Turbulence and the tube

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SIR Horace Lamb, the famed British hydrodynamicist, is said to have remarked, in 1932: ". . . when I die and go to Heaven, there are two matters on which I hope for enlightenment. One is quantum electrodynamics, and the other is the turbulent motion of fluids. And about the former I am really optimistic." (Ref. 1.) One no longer needs to go to Heaven to seek enlightenment about quantum electrodynamics but, on Earth, turbulence still defies satisfactory description. However, new analytical, experimental and computational tools are being forged today in powerful combination to revitalize efforts at understanding the phenomenon. The report by She, Jackson and Orszag² on page 226 of this issue illustrates the type of information that one can now obtain from careful computational

The primary traits of turbulence are that it is vortical (see box) and threedimensional, and that it manifests itself on many scales. In the familiar example of the Earth's atmosphere, the scale-range (conveniently characterized by the flow Revnolds number) extends from several kilometres to a fraction of a millimetre. The generation and interaction of turbulence on these scales is at the heart of the overall problem, but a full quantitative measurement of small-scale details is at present impossible. Most studies consist of ingenious arguments based on features of the governing differential equations³, temporal measurements from one or at most a few point-probes⁴ and qualitative visualizations⁵. On the other hand, recent computer solutions of the equations of motion have shed light on threedimensional structures, at least on their kinematic aspects. Although the Reynolds numbers of the most ambitious calculations are still moderate and less than those found in practice - and there remain some finer questions concerning initial conditions, external forcing and the like — computational studies capture

The vorticity, ω , of a fluid motion is related to the velocity field, u, by $\omega = \nabla \times \mathbf{u}$

Despite the apparent simplicity of this mathematical description, the physical meaning of vorticity is less easily described. In essence, non-zero vorticity corresponds to the local rotation of the fluid; more specifically, it means that a volume element of the fluid is changing its orientation in space. Consider the two examples here in which a volume element follows a circular path.

important features of turbulence and their role is firmly established.

She et al. 2 deal with small-scale structure. It has been known for some time that the behaviour on small scales is intermittent and does not follow gaussian statistics. It is believed that large scales do not directly affect small-scale features at any rate, to a good first approximation for the velocity field — but that small

tube by subjecting it to extension in one direction and compression in the other two; to produce a sheet, one needs extensions in two directions and compression in the third. Kerr^{6,7} pointed out that, on the basis of his calculations, most often two of the principal rates of strain are positive and the third negative, so that the formation of sheet-like structures would be favoured. But, his results do permit a less frequent formation of tubes. Further, as She et al. point out, the mere existence of conditions favouring sheet-like structures is not sufficient for expecting them always to occur, because the strain field is not



The small-scale structure of the scalar in a round jet of water at a nozzle Reynolds number of 4,000. From the concentration field of a dye obtained by laser-induced fluorescence (for details, see ref. 9), one constructs a two-dimensional graph of the square of the gradient of the concentration. This quantity represents the small-scale features of the scalar. The resolution of measurement is between one and two Kolmogorov scales. Much finer scales of the scalar have not been resolved.

scales do directly influence the life, size | constant for long enough periods of time and strength of large scales. If small scales can be modelled satisfactorily, computer simulation of high-Reynolds-number flows of practical interest becomes a viable prospect.

The first contribution of She et al. is the demonstration that the high-vorticitybearing elements are tube-like rather than sheet-like or blob-like. They comment that the structures at lower intensities are sheet-like. A blob can be deformed into a

In both cases there is rotary motion of

the fluid, but only in the left-hand

example is there a change in the spatial

orientation of the fluid element, and

thus non-zero vorticity. (Note that,

more accurately, the element on the

right-hand side must also be deformed

to maintain zero vorticity.)

over spatial extents commensurate with the observed structures. On balance, it appears that intense tube-like structures coexist with less intense sheet-like structures. Unfortunately, it is not yet clear how one might use this fact in building working models. Nor is the dynamical role of tubes clear. Betchov3 showed that vorticity production requires a preponderance of sheet-like structures. Do we conclude that a large part of vorticity production is associated with low-intensity structures, perhaps because they are more spacefilling?

to explain the relatively long life of the structures.

It would be instructive to reproduce

The second main point of the report² is the finding that vorticity and the velocity are roughly aligned, such that the effective nonlinearity is quite small. Basically, from the relation $\mathbf{u} \cdot \nabla \mathbf{u} = \boldsymbol{\omega} \times \mathbf{u} + \nabla (u^2/2)$, it follows that the effective nonlinearity would diminish if **u** and **ω** were aligned with respect to each other. This conclusion on the depletion of nonlinearity is intriguing and is invoked by the authors

these findings in a laboratory experiment; unfortunately, it will not be possible in the near future. One way of tying together the experimental and computational approaches is to explore areas where their strengths overlap. For example, it is now possible to capture experimentally the three-dimensional field of a passive scalar (such as a small amount of dye or heat) mixed by turbulence. The passive scalar also tends to form intermittent structures by, one might think, the stretching due to the small scale. As She et al. demonstrate, this activity is intermittent; so is the small-scale scalar structure. There is experimental evidence to show that the small scales in velocity and scalar fields are poorly correlated spatially. Two uncorrelated intermittent activities may have little to do with each other, so that, even at high Reynolds numbers, small-scale scalar structure may be significantly deformed by the large-scale motion — especially in the case of flows with mean shear. If this is true, it may explain a number of nonuniversal features observed of small-scale scalar fields. Further, because the largescale motion is well correlated over sizeable spatial extent as well as over long

enough times, the tendency for smallscale scalar might be to form sheet-like structures.

