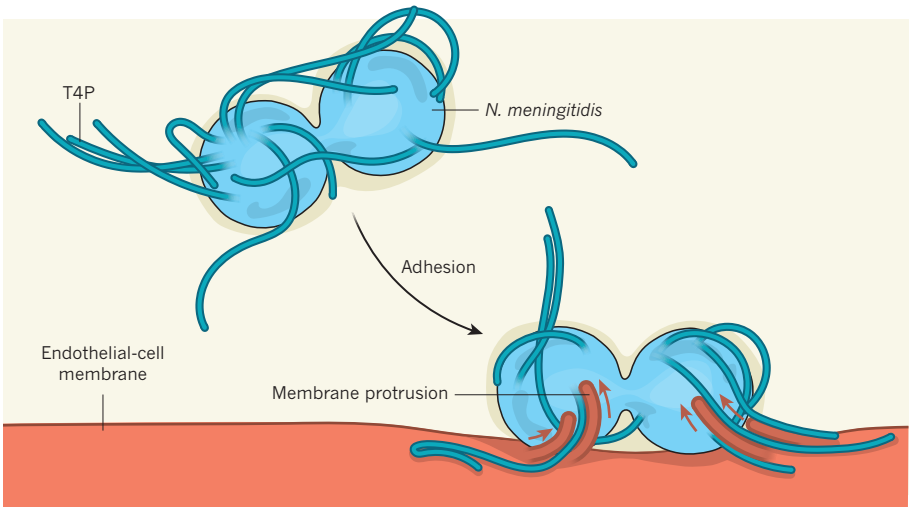

Figure 1 | Enhancing adhesion between a bacterium and an endothelial cell. The bacterium *Neisseria meningitidis* attaches itself to the endothelial cells that line blood vessels in host organisms. The bacterium uses fibres known as type IV pili (T4P) to induce the formation of protrusions from endothelial-cell membranes. These protrusions strengthen the bacterium's hold on the membrane, helping it to colonize cells without being swept away by the surrounding blood flow. Charles-Orszag *et al.*[2] propose that the adhesion of T4P to the membrane drives a process called one-dimensional wetting, in which the protrusions are drawn along the T4P fibres (red arrows). (Adapted from Fig. 5 of ref. 2.)

for forming membrane protrusions, which the authors call one-dimensional wetting, is driven by adhesion between the membrane and the fibre. The membrane protrusions could help to anchor a bacterium to a host cell as its T4P extend and retract, without breaking the adhesive interactions between the T4P and the membrane — thus maintaining the dynamic nature of the fibres.

Because the remodelling of endothelial-cell membranes by *N. meningitidis* had previously been observed only for cultured cells, the researchers studied blood vessels in human skin grafted onto mice to confirm that remodelling also occurs *in vivo*. They then complemented those experiments with *in vitro* studies to explore the mechanism involved. Unfortunately, the *in vitro* experiments did not examine the interaction of isolated T4P with model membranes, because this would have required the appropriate receptor proteins to be introduced into the membranes. Instead, Charles-Orszag *et al.* studied two model systems: artificial cells (known as giant unilamellar vesicles) interacting with filaments of a protein called actin through adhesion between the filaments and molecules attached to the cells; and endothelial-cell membranes interacting with mimics of the fibres found in the extracellular matrix around cells.

The authors show that 1D wetting does indeed occur in these systems, and that it can be understood quantitatively using their model. Their *in vitro* observations highlight the essential feature of this phenomenon: the presence of adhesion between a deformable membrane and a nanoscale fibre. Their observations also suggest that 1D wetting could occur more generally for physiologically important interactions of human cells with other biological nanofibres, and that it could have a major role in cell migration.

Further work is needed to understand 1D wetting in more detail. Systematic studies in which the fibre radius, strength of the adhesive interaction and surface tension of the membrane are varied would improve our understanding. In addition, further developments in microscopy will lead to better visualization of the structure and dynamics of the protrusions involved in 1D wetting.

Charles-Orszag and co-workers' results reveal opportunities for biomimetic strategies for wetting synthetic nanofibres and for producing strong adhesives, and new ways of moving nanoscale objects. Their findings also imply that reducing or disabling the 1D wetting of *N. meningitidis* T4P would limit the bacterium's ability to colonize and infect host cells, opening up a potential avenue for drug discovery. More generally, 1D wetting might enable cell function and health to be manipulated through interactions of cells with nanofibres to which biologically active molecules have been attached. ■

John R. Dutcher *is in the Department of Physics and the Biophysics Interdepartmental Group, University of Guelph, Ontario N1G 2W1, Canada.*
e-mail: dutcher@uoguelph.ca

1. Alberts, B. *et al. Molecular Biology of the Cell* 5th edn (Garland Science, 2008).
2. Charles-Orszag, A. *et al. Nature Commun.* **9**, 4450 (2018).
3. Coureuil, M. *et al. Virulence* **3**, 164–172 (2012).
4. Mikaty, G. *et al. PLoS Pathog.* **5**, e1000314 (2009).
5. Burrows, L. L. *Mol. Microbiol.* **57**, 878–888 (2005).
6. Melican, K., Michea Veloso, P., Martin, T., Bruneval, P. & Dumenil, G. *PLoS Pathog.* **9**, e1003139 (2013).
7. Maïssa, N. *et al. Nature Commun.* **8**, 15764 (2017).
8. De Gennes, P.-G., Brochard-Wyart, F. & Quere, D. *Capillarity and Wetting Phenomena: Drops, Bubbles, Pearls, Waves* (Springer, 2004).

This article was published online on 5 November 2018.

GENETICS

A genomic approach to mosquito control

A high-quality genome sequence for the mosquito *Aedes aegypti* has now been assembled. The sequence will enable researchers to identify genes that could be targeted to keep mosquito populations at bay. SEE ARTICLE P.501

SUSAN E. CELNIKER

Every year, millions of people are bitten by the mosquito *Aedes aegypti*. Thousands die as a result of infection by the viruses the mosquito carries[1], which can cause diseases such as yellow fever, dengue fever and Zika. Current mosquito-suppression methods typically involve pesticides. However, mosquitoes quickly develop resistance to these chemicals[2], and pesticides can accumulate in the food chain, with adverse effects on beneficial insects, other wildlife and humans. New control methods are therefore needed. On page 501, Matthews *et al.*[3] describe a high-quality genome sequence for *A. aegypti* (Fig. 1). This exemplary work could be a major step towards addressing our current inability to manage expanding mosquito populations.

Arguably the most promising alternatives to pesticide-based mosquito control are targeted molecular strategies based on genetics. The first requirement for the success of such strategies is high-quality sequencing of the mosquito genome. This would enable researchers to identify gene targets that could be manipulated to achieve a range of effects: to disrupt the mosquito's host-targeting systems; to make sterile males; to convert females into harmless males; or to render the insect incapable of harbouring viruses.

The repetitive nature of the 1.3-gigabase-long