

EDITORIAL

Stress

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Workplace cartoons aside, stress is an underlying theme of an increasing fraction of this journal's output. Why is this? The comparative physiological approach offers an understanding of how the organism works; as this develops, a natural progression is to ask how these mechanisms help the organism to survive. I cannot think of a life form that exists in an entirely uniform, unvarying environment; therefore, survival depends on maintaining homeostasis under a range of external perturbations.

Some organisms are highly optimized for a tightly focused environmental range, but others must sacrifice peak optimization in order to provide adequate homeostasis over a much wider range of external challenges. In the great struggle for life, both strategies have their place, but in periods of rapid change, such as climate change, more fastidious organisms are likely to lose out to the generalists.

So what is stress? We define it here as any external perturbation to an organism's optimal homeostasis. On this basis, stress is a major part of this journal's core business: a large fraction of our articles report mechanisms of adaptation to thermal, osmotic, desiccation, salt, mechanical, starvation or other stressors. However, one of the things that is particularly attractive about the approach of *The Journal of Experimental Biology* is that stress can be seen in a multilevel context, from molecule to organism and beyond. The papers in this special issue try to survey this huge area, with some important reviews on organismal interactions with the environment (the stability of which is itself under stress through climate change); through to physiological responses at the tissue and organism level, and the cognate endocrine control mechanisms; and right down to the molecular level, with well-written articles on the endoplasmic reticulum and other stressors that act within cells.

Are there any general principles to be drawn from these articles? Well of course, the overt message is that as stress can be identified and studied at a range of scales, then a holistic multiscale approach is the best way to study stress! Another is that new data suggest commonality between stress effector pathways; that is, apparently independent stressors may act through common components of (for example) the innate immune response. Indeed, there is some evidence that one stress modality can condition, or acclimate, an organism's response to a second, different stressor.

Additionally, though, I am struck by an analogy with the physics of stress. When a material is deformed, the force per unit area is defined as stress, and the deformation produced is defined as strain. [And, of course, Young's modulus, E , is σ/ϵ (stress/strain) for the linear portion of the curve.]

Within a certain limit, there is a linear relationship (elastic), with Young's modulus defining the slope; when the stressor is removed,

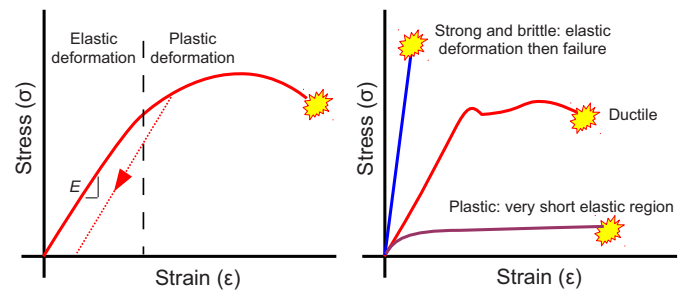


Fig. 1. Stress/strain curves. Left panel: a classical curve, showing elastic and plastic regions, with hysteresis (dashed line); one deformation enters the plastic range – the material does not regain the original shape when the stressor is removed. Right panel: stress–strain curves for a variety of different materials (and possibly organisms).

the material returns to its original size. In biology, this can be seen as the range of stress to which the organism shows resilience – it is a range that can be handled by inbuilt homeostatic mechanisms without any long-term change. Back in the physical world, once the elastic limit is reached, the material is changed forever (plastic deformation); that is, even if the stressor is removed, the material does not recover its original shape because it has been stretched too far. In biology, this plastic region corresponds to long-term changes that alter the organism's optimum range, e.g. changes in gene expression (indeed, the need for an increased genetic repertoire is one of the reasons suggested for the larger genome size of poikilotherms) or perhaps epigenetic marks – the topic of next year's special issue. Such adaptation does not have to be bad, because the organism is in better 'shape' for the new environment. However, if the material (or organism) is stressed beyond the plastic region, failure (or death) occurs. Different organisms could then be 'seen' as characteristic materials; for example, an extreme osmoregulator that survives an extended range of osmotic environments with minimal internal change, but that dies as soon as the mechanism is overstretched, would be brittle, or 'glassy', whereas a classical poikilotherm could be seen as highly plastic. Indeed, the adhesive Blu-Tack shows further biomimetic properties: if stressed suddenly, it fails (snaps), whereas if stressed gradually it acclimates (stretches). There are other properties reminiscent of biology: for example, a ductile material exposed to regular cycling in the plastic zone undergoes work hardening, making it stronger, but more brittle.

Armed with this outrageous analogy, of course, one can have lots of fun. What, for example, is the Young's modulus for an osmoconformer? Or, to return to the workplace, are plastic employees good because they are easily moulded? What is the Young's modulus for a resilient manager? And so on.

While it must be a fundamental law that any analogy can be stretched (stressed?) too far, it is entertaining to explore it in the context of the many distinct stresses and scales described in this special issue.

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