elements of modern-human behaviour existed by then.

We can now add Walter and colleagues' discoveries[4] to this picture. Middle Palaeolithic people might have spread from Africa along the shorelines of Arabia and into southern Asia during, or soon after, the last interglacial. Continuing along the narrow shorelines, to which they were already adapted, they could have progressed all the way to Indonesia at times of low sea level (Fig. 2). Movement along the coasts meant that they could have been spared the degree of habitat disruption faced by inland populations during the rapid climatic fluctuations of the Late Pleistocene. Coastal migration might also explain why they did not immediately replace archaic peoples living inland, such as those known from Ngandong in Indonesia[1]. At what stage the coastal migrants first ventured out to sea is unknown, but from the Australian evidence it seems that it must have been before 60,000 years ago. Such behaviour may have developed through the need to ford rivers or extend coastal foraging areas.

Archaeologists might now concentrate profitably on exposed fossil beaches in regions such as Arabia and India. Such sites may well contain Middle Palaeolithic artefacts that further document the spread of modern humans and their adaptations to a coastal environment. Southern Asia must have formed an important secondary centre for dispersals of modern humans — there, too, it may have been the coasts that provided the first and fastest routes for migration, before movement inland up river valleys. ■

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Fluid dynamics

Turbulence without inertia

Ronald G. Larson

In the 1880s Osborne Reynolds[1] established that fluid inertia (that is, momentum) drives the irregular patterns observed in water flowing rapidly from a pipe, plumes emerging from a smokestack, eddies in the wake of a bulky object, and many other everyday phenomena. Known as 'turbulence', these patterns occur at high values of the Reynolds number, the dimensionless ratio of inertial to viscous force. Over the years turbulence has become better characterized, and we now know it to be accompanied not only by an increase in drag, but also by certain characteristic spatial or temporal velocity fluctuations.

On page 53 of this issue, Groisman and Steinberg[2] show that both an increased resistance to flow and other features of turbulence can occur in fluids with hardly any inertial forces, if the role of inertia is instead played by elasticity, a force present in solutions of long-chain polymers. The flow studied by Groisman and Steinberg is simple: a polymer fluid confined between two parallel disks is sheared by rotation of one of the disks about their common axis. At increased flow rates (but rates still too low to generate much inertia) the flow acquires turbulent characteristics.

Irregular flow patterns have long been seen in polymeric and other elastic fluids, and have even occasionally been dubbed 'elastic turbulence'[3]. One such flow is that of a polymer melt emerging from the end of a capillary tube. Above a critical flow rate, the polymer jet becomes irregularly distorted (Fig. 1). Distortions in this and other polymer flows can mar products made from polymers, such as films and extruded parts. If the polymer is made more viscous by increasing the length of its polymer molecules, the minimum flow rate producing such irregular flow decreases. This is surprising because an increase in viscocity has the opposite effect on inertially driven turbulence. In fact, for fluids containing long polymer molecules, the Reynolds number at the onset of the instabilities can be minuscule, as low as 10^{-15}. Turbulent flow of water in a pipe, by contrast, has a much higher Reynolds number, around 10^5.

It is clear, then, that inertia has nothing whatsoever to do with this kind of polymeric 'turbulence'. For viscous polymers, instabilities and irregular flows set in at a critical value of the so-called Weissenberg number, the ratio of elastic to viscous forces in the fluid, which for polymeric fluids plays the role of the Reynolds number in creating the nonlinearities that lead to unstable flow. Because elastic forces increase with polymer length more rapidly than do viscous forces, fluids containing long polymers are especially prone to such phenomena, despite having viscosities that can be hundreds to millions of times higher than water. Until the work of Groisman and Steinberg[2], quantitative measurements of the length and time scales of elastic turbulence had been lacking.

Still lacking, even now, is a proper understanding of how the length and time scales of elastic turbulence are produced by the underlying elastic forces. In other words, what is missing is an elastic counterpart of the 'cascade' picture of inertial turbulence. In this, the largest eddies in the flow feed their energy into smaller eddies, which in turn drive even smaller eddies, and so on — until the energy stored in the smallest eddies is finally dissipated as heat. The resulting hierarchy of interacting eddies of various sizes is known as well-developed or hard turbulence. In Kolmogorov's 'bucket brigade' description of hard turbulence, a power-law distribution in length scales is produced by this handing off of energy from larger to smaller eddies[4].

