

## Plant ecology

## Fertile arguments in the desert

from Jonathan Silvertown

ECOLOGISTS have been arguing recently about whether interspecific competition is responsible for observed patterns of animal distribution and abundance<sup>1,2</sup>. Whereas interspecific competition once seemed to be the only process of interest to the theoretically minded, now dispersal, predation, mutualism and biogeographic history are also being considered (see ref. 1). Strangely, the recent debate has occurred almost exclusively among animal ecologists but similar arguments among plant ecologists now seem to be approaching the same conclusion.

Plant ecologists studying species distri-



bution in deserts have investigated the cause of the regular spatial patterns of distribution often found in the southwestern deserts of North America and elsewhere. The commonest shrub in the Chihuahuan, Sonora and Mojave deserts is the creosote bush *Larrea tridentata*, which is a conspicuous part of the sparse vegetation. Bushes, or clumps of bushes, commonly have large areas of bare ground between them and often appear regularly spaced. F. W. Went suggested that this regular spacing is the result of allelopathic (toxic) interactions between bushes<sup>3</sup>. Since then, in one of the first quantitative analyses of the dispersion pattern of *Larrea*, S. R. J. Woodell *et al.*<sup>4</sup> showed that *Larrea* bushes are indeed regularly spaced at sites of low rainfall but are aggregated at sites of high rainfall. In an alternative hypothesis they suggested that the regularity is the result of competition for water between roots of adjacent bushes, with reduced competition at sites of high rainfall allowing aggregation.

M. G. Barbour<sup>5,6</sup> found that regular patterns of *Larrea* were in fact quite rare. D. J. Anderson<sup>7</sup> further questioned Woodell *et al.*'s claim that plants in lower rainfall areas compete for water, because bushes at these sites are at such low density that each plant actually has more available soil water than in high rainfall areas where bushes are more crowded. T. J. King and Woodell<sup>8</sup> replied that the quantity of water available to bushes cannot be calculated simply from density and rainfall measurements because plant size,

surface-water runoff and infiltration of water into the ground also has to be considered. They also suggested that periods of severe drought in the past might at some sites be responsible for present-day distributions. Thus, in defending the competition hypothesis, they acknowledge the importance of other factors in structuring *Larrea* communities, as animal ecologists have had to do in defence of similar arguments about interspecific patterns<sup>9</sup>.

The parallel with the debate among animal ecologists can be taken further. Reassessment of competition in animal ecology has been stimulated by the use of 'null models'<sup>10</sup> that predict how community structure would appear in the absence of competitive interactions between species. T. A. Ebert and G. S. McMaster<sup>11</sup> have used this approach to challenge the regularity of spacing patterns in *Larrea*, which they believe may be no more than an artefact of sampling methods, by which plants that grow very near one another are recorded as a single individual. They show that this can cause a random pattern to appear erroneously as a regular one. King and Woodell<sup>11</sup> dispute this claim, but do not explain how the competition hypothesis accounts for regular spaced clumps.

A general conclusion from the use of null models in animal ecology has been that patterns can only provide correlative evidence of the processes that structure communities. Experiments are needed to test hypotheses generated by analysis of pattern distribution. Several relevant experiments have now been performed with desert shrubs. In the field, removing neighbours of individuals of both *Larrea*

and another desert shrub, *Ambrosia dumosa*, affects the physiological water status of other plants<sup>12,13</sup>. Even though this is evidence of competition for water, some of the experimental populations were aggregated. Clearly, competition does not lead inevitably to regular spacing.

We can say more than this. The desert perennial *Eriogonum inflatum* with competitors nearby is more likely to die than those plants with more distant neighbours, yet this does not change the degree of aggregation in the population. The most elaborate recent experiment<sup>14</sup> shows how uncertain the role of root competition in producing regular spacing has become. Despite a fall in shrub density, W. H. Schlesinger and C. S. Jones found no change in the dispersion pattern of *Larrea* and *Ambrosia* at a Mojave site that has been deprived of surface water runoff by a drainage system for 45 years.

