

## BEHAVIOURAL ECOLOGY

## Push on the marching crickets

Stephen J. Simpson and colleagues have a gory story to tell (*Proc. Natl Acad. Sci. USA* doi:10.1073/pnas.0508915103; 2006). They investigated the factors that contribute to the mass movements of flightless Mormon crickets (*Anabrus simplex*), which inhabit western North America. Under certain conditions, these insects form into marching bands millions strong and several kilometres in length, and can walk up to 2 kilometres a day.

From their studies, Simpson *et al.* conclude that group movement is driven not only by 'pull' from in front, in the form of the search for specific nutrients (protein and salt), but by 'push' from behind — cannibalism. The crickets are themselves "walking packages of protein and salt". So unless an individual is in the forefront of the band as it rolls into new territory, its only recourse to satisfy its dietary needs may well be to make a meal of a fellow traveller. As this picture shows, Mormon crickets have a

taste for their own. It was taken near Reno, Nevada, and shows one unfortunate individual, killed by a car, being consumed by another cricket (a phenomenon that sometimes occurs on a mass scale).

To identify which nutritional components might drive mass movement, Simpson and colleagues conducted feeding-preference tests for nutrients placed in the path of a marching band. The results showed, for example, that marching crickets have a decided requirement for protein over carbohydrate, and for salt solution over water (to the extent that the insects fought for the preferred salt concentration).

Other experiments demonstrated that crickets that had had their fill of protein and salt were much less likely to be cannibalistic. And other experiments again, using crickets with compromised movement, showed that the crickets safest from cannibal attacks were those that were able to defend



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themselves with their hind legs. But inactivity or reduced mobility meant that the cricket concerned was literally dead meat.

Cannibalism presumably sets a high cost on group life, so why do the bands of mormon crickets stick together? Simpson *et al.* answer this question by pointing to previous work involving the use of radiotelemetry which showed that the price of leaving the

migratory group is the high likelihood of being killed by a predator. Better, then, to opt for the protection conferred by being one of a crowd, even at the risk of being consumed by one of your own — or, as the authors put it, being a member of a cricket migratory band "is a compromise that makes the best of a seemingly very bad situation".

**Tim Lincoln**

## CELL BIOLOGY

## A break in the chain?

Keith Burridge

**How chains of proteins link transmembrane cell-cell adhesion molecules to the cell's inner scaffold was standard textbook material. But recent research challenges the accepted model, opening a new chapter in the field.**

Many of the adhesion molecules that enable cells to stick together are anchored to part of the cell's internal scaffold — the actin cytoskeleton. This association is usually indirect and serves to transmit tension to sites of adhesion, to cluster adhesion molecules and to provide a framework for the assembly of complexes of signalling proteins. How the adhesion molecules connect to the cytoskeleton has often been difficult to define, but one of the most widely accepted chains of attachment is that of the cadherin adhesion proteins to actin filaments through the  $\beta$ -catenin and  $\alpha$ -catenin proteins, with  $\alpha$ -catenin providing the link to actin (Fig. 1). (Indeed, 'catenin' derives from the Latin for chain.) Two *Cell* papers from the groups of W. James Nelson and William Weis<sup>1,2</sup> challenge the validity of this linkage, and particularly the role of  $\alpha$ -catenin.

Cadherins mediate cell-cell adhesion in many cell types. Much of what is known about

these proteins derives from work on E-cadherin, the type found in the epithelial cells that line ducts and cavities in the body. E-cadherin is concentrated within cell-cell contacts known as adherens junctions, and the extracellular domains of the protein hold adjacent cells together. Adherens junctions associate with a circumferential belt of actin filaments around the cell, and together these structures determine the shape of epithelial cells and maintain epithelial integrity. During tissue development, contraction of the circumferential belt can transmit tension to adherens junctions, causing cells to constrict in their apical regions, inducing the epithelium to curve or fold. This underlies the generation of structures such as the neural tube, which develops from a localized folding of the neural epithelium and eventually goes on to form the brain and spinal cord.

The first indications that cadherins might be

linked to actin filaments came from the localization of adherens junctions with bundles of actin filaments. Furthermore, cadherins partitioned with much of the cytoskeleton in crude subcellular fractionation procedures. Then, nearly 20 years ago, cadherins were shown to interact with at least two cytoplasmic proteins,  $\alpha$ - and  $\beta$ -catenin. Binding studies demonstrated that  $\beta$ -catenin forms a bridge between cadherin and  $\alpha$ -catenin, which in turn binds to actin *in vitro*. From these binary interactions, a chain of attachment seemed logical (Fig. 1), and this model was accepted by the field (and textbooks) with little challenge, except for the qualification that many other actin-binding proteins also bind to  $\alpha$ -catenin<sup>3</sup> and might contribute to this linkage.

The Nelson and Weis groups<sup>1,2</sup> provide several lines of evidence that are inconsistent with this textbook linear model. They report<sup>1</sup> that  $\alpha$ -catenin does not bind simultaneously to actin and to  $\beta$ -catenin in complex with the E-cadherin intracellular domain. They developed a system to test the binding of purified components to 'membrane patches' enriched for cadherins but stripped of the associated proteins. Although  $\beta$ -catenin bound to these preparations and conferred  $\alpha$ -catenin binding, these complexes were unable subsequently to bind to either G- or F-actin.

Recognizing that additional  $\alpha$ -catenin-binding components may be required, the