Desert ecosystems

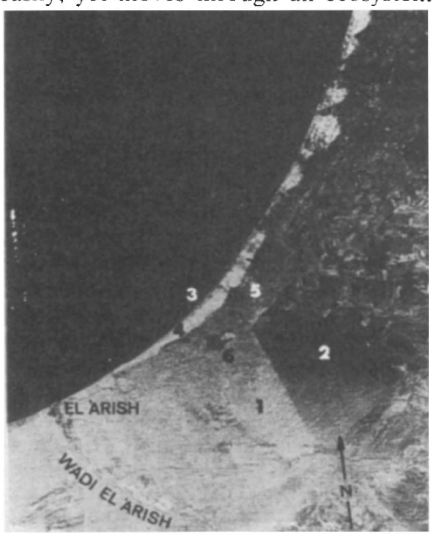
Desertification or xerification?

from N.E. West

'DESERTIFICATION' has become a common term to describe the impoverishment of terrestrial ecosystems induced by man. But the word is much abused, often preventing the realization that deserts occur naturally in certain ecological contexts. Although the impression that deserts are produced by man helps to generate interest, and therefore money, to investigate problems of vegetation diminishment and soil degradation, particularly in regions of semi-arid climates[1,2], it encourages emphasis on treating the symptoms of abuse rather than the causes of the problems.

Deserts vary enormously around the world, with only some of those under the most arid conditions having escaped severe alteration by man[3,4]. Le Hourerou[5] proposed the term 'desertization' to imply the expansion of desert-like landforms and landscapes in arid to semi-arid environments. This word is supposed to avoid issues such as ecological degradation of tropical forests into savanna of forest into maquis, which can still be described as 'desertification'. But the distinction is probably missed by most and, in any case, degradation of terrestrial ecosystems usually does involve drying-out of the previous environment. Water typically enters the biological phases less easily, yet moves through an ecosystem more rapidly, following disturbance introduced by man. If soils are more compacted and organic matter lower, less water can infiltrate and be stored. Accelerated erosion of the soil surface may reduce the depth of the rooting profile as well as removing the most nutrient-rich portions. More xerophytic (drought-tolerant and less water-demanding) plants typically replace the preceding comparatively less xerophytic ones removed by excessive livestock grazing, wood harvest, fire and mechanical damage. This gives the impression that macroclimates must have changed when the major alterations in climate have occurred at soil level. Kovda[6], therefore, proposed the term 'aridization' to describe the drying-out process. The disadvantage of this word is that it can be confused with a permanent change in macroclimate.

I have recently used the term 'xerification' to describe the concomitant diminishment of vegetation and degradation of soils under unsustainable levels of land use in semi-arid areas in western North America[7]. This term could be used in more climatically favourable regions without the value-judgement implicit in



LANDSAT imagery of the boundary between Egypt and Israel as it appeared in 1973. 1, sand, vegetation destroyed; 2, sand occupied by intact vegetation; 3, Mediterrean Sea; 4, shifting sands next to coastal strands; 5, sandy area covered by orchards in southern part of Gaza; 6, fenced area of Sadat and Vetiv ha Asara where grazing and direct plant cutting ceased after 1967. From Danin, A. *Desert Vegetation of Israel and Sinai* (Cana, Jerusalem, 1983). Image provided by NASA.

Albert L. Lehninger (1917–1986)

ALBERT LEHNINGER, who died on 4 March, is known to young and not so young biochemists all over the world as the sole author of the widely used textbook, simply called *Biochemistry*, the first edition of which appeared in 1970. To the older biochemist and to all those engaged in the study of the branch of biochemistry and biophysics called bioenergetics, he is known as one of the founders of their field. He was one of the first to publicize the word bioenergetics in his monograph bearing that name, published in 1965. His first book, *The Mitochondrion*, had appeared a year earlier.

It was his work on this sub-cellular organelle in the late 1940s and the 1950s that first brought him fame. Together with E.P. Kennedy, he showed in 1948 that a particulate fraction of rat liver, identified as mitochondria, catalyses all the reactions of the Krebs cycle and fatty acid oxidation. For this work Kennedy and Lehninger are co-recipients of the 1986 Passano Foundation Award.

