deinopoid spiders apply a more ancient dry adhesive to their capture silk; microscopically thin threads, known as cribellar fibrils, which are densely coiled around a core cable of capture silk. Curious to know how the dry cribellar adhesive impacts on the capture spiral’s ability to ensnare prey, Blackledge and Cheryl Hayashi began destruction-testing spiders’ webs (p. 3131).

But first, Blackledge had to convince spiders to spin webs in the lab. Having collected four cribellare spinning species from sites in Florida and near Hayashi’s University of California lab in Riverside, Blackledge provided the animals with comfortable accommodation to encourage them to spin. Fortunately all three genera were content to spin their webs, but collecting the intact structures wasn’t so easy. Blackledge explains that *Uloborus* spins horizontal disk shapes that were relatively easy to collect, while *Hyptiotes* and *Deinopis* actively hunt with their webs, distorting them as they trap their prey; *Hypoties* holds its triangular web taught, releasing it to entangle trapped victims, while *Deinopis* sits patiently overhead, ready to drop down and force its stretched web over unsuspecting passers-by. Knowing that both webs would be ruined if the spiders attempted an attack, Blackledge designed frames to capture the webs before they struck.

Having gathered the delicate structures, Blackledge collected short lengths of the dry composite spiral silk and measured the pseudoflagelliform fibres’ diameter to calculate the core’s cross-sectional area. They then carefully attached the dry composite spiral silk and measured the pseudoflagelliform fibres’ diameter to calculate the core’s cross-sectional area. Calculating the increasing load until the silk snapped. Blackledge explains that the development of stretchy core cables eventually snapped at twice their original length. Despite the broken core cables, the silk kept on stretching as the delicate cribellar fibrils remained intact until the silk had stretched up to five times its original length.

Comparing the thickness of the dry cribellate silk with capture silks coated in liquid adhesive, Blackledge realised that both silks were equally stretchy, but the dry silk’s core cable was nowhere near as stretchy as the core cable from liquid coated webs. Blackledge explains that early in evolutionary history, araneoid spiders also spun cribellate silks before abandoning them in favour of less costly liquid adhesives, and he suspects that the development of stretchy core cables could have allowed the arachnids to swap wet adhesives for dry.


**BUTTERFLY’S BLUE GENES**

Most people are lucky to encounter one surprise during the course of their research, but when Adriana Briscoe began investigating opsin gene duplication in butterfly eyes, she hit the surprise-jackpot. All Briscoe knew when she began investigating the expression of phototopic genes (opsins) was that the eyes of *Lycena rubidos* butterflies expressed four photopigments, rather than the three found in most other butterflies. Her long-time colleague, Gary Bernard, had also found that the distributions of these photopigments were different between the sexes. From this starting point Briscoe, Bernard and the rest of her team decided to clone the genes (p. 3079) to find out whether they were dealing with a gene duplication or an allele (slightly different copies of the same gene on different chromosomes).

Extracting mRNA from butterfly eyes, Marilou Sison-Mangus cloned all four butterfly eye opsins genes, and could clearly see that the extra opsin wasn’t an allele; one of the other three regular opsin genes had been duplicated to give rise to the extra photopigment. But which one? Briscoe explains that insect eyes usually express one ultraviolet (UV)-sensitive pigment, one blue-sensitive pigment and a long wavelength sensitive pigment. In most cases, it’s the long wavelength gene that has doubled up. But when Briscoe and the team aligned the butterfly’s gene sequences, they realised this couldn’t be the case. The extra gene had all the hallmarks of a blue opsin: surprise number one.

But which blue gene gave rise to which blue pigment? Knowing that the photopigments’ distributions were different in the male and female’s eyes, Briscoe decided to match the photopigments’ locations with the gene expression patterns to identify the gene that was responsible for the extra blue photopigment. Bernard mapped the photopigment distributions and found that the only blue opsin that occurred in the dorsal regions of both male and female eyes was the opsin tuned to 437 nm. Next, Marilou Sison-Mangus painstakingly explored each gene’s expression pattern with RNA probes and identified the blue genes responsible for the 437 nm and 500 nm photopigments.

The gene mapping also threw up the second and third surprises. Firstly, the opsin gene expression patterns in the dorsal region of the male’s eye were unique and unlike the patterns in any other butterflies’ eyes, and secondly, some visual receptors in the dorsal region of the female’s eye expressed two opsins simultaneously in a single cell. No one had ever seen a receptor cell expressing both blue and long wavelength opsins before; usually they only express one.

Finally, Briscoe explains that in most butterfly eyes each ommatidium is composed of 9 photoreceptor cells; 2 of the 9 cells (R1 and R2) express either the UV or the blue opsin, while the remaining 6 or 7 express the long wavelength opsin. The fourth surprise in this roller coaster ride came when Briscoe realised that by expressing different combinations of the two blue opsins and UV opsin in the R1 and R2 cells of the ventral eye *L. rubidos* has increased the number of ommatidia from the three found in most butterfly eyes, to six, Briscoe suggests that this increased colour sensitivity, coupled with the early evolution of the second blue opsin gene, could have driven many lycaenid butterflies to evolve their startlingly blue wings.