Some support for this view comes from the figure, where the small-scale structure of the scalar is shown in a section of a turbulent jet. From a cursory study of several parallel sections of the jet, it has been concluded that the structure is indeed sheet-like; this appears to be the case with other inhomogeneous shear flows such as wakes. One can further speculate, on the basis of some experimental results⁸, that the conclusion might also hold for homogeneous shear flows. \Box

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GENE EXPRESSION —

Chromatin contract to silence

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In female mammals, the chromatin in one of the two X chromosomes remains condensed in a mitotic-like conformation throughout interphase and is largely nontranscribed. This type of compact, inactive heterochromatin is also associated with position-effect variegation in the fruit fly, Drosophila melanogaster 1,2, in which it is responsible for an inherited form of gene inactivation. On page 219 of this issue, Reuter et al.3 report the use of a combination of genetics and molecular biology to identify a protein in *Drosophila* that seems to be intimately involved with this type of heterochromatin formation. This protein is the product of the

Suvar(3)7 gene at chromosome position 87E. Reducing the number of copies of this gene from two to one suppresses the extent of position-effect variegation (that is, it decreases the amount of heterochromatin), whereas increasing the number of copies to three or more enhances variegation progressively in response to gene dose. Genes having this property could well encode structural components of heterochromatin. Indeed, a second gene that also increases the extent of position-effect variegation in proportion to gene dose, Suvar(2)5 (also known as Su(var)205), is known to encode a *Drosophila* protein that is confined to heterochromatin, called HP-1 (ref. 4; S. Elgin, personal communication).

What makes the proteins encoded by Suvar(3)7 and Suvar(2)5 particularly interesting is the unusual nature of the gene regulatory processes that they affect. When a chromosome rearrangement joins the middle of a region of heterochromatin to a region of the chromosome that is normally packaged into standard chromatin, a zone of gene inactivation spreads progressively from the chromosome breakpoint to cover one or more adjacent genes. Most strikingly, although the extent of this heterochromatin spreading is different in different cells of the embryo, the inactivated zone is stably inherited by the progeny of each of these cells. Thus, in position-effect variegation, as in mammalian X-chromosome inactivation, genes are turned off in a manner that seems to be inherited from parental chromosome to daughter chromosomes.

The inheritance of the repressed gene state cannot be attributed to feedback loops of diffusible gene regulatory proteins, because the second copy of each affected gene in a diploid cell remains normally active. The models proposed to explain this form of gene regulation instead suggest that heterochromatin forms by a highly cooperative, 'crystallization' event, which is nucleated from special chromosomal sites and then spreads along the chromosome to take in hundreds of kilobases of DNA⁵. It seems

that the ability to create new regions of heterochromatin is lost early in embryogenesis and that, thereafter, the established pattern of heterochromatin is inherited. One might propose that some proteins in the heterochromatin remain bound to the DNA when it is replicated, being inherited by the two daughter DNA helices along with the histone octamers from the parental nucleosomes⁶. These proteins would then nucleate the cooperative reassembly of heterochromatin on each daughter chromosome.

The orderly development of multicellular organisms requires that progeny cells 'remember' the patterns of gene expression of their parents. To what extent is this cell memory dependent on chromatinbound proteins that are directly inherited during DNA replication, as postulated for the maintenance of heterochromatin?

Models of this type are generally not invoked to explain the regulation of individual genes. But a popular model for the regulation of the bithorax complex of homoeotic genes invokes the progressive inactivation of large chromosomal regions⁷, and there are reasons to believe that genes in the *Polycomb* class might act to keep genes turned off in the bithorax complex by mechanisms analogous to those in heterochromatin formation³.

Help in analysing the effect of chromatin structure on the expression of genes should also come from recent work on the yeast Saccharomyces cerevisiae. Yeast chromosome III has three widely separated genetic loci that code for mating-type information. Only one of the loci, MAT, is expressed; it determines the mating type of the cell, a or α haploid, or a/α diploid. The other two loci, *HML* and *HMR*, are transcriptionally silent, in spite of the fact that they contain the same DNA sequences (a or α) as the expressed locus, MAT. If the mating type genes HML or HMR are replaced by other yeast genes, these will also be kept transcriptionally repressed. The repression is through cisacting DNA sequences, termed silencers8, which are separated by a kilobase or more from the promoters of the genes whose expression is affected. The silencers contain autonomously replicating sequence elements that can function as origins of replication; DNA replication has in fact been implicated in the establishment of silencing9.

The silencers also have binding sites for two abundant DNA-binding proteins, RAP1 (ref. 10) and ABF1 (ref. 11). These two proteins bind upstream of many important housekeeping genes, and they are essential for the viability of the yeast cell. Genetic analysis has shown that four additional proteins, SIR1-4, are necessary for silencing¹². The genes encoding the four proteins have been cloned, sequenced and mutated, but their functions are still unclear. Interestingly,