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## Book Reviews

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*Evolutionary Genetics and Environmental Stress.* By A. A. HOFFMANN and P. A. PARSONS. Oxford University Press. 1991. Pp. ix + 284. Hardback £35. ISBN 0 19 857732 X.

Organisms are frequently said to be 'stressed' by events such as anoxia, chemical pollution and heat shock, but it is often not clear if there is any real connection between them. Hoffmann & Parsons make a convincing case that the idea of stress provides fundamental insights into evolutionary responses to potentially lethal environmental conditions. Part of the clarity in the text comes from the very early definition of stress, which is exposure to any potentially injurious physical factors. In its early stages a stressor causes a temporary reduction in survival probability or fertility, but persistent stress results in permanent damage and, eventually, death in susceptible individuals. Stress therefore involves conditions worse than the merely suboptimal. It is of evolutionary interest because it produces periods of intense selection, sets the limits to the geographic distributions of species and has the potential to cause extinction, as attested both by the fossil record of extinctions and by documentation of population crashes in response to physical stressors. Work on stress has been given a new lease of life (and funding) by intimations of global warming.

Organisms can respond to stress either by evasion tactics such as dormancy or migration, or by resistance, such as the detoxification of an insecticide. Both types of response can be facultative, as occurs in the induction of heat shock proteins or fixed, as in the colour morphs of the snail *Cepaea*. Studies of responses to stress therefore include work on environmental physiology, as well as genetic analysis. The mechanisms of stress resistance in individual cases are occasionally known. For instance, a number of studies have revealed spatial associations between particular enzyme variants and environmental gradients. More often, mechanisms are not understood, and it is not clear if there are generalisations to be made across different organisms. The consistent association between the environmental temperature encountered by species or individuals and the temperature optima of their enzymes and membranes provides a notable exception. Stress-resistance is usually studied using either comparisons of species

and populations or the techniques of quantitative genetics to analyse some measure of fitness in the presence of the stressor. These techniques have been used to demonstrate associations between geographic variation in stress resistance and the presence of the relevant stressor, and the presence of substantial genetic variation for stress resistance in natural populations.

One important issue is the conditions under which responses to stress are expected to be phenotypically plastic, through changes in individual behaviour or physiology, as opposed to a fixed attribute of a particular genotype. Sometimes these may be alternatives, if they share common pathways, as appears to be the case for desiccation resistance in *Drosophila melanogaster*. Theoretical work suggests that frequent encounters with a stressor, rapid onset of stress and high costs of plasticity may all favour a fixed resistance mechanism. There has been very little empirical work on this topic, and it is a potentially fruitful area for future experimental studies.

The concept of stress becomes an even stronger one if different stressors can be combatted by the same mechanisms. Sometimes these are highly specific, as in the biochemical pathways involved in detoxification of insecticides. General mechanisms do none the less occur. The heat-shock proteins (hsps) were named as such because they were first observed in cells exposed to temperature stress, but they can also be induced in response to a number of other stressors such as heavy metals, anoxia, ethanol and cold shock. The mechanisms by which these very different physical variables can induce the same set of genes are of great interest. The presence of denatured or otherwise abnormal proteins in cells may be part of the trigger. There is little information on the effect of hsps or the loci producing them on resistance to these other stressors. As well as the hsps themselves, the heat-shock loci produce factors that bind to many other loci in the genome, and halt transcription, preventing protein-synthesis while protein structure is compromised. Another general mechanism of stress resistance may be lowered metabolic rate, since species and genotypes resistant to a variety of stressors have a lower metabolic rate than susceptible ones when both are reared in a benign environment. Similarly, plants resistant to ecological stressors such as low phosphate levels or intermittent flooding usually have slow

growth rates under optimal conditions. Some caution is required in interpretation of these effects, because slow growth could be a consequence rather than a cause of resistance. None the less, these findings are crying out for further study at the biochemical level, to understand exactly how these traits are connected to stress resistance. Both mutation and recombination rates often increase in response to stressors such as low and high temperatures. These again could be adaptive mechanisms to increase genetic variation of maladaptive side effects of exposure to stress.

