The right sort of roughness

Peter Carpenter

Skin-friction drag affects any surface moving through a fluid. But a new theory of near-wall turbulence has led to a strange new drag-reduction technique: applying a random arrangement of bumps.

Every surfer believes that polished boards create less drag. This view has always been somewhat at odds with the natural world, where smooth surfaces are rare. It was further undermined with the discovery by the US space agency NASA¹, and by scientists studying the hydrodynamic effects of shark scales², that surfaces with riblets — minute streamwise grooves — experience lower drag than smooth ones. The 'smooth is best' precept has now received a further setback. On page 753 of this issue³, Sirovich and Karlsson show that surfaces covered with special randomized patterns of small v-shaped protuberances produce lower drag than smooth ones. But regular patterns of the same protuberances lead to a rise in drag, so the surfers are not completely wrong!

The type of drag in question is skin-friction drag, which is caused by the action of viscous shear forces on the surface. It is much greater when the flow is turbulent, as in most engineering applications. Drag reduction has been an obsession of practical aerodynamicists for most of the twentieth century; equally, achieving a theoretical understanding of turbulence has remained the elusive goal of many of the finest minds during the same period. Hitherto, the discovery of new drag-reduction methods has done far more to advance the theories of turbulence than vice versa. In this respect Sirovich and Karlsson are a rare exception, because their drag-reduction method is the fruit of their efforts to develop a theoretical understanding of the turbulent air flow in a two-dimensional channel.

The turbulent flow near a channel wall is bewilderingly complex. This complexity is inherent in the flow physics, and stems, fundamentally, from Newton's laws of motion. Turbulence is found when the effects of viscosity are weak — it is an intriguing paradox of fluid mechanics that the weaker the direct action of viscosity the more complex its effects. Most authorities now agree that deterministic structures (usually termed coherent structures), which are predictable in their general form, are created in turbulent flow. But these coherent structures have a broad, random distribution in time and space, and they are characterized by a very wide range of length and timescales.

It is generally believed that the crucial dynamics occur in a thin, highly sheared,

region, extending up to 2 mm from the wall in the channel-flow experiment of Sirovich and Karlsson. A veritable zoo of coherent structures has been identified by successive experimentalists, and a common view of the dynamics has emerged (Fig.1). Low-speed streaks appear near the wall⁴, caused by streamwise vortices. As these vortices develop they lift up away from the surface ('ejection') and finally 'burst'. Bursting generates intense turbulence and appears to initiate the formation of new vortex structures.

The key questions are, what causes ejection and bursting, and how does bursting create new vortices? There are many different theoretical models. For example, Smith *et al.*⁵ believe that the bursts occur when a vortex approaches the wall, causing strong local flow deceleration and subsequent flow separation (eruption) from the wall, thereby triggering the birth of a new vortex. On the other hand, Sirovich and his co-workers believe that, although the vortices are the primary structures, there are also much weaker oblique plane waves propagating downstream, and that bursting and rebirth come about through an interaction between a pair of waves and the vortices. In previous computer simulations they found that artificially randomizing the phases of the vortices and oblique waves led to less bursting and consequently to a reduction in drag.

Figure 1 Simplified side view of near-wall turbulence. Most of the shear stress (or drag) is created where vortices 'burst' away from the wall. An unproved idea is that bursting is caused by an interaction between the vortices and pairs of oblique plane waves. It has led to an effective new method of drag reduction³: randomized bumps that break up the phase coherence of the vortices.

The authors attempted to mimic this effect in the experiments by randomizing the arrangement of the v-shaped protuberances. In this way up to 12 per cent drag reduction was obtained, accompanied by reduced bursting frequency, in line with the numerical experiments. But regular arrays of the same protuberances led to a substantial drag rise.

Oddly, the maximum drag reduction achieved with conventional riblets is also about 12 per cent. But riblets are thought to make the vortices more ordered — it would be difficult to explain their effect in terms of phase randomization. Equally, it is difficult to explain the effects of phase randomization in terms of the other theoretical models. The secrets of turbulence appear to be as elusive as ever.

The near-wall turbulence for aircraft wings is quite similar to channel flow. So, would this new technique be practical for aircraft? For a large subsonic airliner in cruising flight, the near-wall region is less than 200 μm thick, owing to the much greater flow speed. Accordingly, this is roughly the required span of an individual protuberance. This may seem very small, but it is about ten times the spacing of the riblets that have been successfully flight-tested.

However, skin friction only contributes 30 per cent of the total drag for a typical subsonic airliner. (The other two main sources of drag are lift generation and shock waves.) So the randomized protuberances could lead to an overall drag reduction of about 3 per cent. In terms of the total amount of fuel used by the world's airlines in one year, this would still constitute a very large saving, but it only represents about 0.3 per cent of the running costs of an individual aircraft. To be acceptable to an airline, a drag-reduction method must not increase the aircraft's weight (as this causes a proportionate rise in drag), increase the capital cost appreciably (as this represents a much larger proportion of the

running costs than fuel), or interfere with maintenance, either compromising safety or increasing costs.

