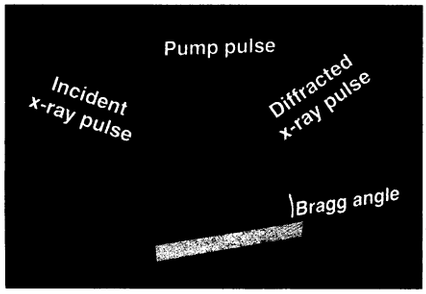


**Now you see it...** An intense, ultrashort optical pulse leads to melting, which is monitored through time-resolved x-ray diffraction.

photochemistry, where the valence electrons are involved in chemical change and their features in the visible spectrum can often be associated reliably with the molecular geometry, especially for small, well-studied molecules. But the connection between valence electronic spectral properties and collective structure is not easily made, and it is therefore often difficult to infer the evolution of condensed matter structure from transient optical spectroscopic features. Also, light can often not penetrate the samples sufficiently. In fact, the melting transition, now monitored with x-rays, was observed earlier with visible light (*9*, *10*). Fast melting was indicated, but it was uncertain whether it was homogeneous because the light did not penetrate the sample.

The development of ultrafast x-ray probes promises new capabilities for monitoring time-dependent structural changes in complex systems including crystalline solids and biological molecules, and is particularly promising for probing collective changes in crystal structure. But its development also issues a challenge. Now that we can watch collective structural evolution, can we devise ways to initiate it in a synchronized, or phase-coherent, manner?

In femtochemistry, absorption of an ultrashort optical pulse launches all the photochemically active molecules along the reaction path at the same time. Only then can probing light pulses be used to record snapshots of all the excited molecules as they pass through a sequence of well-defined transient molecular structures. Ultrashort optical pulses can also launch coherent collective motion of ions or molecules in crystal lattices, in some cases along the paths that lead them part of the way toward—but generally not all the way into—the positions they occupy in new crystalline structures, that is, part of the way along "collective reaction coordinates" (*11*). There is some evidence that collective structural change can occur as a result (*12*). Methods permitting more extensive optical control over collective behavior have been demonstrated (*13*, *14*). Only if they can be extended substantially will we truly gain the ability to watch the transformation of condensed matter as it passes through a sequence of well-defined transient collective structures (*15*–*17*).

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NOTA BENE: DEVELOPMENT

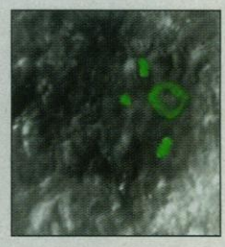
## Whirling Dervishes

Although paired organs such as the kidney and the lung are neatly arranged on each side of the body's midline, other tissues such as the heart and the liver prefer to be on either the left or the right. But how does a developing embryo ensure that, for example, the tube of mesodermal tissue that will eventually form the heart, curves to the left of the body's axis? Recent findings by Okada *et al.* (*1*) add to growing evidence that the clockwise rotation of cilia on embryonic nodal cells is the initial event that determines the asymmetric placement of certain organs.

Nodal cells, each bearing a single cilium, are clustered in a triangular pit (node) that first appears as the neural plate is laid down (at day 7.5 of mouse embryonic development). In work published last year (*2*), Okada and colleagues reported that in mice lacking either the KIF3A or KIF3B motor protein kinesins, the asymmetry of organ placement was random (*2*). These two proteins (together with KAP3) form a complex that transports structural components of the cilium along its hollow interior. Animals deficient in either KIF3A or KIF3B are devoid of cilia.

In their new work (*1*), the Japanese group decided to investigate why the organs of the *iv* and *inv* mutant mouse strains are in abnormal locations. In the *iv* strain, which carries a mutation in a gene encoding dynein (a component of ciliary architecture), the location of organs is randomly asymmetric, whereas in *inv* mice (which carry a mutation in the *inv* gene, whose function is unknown), organ placement is completely reversed. The investigators suspected that a defect in nodal cilia could be the culprit common to both abnormalities.

To test their hypothesis, they analyzed movement of nodal cilia with a video fluorescence microscope, following the trajectory of fluorescent beads chemically attached to the ends of the cilia (large green circle in figure). The movement of unattached fluorescent beads (green spots in figure) revealed how ciliary motion altered the flow of extraembryonic fluid in the node. The authors discovered that, unlike nodal cilia in normal mice that rotated clockwise (apparently in synchrony), those in *iv* mice were motionless. The normal clockwise rotation of nodal cilia moved the fluid to the left of the node but, in the absence of ciliary movement, there was no fluid flow. In *inv* mice, the cilia appeared to rotate normally, but a closer look revealed that they were moving asynchronously. This, together with an abnormality in the shape of the node, generated turbulence in the fluid flow.



Intriguingly, nodal fluid flow begins about the time that left-right determination of tissue location first becomes manifest; asymmetric expression of left- or right-determining genes such as *lefty-2* soon follows. The Japanese group speculated that the fluid might contain morphogens (molecules that dictate development of embryonic tissues) that would accumulate on the lefthand side of the node, driven by the clockwise rotation of the cilia. Accumulation of sufficient morphogen would induce expression of genes in ventral node cells, which would then direct expression of left- or right-determining genes in adjacent mesodermal cells (these are the cells that will eventually develop into organs).

Next, Okada and collaborators plan to identify the fluid morphogens and to alter left-right determination in mouse embryos by mechanically perturbing fluid flow.

—ORLA SMITH

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