For 'elastic turbulence', the basic mechanisms of instability in the base flow have only recently been discovered. These mechanisms involve 'normal stresses'. Normal stresses are produced by the stretching of polymer molecules in a flow, leading to an elastic force like that in a stretched rubber band. If the streamlines are circular, the polymer 'rubber bands' press inward from all directions, generating a radial pressure gradient. This

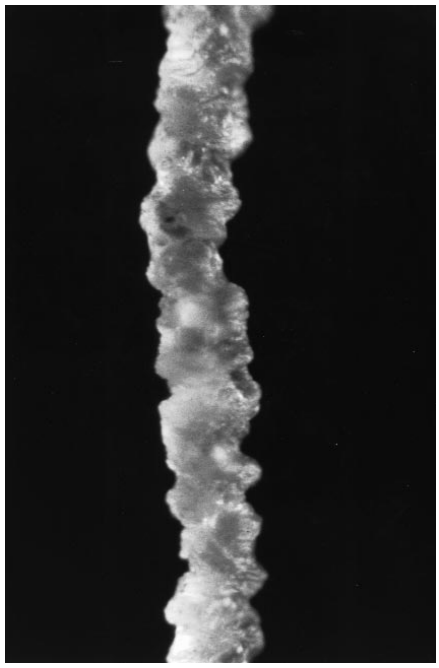

Figure 1 **Is this jet turbulent? Turbulent flow is normally associated with a high Reynolds number. A polymer melt of high viscosity (5,000 pascal seconds) and low Reynolds number is greatly distorted after emerging from a capillary tube[12]. Groisman and Steinberg[2] now confirm that the defining features of turbulence can appear in the flow of a polymer solution with high elasticity but low Reynolds number.**

pressure, or 'hoop stress', leads to the famous 'rod climbing' phenomenon described by Weissenberg[5] in 1947, and observed by every cook who has been annoyed by the climbing of cake batter (which contains natural polymers) up the shaft of an egg beater.

Despite being non-inertial, there is a strong analogy between such elastic forces and inertial instabilities involving curved streamlines, such as the flow in the gap between an inner rotating and a concentric outer stationary cylinder (circular Couette flow). In either the parallel-disk flow studied by Groisman and Steinberg, or the circular Couette flow, inertial forces try to fling fluid outwards, producing a radial pressure gradient, whereas elastic forces squeeze fluid inwards, tending to drive fluid up the rotating inner cylinder. This is what generates rod climbing. Whether the stresses are inertial or elastic (directed outward or inward), there is a radial pressure gradient that, if large enough, can drive an instability, leading to a more complex flow. For non-elastic fluids, the precise mechanism for the initial instability in inertial circular Couette flow was worked out by Taylor[6] in 1923, whereas analogous work for purely elastic instabilities[7–9] dates only from around 1985–95.

Beyond these initial instabilities, the first steps are being taken to work out the cascades of instabilities in elastic flows[10]. The work of Groisman and Steinberg leaps over these cascades, deliberately inciting highly unstable flow by having a large gap between the disks, relative to the disk diameter, so that the stabilizing influence of the viscous drag is minimized. The result is a flow with the power-law structures characteristic of hard, well-developed turbulence, but with negligible inertia.

Analogous 'hard elastic turbulence' should now be sought in other viscoelastic flows, such as Taylor–Couette, Taylor–Dean (curved pipe) flows, or the jet flow from a capillary. Or, it could be sought in structured fluids such as liquid crystals, where an ill-characterized irregular flow, sometimes called 'director turbulence', is observed[11]. Equally important would be a detailed experimental characterization and theoretical explanation of the transition from the first instability in the base flow to the highly irregular patterns of elastic turbulence. In addition, it would be interesting to see how other well-known features of ordinary inertial turbulence, such as efficient fluid mixing, are reproduced in elastic turbulence, and whether these can be put to practical use. ■

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Apoptosis

Gone but not forgotten

Douglas R. Green and Helen M. Beere

Multicellular animals are confronted daily with death and its consequences, at least at the cellular level. Cells die from wear and tear, as a part of differentiation and selection, and through mechanisms that provide for normal cellular turnover. Cells usually die by apoptosis, a death process that is controlled by intrinsic cellular mechanisms. But, in cases of severe injury, cells may instead undergo necrosis (a 'passive' death resulting in cellular lysis). Under both circumstances, the dead cells are rapidly cleared from the body, but each leaves imprints of its passing that can have long-term consequences. Dead cells do tell tales, and one way in which they do so is described by Fadok and colleagues[1] on page 85 of this issue.