Ironically, the unknown causes of regular spacing in desert plant communities may bear some relationship to the way in which animal and plant ecologists have ignored each other. Few have considered, for example, that these plant patterns could result from the activities of seed-gathering rodents<sup>16</sup> which forage and remove seeds between bushes and which are ubiquitous in these deserts. □

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## Excitation-contraction coupling

## The messenger across the gap

from Andrew P. Somlyo

EXCITATION of the surface membrane of muscle results in release of calcium that is stored in an intracellular membrane system, the sarcoplasmic reticulum (SR); the  $\text{Ca}^{2+}$  released into the cytoplasm, acting through regulatory sites on Ca-binding proteins, initiates contraction. The signal from the membrane is communicated to the SR at specialized regions (triads, diads or surface couplings) where the cytoplasmic leaflets of the SR are connected to the surface membranes by quasi-periodic bridging structures across a 12–20 nm gap (refs 1,2; see figure). A major unresolved problem is the mechanism by which depolarization (or some other change in the surface membrane) is communicated

across this gap, to increase  $\text{Ca}^{2+}$  release from the SR lumen into the cytoplasm. Hypotheses to answer this question have been proposed and some, like Peter Pan, retain their youth with sustenance from the Never Land. Will the suggestion that inositol trisphosphate is the messenger across the gap, as put forward for skeletal muscle on page 347 of this issue<sup>3</sup>, stay forever young?

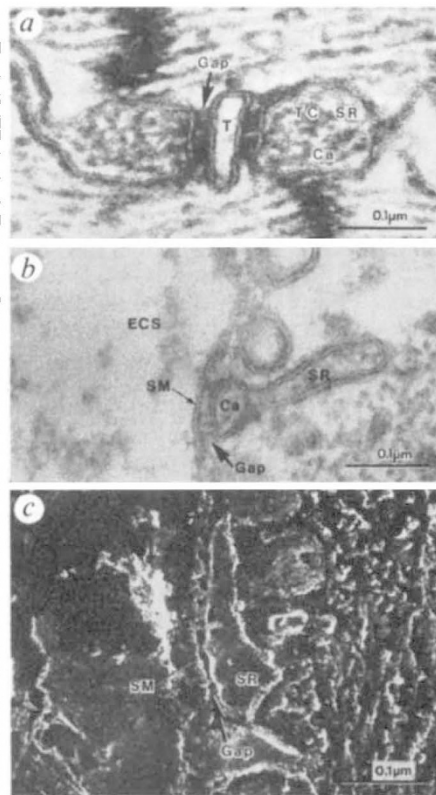
Three main classes of mechanisms have been suggested for excitation-contraction (EC) coupling: first, direct spread of ionic current from the extracellular space through the bridging structures into the SR<sup>4</sup>; second, mechanical transduction via a mobile protein between the two mem-

brane systems, initiated by voltage-dependent movement of fixed electric charges in the surface membrane<sup>5</sup>; and finally, chemical transmission by a diffusible second messenger. The absence of an ionic current between the extracellular space and the SR together with the fact that  $\text{Ca}^{2+}$  release is not electrically silent<sup>6</sup> have excluded the first mechanism<sup>4,6</sup>, and no experimental test of the second hypothesis has yet been performed. Meanwhile, as the idea of EC coupling by chemical transmission is becoming increasingly attractive, much recent evidence suggests that inositol 1,4,5-trisphosphate ( $\text{InsP}_3$ ) is the second messenger<sup>7</sup> in many cells. In non-muscle cells,  $\text{InsP}_3$  is released by the transmitter-initiated hydrolysis of polyphosphatidyl inositol in the surface membrane and it is thought to mediate transmitter-induced calcium release from the endoplasmic reticulum, a structure that is homologous with SR in muscle. The inevitable question is whether  $\text{InsP}_3$  is also the messenger in EC coupling.

When considering EC coupling, it is important to remember that there is an additional mechanism involved in smooth muscle. Whereas in both smooth muscle and striated (skeletal and cardiac) muscle contraction is triggered by electrical depolarization of the surface membrane and its invaginations (the T-tubules) and usually by an action potential, in smooth muscle, neurotransmitters also stimulate contraction or cause relaxation through a 'pharmacomechanical coupling' independent of changes in membrane potential.

Until the discovery of  $\text{InsP}_3$ , the most popular candidate as the messenger in EC coupling was  $\text{Ca}^{2+}$  itself. According to this theory the rapid influx of small quantities of calcium current carried by the action potential triggers the 'calcium-induced calcium release' from the SR, which provides the larger quantity of  $\text{Ca}^{2+}$  required for initiating contraction. The strongest evidence for calcium-induced calcium release<sup>8</sup> is in cardiac muscle, much of it from the elegant studies of Fabiato<sup>9</sup>. In skeletal and smooth muscle, however,  $\text{Ca}^{2+}$  can be released from the SR even in the absence of extracellular  $\text{Ca}^{2+}$  (and, therefore,  $\text{Ca}^{2+}$ -influx); nevertheless it is still possible to argue that trigger calcium is released from a protected region, perhaps the cytoplasmic leaflet of the plasma membrane<sup>4,10</sup>.