For these reasons, riblets have not yet been used for commercial aircraft; and the new method would probably be no more attractive. However, it may have advantages for other engineering applications, for example high-speed sailing yachts, such as America's Cup competitors, for which skin friction contributes more than half the total drag. □

Apoptosis

CED-4 is a stranger no more

Michael O. Hengartner

The nematode worm *Caenorhabditis elegans* has been used with great success to identify the basic components of the machinery underlying apoptosis (programmed cell death)¹. Indeed, of the three key cell death genes that have been identified in *C. elegans*, two — *ced-3* and *ced-9* — have mammalian homologues that also function in apoptosis. But the sequence of the third gene, *ced-4*, revealed no obvious mammalian homologue, and precious little in terms of possible mechanism of action. A flurry of activity has changed that. A paper by Zou *et al.*, published earlier this month in *Cell*², provides a homologue. And work by Chinnaiyan *et al.* (page 728 of this issue³) and by Seshagiri and Miller in *Current Biology*⁴ lays down some choreography for the part that CED-4 protein plays in the molecular dance of death.

Over the years, genetic screens in *C. elegans* have led to the identification of about a dozen cell death (*ced*) genes that are responsible for one aspect or another of the apoptotic process. Three of these genes stand out. Two, *ced-3* and *ced-9*, antagonizes the proapoptotic activities of *ced-3* and *ced-4*, and thereby protects cells that should survive from any accidental activation of the death programme.

Real stardom for *ced-9* and *ced-3* only came with their cloning, when it became clear that they encode components of a universal and highly conserved death machinery: CED-9 protein turned out to be a member of the Bcl-2 family of cell death regulators, whereas CED-3 was homologous to a family of proapoptotic cysteine proteases, known as the caspases. Indeed, identification of CED-3 as a caspase homologue was the first evidence of this family's involvement in apoptosis. In both worm and human, CED-9/Bcl-2 act upstream of the 'execution caspases' such as CED-3 and caspase-3, somehow preventing their proteolytic processing from zymogens into fully active killer enzymes. But how Bcl-2 *et al.* perform this feat remains very much up in the air.

The *ced-4* story was initially much less glamorous. Because the gene encoded a pre-

Peter Carpenter is in the Department of Engineering, University of Warwick, Coventry CV4 7AL, UK.

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4. Kline, S. J., Reynolds, W. C., Schraub, F. A. & Runstadler, P. W. *J. Fluid Mech.* **95**, 741–773 (1967).
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viously unknown protein without any mammalian homologue, its cloning did not provide much help in explaining how it might promote cell death. Genetically, *ced-4* had been placed between *ced-9* and *ced-3* in the pathway leading to cell death⁵, suggesting that it might act as an adaptor, linking the upstream regulator CED-9 to the downstream death effector CED-3.

Strong experimental support for this view came from four papers published early this year, which collectively showed that CED-4 can interact directly and simultaneously with both CED-9 and CED-3 (reviewed in refs 6, 7); the interaction must be quite stable, as CED-4 and CED-9 colocalize when co-expressed. These observations point to a model in which CED-9 prevents cell death by directly binding to a CED-4/CED-3 complex, presumably keeping it in

an inactive conformation. Upon reception of a death-inducing stimulus, the complex (already dubbed the 'apoptosome' by pundits) might dissociate, freeing CED-4/CED-3 to get to work (Fig. 1a).

But being a linker between CED-9 and CED-3 cannot be the whole story: the fact that CED-4 is essential for cell death in *C. elegans* indicates that it must have a specific proapoptotic activity. The two papers by Chinnaiyan *et al.*³ and Seshagiri and Miller⁴ describe just such an activity: both groups found that processing of proCED-3 into the active protease was dramatically increased in cells that also expressed CED-4. This stimulation was inhibited by mutations known to knock out the death-promoting activity of CED-4, or by co-expression of wild-type (but not mutant) CED-9. These observations fit very nicely with the apoptosome theory, and also explain why CED-3-mediated killing is so inefficient in the absence of CED-4.

How does CED-4 promote CED-3 activation? One possibility is that CED-4 is itself a protease that can cleave proCED-3. Arguing against this hypothesis, Chinnaiyan *et al.*³ showed that processing of an enzymatically inactive CED-3 mutant is not stimulated by CED-4. Thus, the obvious alternative is that CED-4 promotes CED-3 autoprocessing. A hint of how it might do so comes from the observation that CED-4 contains a predicted nucleotide binding site, which seems to be functional because point mutations in the site abolish the proapoptotic activity of CED-4 in a number of experimental systems^{3,4,8}.

Furthermore, Chinnaiyan *et al.*³ have now shown that purified wild-type CED-4

Figure 1 Caspases are cysteine proteases that promote apoptosis in mammals and have homologues in *C. elegans*. **a** and **b** depict highly simplified schemes of how they might be activated in the two systems. **a**, In *C. elegans* the inactive CED-9/4/3 complex is bound to cell membranes by CED-9's hydrophobic tail. On reception of a death-inducing stimulus, something happens (here shown as the physical separation of CED-4/3 from CED-9) which allows processing and activation of the 'execution caspase' CED-3. **b**, In mammals, the Bcl-2 family might perform double duty, preventing cytochrome *c* from leaving mitochondria and also possibly binding to Apaf-1 (one of the so-called apoptosis protease-activating factors, of which cytochrome *c* is Apaf-2). The death-inducing stimulus would allow formation of the full Apaf complex, possibly including Apaf-3, leading to caspase-3 processing and activation. Question marks point to aspects that are even more egregiously speculative than the rest of the model.