Apoptotic cell death was originally distinguished from necrosis on the basis of morphological differences and of the propensity for necrotic, but not apoptotic, cells to induce an inflammatory response. Although it is still unclear how necrotic cells trigger inflammation, this ability may be critical in linking tissue damage (and the consequent probable entry of harmful pathogens) to the generation of an immune response. In contrast, apoptosis is often described as a 'silent death'. The earthly remains of cells resulting from an apoptotic death are buried (or rather, 'eaten') by phagocytic cells, effectively eliminating all physical evidence of death. However, the physiological ghosts of this form of death persist in the form of altered behaviour of the phagocytes.

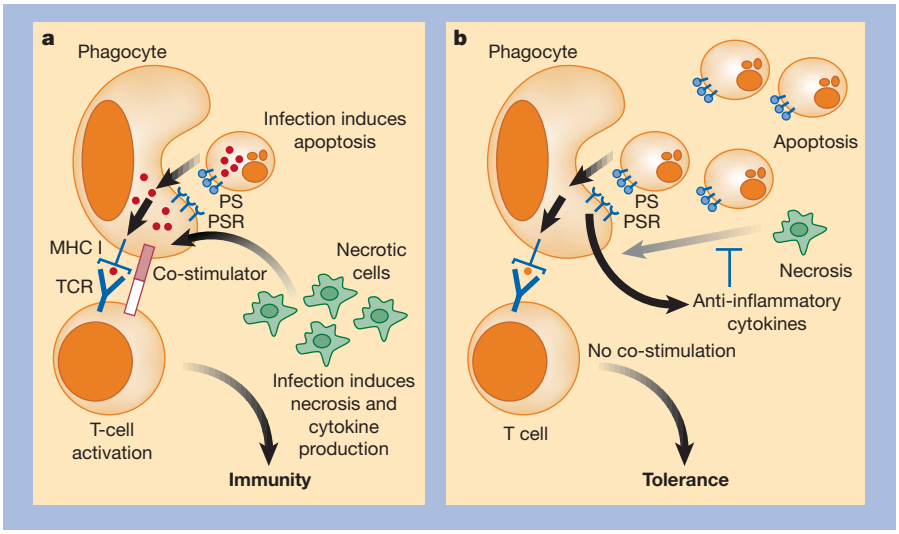

Figure 1 **Phagocytes (including dendritic cells and macrophages) can engulf and degrade both apoptotic and necrotic cells. Apoptotic cells are taken up by means of their interaction with the phosphatidylserine (PS) receptor (PSR) on the phagocyte (as shown by Fadok *et al.*[1]), as well as via other receptors (not shown). The balance between apoptosis and necrosis determines the biological response of the phagocyte.** a, **Under conditions of, for example, viral infection, necrosis and the release of pro-inflammatory cytokines are observed. Ingestion of the infected (apoptotic) cells by dendritic cells stimulates the presentation of viral antigenic peptides (red circles) by the class I major histocompatibility complex (MHC I) molecules. Necrotic cells are also ingested and, together with the pro-inflammatory cytokines, this process induces the expression of co-stimulatory molecules on the surface of the dendritic cell. Display of both of these markers stimulates T-cell activation and immunity. TCR, T-cell receptor.** b, **Under conditions of normal tissue turnover, apoptosis predominates. Ingestion of apoptotic cells by macrophages stimulates the release of anti-inflammatory cytokines and suppression of pro-inflammatory cytokines. Dendritic cells display peptides (small orange circle) from the engulfed apoptotic cell; however, expression of co-stimulatory molecules is not induced and T cells become tolerant to the displayed (self) peptide.**