The new experiments<sup>3</sup> show that in rabbit skeletal muscle,  $\text{InsP}_3$  satisfies the first requirement of an EC coupling messenger — it can release  $\text{Ca}^{2+}$  from the SR. This is demonstrated both with measurements on isolated SR vesicles and by the contractile response of skinned (membrane-free) muscle fibres to  $\text{InsP}_3$ . However, such contractions are small, slow and somewhat variable, and seem to require abnormally low free  $\text{Mg}^{2+}$  concentrations to prevent the rapid breakdown of  $\text{InsP}_3$ . None of these objections, as Volpe *et al.* and



Electron micrographs of a triad in striated muscle (a) and surface coupling in smooth muscle (b, c); c has been freeze-etched and rotary shadowed to give a clear view of the junctional gap between the sarcoplasmic reticulum and surface membranes. T, T-tubule, an invagination of the extracellular space in striated muscle; TC SR, terminal cisterna of the sarcoplasmic reticulum; SM, surface membrane of smooth muscle (a from ref. 2; b, c, from ref. 19).

others taking a similar approach<sup>11</sup> would argue, are insurmountable. For example, in live muscle,  $\text{InsP}_3$  may be released into the diffusionally-restricted junctional space between the SR and surface membranes (see figure); this could cause a rapid and large rise in the local concentration of  $\text{InsP}_3$  near the terminal cisternae of the SR where  $\text{Ca}^{2+}$  is thought to be released. Such regions could even contain lower concentrations of free  $\text{Mg}^{2+}$  and of the ( $\text{Mg}^{2+}$ -dependent) phosphatases that break down  $\text{InsP}_3$ . If the electron-lucent core of the bridging structures is a lipid, as has been suggested<sup>2</sup>, it could contain  $\text{InsP}_3$  precursors. But, for now at least, these are *ad hoc* explanations of the failures of experiments to support the hypothesis.

A more devastating blow to the role of  $\text{InsP}_3$  as a physiological messenger of electromechanical coupling in skeletal muscle would be if depolarization were found to increase only the turnover of (mono)phosphatidyl inositol, and not that of the polyphosphatidyl inositol precursor of  $\text{InsP}_3$ , and/or if an increase in  $\text{InsP}_3$  were shown to be a slow side-effect of acetylcholine released by nerves, and did not precede twitch contraction. The increased phosphatidyl inositol turnover evoked in frog skeletal muscle by depolarization with potassium is far more prolonged than

the duration of  $\text{Ca}^{2+}$ -release<sup>12</sup>, and  $\text{InsP}_3$  seems to cause only very modest calcium release from isolated cardiac SR<sup>13</sup>.

The most compelling evidence for a physiological role of  $\text{InsP}_3$  in muscle is neither in electromechanical coupling nor in striated muscle, but as a transmitter of pharmacomechanical coupling in smooth muscle. In these tissues various physiological transmitters (cholinergic, adrenergic and peptides) can stimulate the breakdown of polyphosphatidyl inositol to produce  $\text{InsP}_3$  (refs 14–16). It is important to note that depolarization does not stimulate breakdown of polyphosphatidyl inositol in smooth muscle<sup>14,15</sup>, whereas cholinergic agents can stimulate such breakdown during pharmacomechanical coupling<sup>15</sup>. The release of calcium from smooth muscle cells by  $\text{InsP}_3$  has been demonstrated in two laboratories using different methods<sup>17,18</sup>. Furthermore, low micromolar concentrations of  $\text{InsP}_3$  can cause sustained, graded contractions of smooth muscle even in the presence of the highest free  $\text{Mg}^{2+}$  (1–2 mM) likely to be present *in vivo*<sup>18</sup>. Therefore, it is probable that  $\text{InsP}_3$  mediates at least the part of pharmacomechanical coupling that is caused by a voltage-independent release of  $\text{Ca}^{2+}$  from the SR, and seems to be similar to  $\text{Ca}^{2+}$  release from the endoplasmic reticulum of non-muscle cells.

It remains to be determined whether the effects of  $\text{InsP}_3$  on the SR of skeletal muscle will turn out to be a major physiological mechanism or simply an interesting laboratory phenomenon like the effects of fatty acids and adenine nucleotides, which can release  $\text{Ca}^{2+}$  or potentiate  $\text{Ca}^{2+}$ -induced  $\text{Ca}^{2+}$ -release. The new findings will, at the least, stimulate the hunt for an identifiable messenger of EC coupling. In the background, like the crocodile following Captain Hook, the spectre of a more physiologically active inositol phosphate lurking in  $\text{InsP}_3$  preparations haunts investigators in this exploding field. □

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