hydraulic jump seen in the mound of white water just past the rock.

In simulations of the 1972 chinook, Durran found that the higher wind speeds, called supercritical flow, near the crest removed the wave and provided the first lee-side acceleration. He also found that an inversion was essential to the development of that supercritical flow. Only later did wave breaking play a role, he says. In contrast, Clark and Peltier have cited a case in which supercritical flow appeared without an inversion being present.

Although researchers continue to debate just what triggers a particular episode of severe downslope winds, there is growing agreement that the underlying driving mechanism once triggering occurs resembles hydraulic flow, an explanation that a few years ago seemed too simple for such a complex phenomenon. "The bora, chinook, and water flowing over a rock are fundamentally the same phenomenon," says Durran. Clark will not go quite that far, preferring to emphasize that waves within the atmosphere can never behave exactly like waves on the surface of water. That can lead to some differences from predictions of hydraulic jump theory, he says.

Researchers had tended to view the bora and chinook as involving different processes because these winds looked so different. The chinook, a warming wind, involves the whole troposphere flowing over the Rockies, while the bora involves a shallow pool of cold air spilling over the Dinaric Alps. But Ronald Smith of Yale University has developed a mathematical description of mountain wave behavior in terms of hydraulic theory that works equally well for both the bora and chinook. When adjusted to a common scale, says Smith, the two bear a strong resemblance, and the same hydraulic theory applies to both.

Parlaying new understanding of the basic mechanism of severe downslope winds into better forecasting will take much more work in computer simulations. Forecasters can already say when conditions will favor a chinook. The problem is predicting when it will be an exceptionally severe one, and that will probably require more details on upstream conditions that lead to the strongest winds. ■ RICHARD A. KERR

ADDITIONAL READING

T. L. Clark and W. R. Peltier, "Critical level reflection and the resonant growth of nonlinear mountain waves," *J. Atmos. Sci.* **41**, 3122 (1984).

D. R. Durran, "Another look at downslope windstorms," *ibid.*, submitted.

J. B. Klemp and D. R. Durran, "Numerical modelling of bora winds," *Meteorol. Atmos. Phys.*, in press.

R. B. Smith, "On severe downslope winds," *J. Atmos. Sci.* **42**, 2597 (1985); "Aerial observations of the Yugoslavian bora," *ibid.*, submitted.

Briefing:

The Tissue Specificity of The Drosophila P Element Is Explained

Introduction of new genes into the fruit fly (*Drosophila melanogaster*) can be readily achieved with the aid of a method that was devised a few years ago by Allan Spradling and Gerald Rubin of the Department of Embryology of the Carnegie Institution of Washington (which is in Baltimore). The method depends on the use of "P elements," segments of DNA that can, under certain conditions, move from place to place in the drosophila genome. Spradling and Rubin took advantage of the P element's ability to integrate into fruit fly DNA and adapted the P element as a vehicle for introducing new genes into that organism. They found that they could in effect cure genetic defects in fruit flies.

The hope was that the element could be developed as a gene transfer vehicle for mammalian cells, too. Attempts to do this proved futile, however, although Spradling and Rubin supplied P element clones to perhaps 100 investigators who wanted to try. Now, Rubin, who moved to the University of California at Berkeley about 2 years ago, and his Berkeley colleagues Frank Laski and Donald Rio have new information about the P element that not only explains its distinctive pattern of activity in the fruit fly but may also account for its failure to work in mammalian cells.*

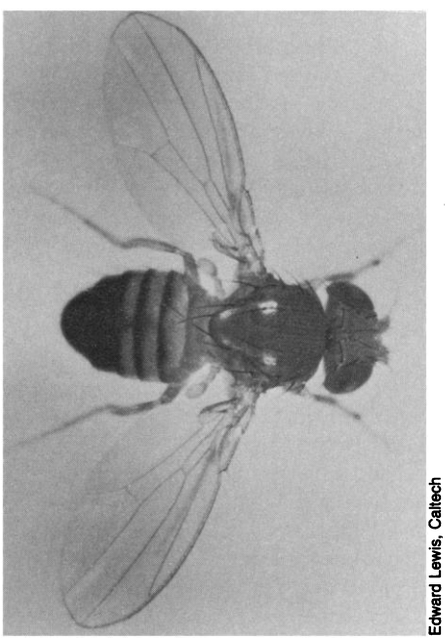
P element movement in drosophila occurs only in the germline cells, which give rise to the sperm and eggs, but not in the somatic tissues. The movement depends on the activity of a "transposase" enzyme that is encoded in the full-size P element.

The question then becomes how germ cells can make active transposase whereas somatic cells cannot. The two types of cells can transcribe the transposase gene into RNA, the Berkeley workers find, but they then process the RNA differently.

Earlier work had shown that the complete P element contains four regions with the potential to code for protein structure. According to Roger Karess who worked with Rubin at Carnegie, all four are needed to make the transposase. They are separated by short, noncoding segments (introns) that would have to be cut out of the original RNA transcript to produce the messenger RNA for the active transposase protein.

*F. A. Laski, D. C. Rio, G. M. Rubin, *Cell* **44**, 7 (1986); D. C. Rio, F. A. Laski, G. M. Rubin, *ibid.*, p. 21.

However, when Laski, Rio, and Rubin examined the mRNA from whole fruit fly embryos, what they found appeared inconsistent with Karess's observations. The intron between the third and fourth coding regions was not spliced out, and thus the fourth coding region would not be represented in the transposase protein. "There was only one logical way out of the contradiction," Rubin explains. "The messenger RNA we were looking at wasn't functional."



Edward Lewis, Caltech

The invaluable fruit fly

To test the hypothesis that the third intron must be removed to produce a functional transposase mRNA, Laski, Rio, and Rubin prepared a mutant P element that was altered so that the third intron could not be spliced out. As expected, this P element worked neither in the somatic nor in the germ cells. Conversely, when the Berkeley workers repeated the transfer with a P element from which the third intron had been precisely removed, this modified element worked in all fruit fly cells.

The Berkeley workers conclude that normally the P element is active in germ cells and inactive in somatic cells because only the germ cells have the ability to remove the third intron.

Rubin, Laski, and Rio do not yet know the basis for the different splicing patterns of transposase mRNA in germ and somatic cells. "There is nothing unusual about the structure of that third intron," Rubin notes. Nevertheless, the results raise the possibility that the P element from which the third intron is removed will work in mammalian cells just as it did in drosophila somatic cells. ■ JEAN L. MARX