The team found that when the nuclear envelope deforms like an elastic solid when it is supported by a lamina. Furthermore, they found evidence of buckling folds that radiated out from the mouth of the pipette in the direction of tension, which would not occur if the lamina is a two-dimensional fluid. But what happens when the deforming force is removed – does the nucleus spring back to its original shape? For quick deformations, Rowat’s team found that the answer is yes, but for longer deformations (more than ten seconds), relaxation is slower. In other words, like most biological materials, the nuclear lamina is best described as a viscoelastic solid.

This work could have profound implications for understanding a mysterious group of genetic diseases known as laminopathies, which are caused by mutations in the lamin-encoding gene. These diseases are typically characterized by defects in muscle, adipose and nervous tissues, but one unique variant causes a rare syndrome in which patients appear to age prematurely and die before their teens. The cell nuclei of patients with laminopathies often exhibit irregular shapes and there is evidence that they are mechanically fragile. Rowat’s team is next going to measure how nuclei isolated from these patients stand up to mechanical stress, which is an important first step to understanding the mechanisms underlying laminopathies. Furthermore, by exploring what goes wrong when nuclear lamina function is disrupted, these studies will illuminate the selective pressures that were responsible for the appearance of the nuclear lamina in higher eukaryotes.

10.1242/jeb.01650


Douglas Fudge
University of British Columbia
dfudge@interchange.ubc.ca

The arthropod exoskeleton is both a blessing and a curse. It provides mechanical protection, a permeability barrier and a lightweight skeleton; but it must be moulled regularly as it becomes constricting when the animal grows. The multi-stage moulting process involves the growth of a new flexible cuticle underneath the old one, the detachment from and shedding of the old one (ecdysis), then the expansion of the new one before it is finally tanned – that is, made rigid by chemical crosslinking. Classic neck-ligation experiments (tying the neck with ligature silk to prevent soluble brain factors from reaching the rest of the body) showed that brain-derived factors are responsible for moulting. Since then, it has become clear that moulting is under multiple, sequential neurohormonal control. In March this year, two research groups simultaneously revealed an important missing link in this pathway – the last step in the process, the tanning hormone bursicon.

Bursicon is released from the insect’s head after ecdysis; if an insect’s neck is ligated soon enough after moulting, its cuticle does not tan. Painstaking work has shown that bursicon acts through the second messenger cyclic AMP. Second messengers signal the arrival of hormones (like bursicon) at the cell surface to target molecules in the cell. Bursicon’s effects can be mimicked in neck-ligated insects by injecting their abdomen with either cyclic AMP analogues or a central nervous system-derived peptide fraction of around 30 kDa in size, too large to be a neuropeptide. That is, bursicon is a protein hormone, explaining why it has proved hard to identify.

To determine bursicon’s peptide sequence, one of the groups laboriously purified