Several lines of evidence suggest that genes causing resistance to stress may incur a pleiotropic cost, because the resistant phenotypes are at a disadvantage to susceptible ones in the absence of the stressor, especially in competitive conditions. This may be another reason why many responses to stress, such as the induction of heat shock proteins, are inducible rather than constitutive. Resistance may be metabolically costly if, for instance, ATP is used to move ions against a concentration gradient, leaving less energy for reproduction. Conflicts between vital processes may also occur. Xeric plants are in general incapable of high rates of photosynthesis, because their stomata are adapted to prevent water loss, which also limits the rate of gas exchange. Resistance may expose organisms to ecological risk. Birds often put on fat in anticipation of acute food shortage, but the extra weight makes them less manoeuvrable and hence more vulnerable to predator attack. Resistance to chemical pollutants such as insecticides and heavy metals often seems to be associated with reduced fitness in optimal conditions, but the reasons are unknown.

The final topic discussed is the role of physical stress in setting the limits to species distributions, and its importance for conservation policy. There is no shortage of hypotheses for why species ranges do not extend further than they do, and empirical studies are few and inconclusive. Gene flow from central areas could prevent further adaptation at the margins of the species distribution, peripheral populations may lack the necessary genetic variability to adapt or there may be absolute physiological constraints on what can be tolerated. Some support for the latter point of view comes from work on the northern limits to the geographic ranges of 50 species of North American birds, where it was demonstrated that the resting metabolic rate at the coldest time of year (January) was about 2.5 times the basal metabolic rate, irrespective of variation in body size or ecology. Intriguing as this finding is, it is a mystery why such a constraint on metabolic rate should occur. Adaptation to stress may or may not be important for the conservation of endangered species. Conservation genetics is going through a period of some turmoil as it is appreciated that magic population numbers for avoiding inbreeding depression and preservation of the right kinds of genetic variability may be much less

important than population dynamics for predicting population persistence. It is not clear where physical stresses will fit in to the final picture, although deterioration in stress-resistance under benign conditions in captivity may well turn out to be an important issue.

I am left with the strong impression that most of the important work on evolutionary responses to stress remains to be done. We are largely in ignorance of the extent to which different stresses are encountered or the mechanisms by which they are combatted in nature. If only by exposing ignorance, Hoffmann and Parsons would have made a valuable contribution, but by collecting together disparate sources of information and pointing some ways forward, they have produced an excellent work of reference.

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*The Aquatic Ape: Fact or Fiction?* Edited by MACHTELD ROEDE, JAN WIND, JOHN PATRICK and VERNON REYNOLDS. Souvenir Press (E-A) Ltd, 43 Gt Russell St, London WC1B 3PA. 1991. 369 pages. Hardback £20. ISBN 0 285 63033 4.

The Aquatic Ape Theory (AAT), first conceived by Alister Hardy in 1929, proposes that an aquatic stage occurred in the early evolution of man from proto-ape ancestors. Hardy took the advice of colleagues that he would never become a Professor at Oxford or a Fellow of the Royal Society if he published such a controversial idea, and he suppressed it for 30 years until he had achieved these two aims, and then broached the theory cautiously, where it lay dormant for another 10 years until taken up enthusiastically by Elaine Morgan, who promoted it in two books – *The Descent of Woman* (1972) and *The Aquatic Ape* (1982). These caused considerable general interest but were not taken seriously by the palaeoanthropologists.

The book under review contains an updated version of the proceedings of the first international conference (in 1987) on the AAT hypothesis, and presents the views of a number of its proponents and critics. The result is a most useful and entertaining examination of current ideas on early hominid evolution. The argument centres on the evolutionary stage when the hominid line separated from the proto-apes during the late Pliocene in East Africa. Geological evidence suggests that the climate became drier and the forest habitat of the proto-apes receded leaving a woodland-savannah mosaic into which only the hominid line moved successfully. Its descendants were walking upright by nearly 4 million years ago, at the time of the *Australopithecus afarensis* hominids which included 'Lucy' and other fossil fragments from Hadar and Laetoli; but this advanced bipedalism, obviously better adapted to savannah than to arboreal life,