Lehninger is perhaps best known for his second classical paper, also published in 1948. In that paper, together with his student Morris Friedkin, he described phosphorylation of adenosine diphosphate by inorganic phosphate, coupled to electron transfer between reduced nicotinamide nucleotide and oxygen (catalysed by the so-called respiratory chain). The phenomenon of oxidate phosphorylation itself had already been established by Engelhardt and Kalckar in the 1930s and before the Second World War Ochoa and Belitzer had made it likely that it was of a fundamentally different type from the only known phosphorylation reaction, linked with anaerobic processes. But the direct demonstration of what became known as respiratory-chain phosphorylation had proved elusive. Lehninger succeeded in two types of experiments, one with reduced nicotinamide-adenine dinucleotide as substrate and the other with 3-hydroxybutyrate, which reduces the nucleotide in a reaction that could not conceivably be associated with phosphorylation.

With the hindsight of nearly 40 years, we now know that the results of the first type of experiment were not as unambiguous as they at first appeared, but the second type remain valid and were sufficient to establish the concept of respiratory-chain phosphorylation. Lehninger was also the first to demonstrate phosphorylation in the terminal reaction of the respiratory chain — the oxidation of reduced cytochrome *c* — once again after failures by others. He was also one of the first to observe the energy-linked uptake of calcium by mitochondria and the first to make a thorough study of its characteristics.

Lehninger was one of a vanishing type: the highly successful research worker who is also a prolific writer. Also unusual is that all his books are monographs. How he was able to face up to the tremendous chore of regularly bringing out new editions of his monumental textbook was a source of wonder to many. Surely the explanation is that he enjoyed writing. That he was a such a lucid writer was no surprise to those of us who used to listen spellbound to his lectures at international meetings, delivered without any notes. The polish of these performances could only have been achieved with meticulous preparation.

Lehninger's early work was done at the University of Chicago where he moved in 1945 after obtaining his Ph.D. at that nursery of American biochemists at that time, the University of Wisconsin. He spent the academic year 1951–1952 at the University of Cambridge, England, and in 1952 was appointed Professor of Physiological Chemistry at Johns Hopkins University, Baltimore, where he stayed until his death. His colleagues at Johns Hopkins had planned a symposium, appropriately called *The Mitochondrion, 1986*, to be held in his honour on 5–7 June. The symposium will still be held, now sadly to his memory as well as in his honour. E.C. Slater

words such as desertification, desertization and aridization.

Irrigated lands in arid to semi-arid climates which have increased water tables and/or direct salt input from the irrigation water eventually support lowered crop production. Even some originally forested lands in southern Australia develop such problems when the high transpiration draw-downs affected by the forest are eliminated after tree removal. The only plants that can grow on some of the most heavily salinized former farm- or forestlands are shrubs normally found in desert regions. The soil moisture is excessive, but the soils are physiologically dry to non-desert species. The term xerification would more usefully describe these processes than the narrow word salinization.

Clearly it will be difficult to distinguish natural xerification from that induced by man. But palaeoecological evidence shows that deserts are relatively young ecosystems on all continents, and thus have arisen naturally. Therefore it is important to elucidate natural, as well as man-made, mechanisms. □

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Muscle contraction

Crossbridges, force and motion

from Richard T. Tregear

THE basic question that concerns those interested in crossbridges is 'what makes muscle pull?'. Many different answers have been suggested over the years. Some simple and elegant experiments described at a recent workshop* exclude several of them. Other experiments place constraints on the part of the myosin head (or crossbridge) responsible for the generation of force consequent on ATP hydrolysis. The picture which emerges is of a crossbridge (which projects from the thick filament of muscle fibres) that can easily attach and detach from actin (in the thin filament) in the first part of the enzymatic cycle and then 'harden' its attachment and produce force in the later part of the cycle. The contractile process occurs in the middle of the myosin head, does not need the other head of the myosin molecule, much of the tail region of the molecule or even, perhaps, the light chains of the head itself. The process probably involves only a small change in the shape of the head, is reversible and can sometimes couple to a huge interfilament movement. Altogether this picture is not very like the usual undergraduate idea of crossbridge rotation and force production.

