PHOTONICS

Wingèd light

In man-made optical emitters such as LEDs, paradoxically most of the light remains trapped inside. So physicists have recently begun to exploit structures known as photonic crystals to try to extract more light. But while they have been trying to forge the most efficient devices, it now turns out that the swallowtail butterfly has already mastered the art of emitting light.

The eye-catching *Papilio nireus* butterfly from eastern and central Asia has wings emblazoned with iridescent blue-green patches (pictured). Pete Vukusic and Ian Hooper have used electron microscopy to learn more about the optical properties of these spots (*Science* **310,** 1151; 2005). Their images reveal that the scales on the butterfly's wings contain an intricate nanostructure of all-natural photonic crystals.

The crystals are just one part of a sophisticated photonic system that provides intense, directed light and endows the butterfly with its striking colours. A two-dimensional photonic crystal 'slab', about two micrometres thick, is set within the solid outer wing layer, and consists of hollow cylinders arranged in triangular domains. Highly fluorescent pigment, whose peak excitation wavelength matches that of the blue of the sky, is infused throughout the slab. And layered structures, known as Bragg reflectors, sit about 1.5 micrometres below the crystals.

Photonic crystals can stop light of specific frequencies from propagating within them. In this way, they can be used to limit or inhibit the emission of an optical emitter that is embedded deep within the crystal.

The optical properties of the crystals in this butterfly's scales are such that emission from the fluorescent dye is inhibited only within the crystal plane. Added to the effects of the underlying Bragg reflectors, this means the fluorescent light has nowhere to go but up and out. The effect is clearly seen in the optical-microscope image of a single scale (inset). Notably, similar techniques are used in the latest ultra-efficient LEDs.

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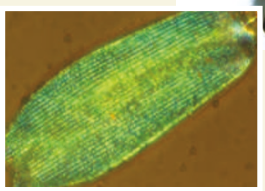

An astonishing variety of natural photonic structures are being uncovered not just in butterflies, but also in other insects, birds and fish. Although nature and technology have evolved independently, they have ultimately come up with the same design.

Amber Jenkins

OBESITY

Aquaporin enters the picture

Gema Frühbeck

The aquaporins are membrane channels that were originally identified as regulators of a cell's water balance. A member of the aquaporin family is now implicated as a central agent in controlling fat metabolism.

Mammals, like other organisms, have had to cope with feast and famine over evolutionary time. Hence the selection of genes that in times of plenty allow fat accumulation — principally in the form of triglycerides in fat cells — and in times of need promote the mobilization of those reserves. But the complex pathways involved in controlling this energy balance, which are implicated in the development of obesity and diabetes, are far from fully understood.

Hibuse *et al.*[1], writing in *Proceedings of the National Academy of Sciences*, add to the picture by showing that deficiency of a certain integral membrane protein in fat cells, called aquaporin-7, is associated with adult-onset obesity in mice. Together with previous work[2,3], the finding provides evidence not only that aquaporin-7 transports glycerol *in vivo*, but also that fat-cell permeability to glycerol is a key element in regulating fat accumulation.

The aquaporins are a family of proteins that facilitate the movement of water across the cell membrane, and in some cases they also act as a channel for molecules such as glycerol and other small solutes[4]. Aquaporin-7 is one such channel[5–7], and its synthesis in fat cells has been reported to be sensitive to various stimuli, including fasting and insulin.

Triglycerides consist of three fatty acids bound to a glycerol backbone. They are stored in fat tissue to serve as the body's principal fuel reserve. Fatty acids are mobilized to provide energy during prolonged exercise or starvation, and are used preferentially as fuel by muscle and liver. Glycerol, meanwhile, is both a triglyceride precursor, as glycerol-3-phosphate, and along with free fatty acids is one of the initial metabolic products of triglyceride mobilization. Following its release into the bloodstream from fat cells, glycerol is taken up mainly by the liver and converted to glucose. During fasting, glucose output from the liver is the main source of blood glucose, and the release of glycerol from fat cells is a major source of the necessary substrate. Thus, during fasting, effective systems are required to increase glycerol release from fat cells and its uptake by the liver. Loss of glycerol transport affects the levels of metabolites and hormones in the blood, and interferes with the triglyceride–fatty-acid cycle, as well as with glucose metabolism in the liver and in muscle (a glucose consumer).

The latest insights into aquaporin-7 function have largely stemmed from two independently created[1–3] lines of mice that lack the protein. In one set of findings[1,2], these knockout mice maintained similar body weights to normal mice until 12 weeks of age, at which time they started to accumulate fat and become heavier, even though their food intake was the same as that of normal mice. The other line of knockout mice[3] did not put on excess weight, a difference that Hibuse *et al.*[1] ascribe to the different genetic backgrounds of the two lines. But in other respects, such as the remarkable fat-cell enlargement seen, the mice showed similar responses to aquaporin deletion.

The enlarged fat cells resulted from the larger size of lipid droplets. Lipid synthesis and breakdown were unaffected by deletion of aquaporin-7, but there was a threefold decrease in glycerol release[3]. Thus, the reduced membrane permeability to glycerol results in an increase in steady-state glycerol concentration, which leads to increased glycerol-3-phosphate and hence triglyceride biosynthesis (Fig. 1). Activation of glycerol kinase, the enzyme that catalyses the conversion of glycerol to glycerol-3-phosphate, favours recycling of free fatty acids and results in a progressive accumulation of triglycerides[1–3].

In the latest work, Hibuse *et al.*[1] observed that 20-week-old obese knockout mice showed severely impaired glucose tolerance, seen as abnormally high blood-glucose levels. This was accompanied by decreased effects of insulin on fat tissue, liver and muscle. Such signs are characteristic of the whole-body insulin resistance associated with adult-onset obesity. Under normal circumstances, insulin