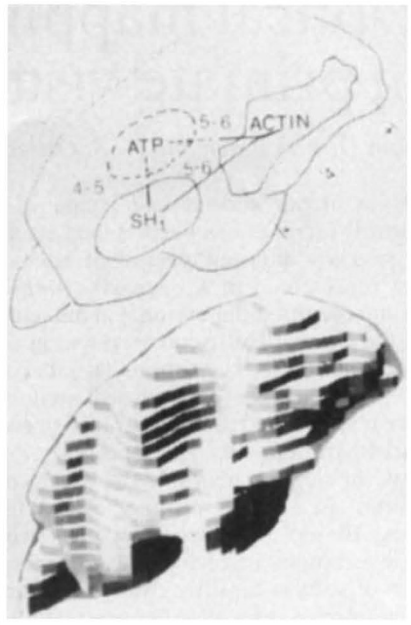
The evidence for these statements comes from a wide variety of approaches. The idea of the rapidly attaching weak-binding crossbridge comes from enzymatic studies (E. Eisenberg, NIH), and fits both with the way in which measurements of crossbridge attachment by X-ray diffraction (H. Huxley, Cambridge; K. Wakabayashi, Osaka) and mechanical stiffness (R. Simmons, London) are seen to rise before tension during activation and with the large number of disorganized myosin heads still seen by electron proton resonance (EPR) when a muscle is contracting strongly (D. Thomas, Minneapolis). On the other hand, the strong-binding, tension-generating state may be best seen by electron microscopy of muscle in rigor (M. Reedy, Duke); at any rate, no regular crossbridge angle other than that of rigor (thought to represent the maximal force-producing state) has been detected by EPR or polarized fluorescence in actively contracting muscle.

The resolution of immuno-electron microscopy is now good enough to divide the myosin head into a regulatory neck and an operative body, and within that body to locate the three regions most likely to transfer energy across it: the nucleotide- and actin-binding regions and the interlinking part close to the active thiol residue (T. Wakabayashi, Tokyo; see figure). Furthermore, both the sequence near the thiol and the purine-binding sequence have turned out to be highly conserved, being invariant from slime mould to rabbit, as if they perform an essential function (J. Spudich, Stanford).

Optical microscopy has now improved to such an extent that one can see the motion of unrestrained actin filaments over myosin, or of particles coated with myosin over actin (Spudich; T. Yanagida, Osaka). Such motion occurs even when the myosin is single-headed or its tail is greatly reduced in length. These experiments appear to eliminate contractile mechanisms dependent on either head–head interaction[1] or a change in tail structure[2].

Chemists have managed to attach a photolysable group to ATP (caged ATP) that can be diffused into muscle before lysis releases the ATP itself[3]. The resultant synchronized events indicate that phosphate release and tension generation occur together and that both processes are reversible (Y. Goldman, Philadelphia). It follows that tension can be regenerated without coupled ATP hydrolysis, which was not envisaged in earlier ideas of the crossbridge cycle[4].

An ingenious argument was presented from a couple of conceptually simple experiments. If actin is proteolytically cut loose in a myofibril or if myosin heads are floated into a brush of actin filaments, they remain still until ATP is added and then they move, quite rapidly. Yanagida measured the speed of motion and the rate of ATP hyrolysis and deduced that each crossbridge stroke using ATP moves the myosin molecule by at least 60 nm, which is too far for the crossbridge to stretch[5,6]. Is there something wrong with Yanagida's logic, or with the crossbridge theory itself? □



Location of the ATP-binding and reactive thiol (SH_1) sites on the surface of the myosin head (top), determined from avidin binding and three-dimensional reconstruction of the actin-myosin head complex (bottom). ATP binds to the back of the head. Units, nm. (Courtesy of M. Tokunaga, A. Tomioka, C. Toyoshima, K. Sutoh, K. Yamamoto & T. Wakabayashi, unpublished observations.)

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* Workshop on the interaction between actin and myosin in skeletal muscle held at Alpbach, Austria, 17-22 March 1986. Supported by the European Molecular Biology Organization and the Muscular Dystrophy Association of America.