whether it has the fine-grained texture of a lava or is a coarse agglomeration of particles. Pathfinder is a good deal less capable than a human field geologist, but color imaging from the lander has already suggested that Barnacle Bill is uniform down to the centimeter scale, as expected of an andesite.

If Barnacle Bill continues to look like an andesitic lava, it could lend support to a new picture of Mars's geologic past. Today, Mars has neither oceans nor any signs that plate tectonics is at work there. It appears to be a "one-plate planet," encased in a single, thick layer of cold, immobile rock, as our moon has been for billions of years. But geophysicist Norman Sleep of Stanford University proposed in 1994 that the great northern lowlands of Mars, which cover one-third of the planet and lie 3 kilometers below the ancient highlands of the southern hemisphere, are the martian equivalent of ocean basins. Sleep proposed that they formed 3 billion to 4 billion years ago by the same drifting of plates still operating on Earth.

Sleep's proposal has been controversial. "It's worth considering the concept of plate tectonics on an early Mars," says planetary physicist David Stevenson of the California Institute of Technology in Pasadena. But he adds that "it's hard to know how to test it or develop a convincing theoretical argument."

Andesitic lavas would certainly bolster the case for plate tectonics on Mars if they turned up all across the planet. But finding more even at the Pathfinder site won't be easy. Barnacle Bill was the only bona fide rock to be cleanly analyzed in the rover's first 18 days. Operational problems, apparently resolved now, caused repeated delays (see sidebar), but the rocks themselves are presenting challenges as well.

APXS analysis of another rock, called Yogi, at first suggested that it was more basaltic than Barnacle Bill, but a closer look at the rock face analyzed by APXS revealed what looked like a coating of dust and weathered minerals, says team member Ronald Greeley of Arizona State University in Tempe. Viking lander images had suggested that martian rocks would have such problem coatings (*Science*, 19 April 1996, p. 347). That leaves the makeup of the rock itself still uncertain, says Greeley. Although the APXS analysis of the third rock, Scooby Doo, had not been released at press time, team members are now describing it as more like a crust of solidified soil than a rock.

Both the lander and rover seem to have weeks and even months of productive work ahead, however, so team members remain upbeat about getting a look at a lot more rocks. "Things are never quite as simple as you might like them to be," says Greeley, "but that makes it interesting."

–Richard A. Kerr

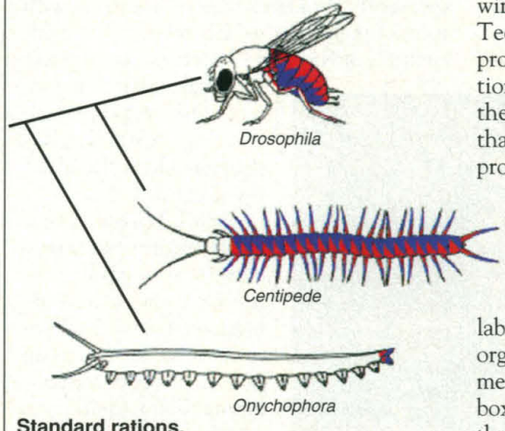
MEETING BRIEFS

A Developmental Biology Summit in the High Country

ALTA, UTAH—The ski hills surrounding this old silver-mining town provided an exhilarating setting for more than 1000 scientists who gathered here from 5 to 10 July for an unusual joint conference of the International Society of Developmental Biologists and the Society for Developmental Biology. A head-spinning assortment of topics from evolving gene families to fruit fly eyes abetted the high-altitude daze.

Segmentation's Origins

Biologists have long believed that the diverse body segments of most insects—head segments with antennae, for example, and thoracic segments with wings and legs—evolved from the many identical segments of more primitive arthropods that looked like today's centipedes and millipedes. In the 1980s, researchers thought they might have a simple explanation for the genetic changes responsible for this diversification of segments: a duplication and diversification of genes. But at the Utah meeting, Jennifer Grenier and colleagues in the lab of developmental biologist Sean Carroll at the University of Wisconsin, Madison, described new results challenging that explanation.



GRENIER *ET AL.*, *CURRENT BIOLOGY*

Standard rations.
Insects don't have more *Hox* genes than related groups; they just use them differently. (Red shows *Ubx* expression; blue is *abd-A*.)

The older explanation grew out of the discovery that the fruit fly genome carries eight consecutive "*homeobox*" (*Hox*) genes, named after the conserved DNA sequence they all contain. Because each *Hox* gene helps a particular segment acquire its unique identity during development, the find suggested that the insects' evolutionary ancestor had only a few *Hox* genes, and that insects acquired distinct structures on their segments as extra copies of these genes accidentally cropped up in insect DNA and then specialized.

If so, then other surviving descendants of this hypothetical ancestor would be expected to lack some of the fly's eight *Hox* genes. But Grenier and her colleagues now report that they have detected all eight genes in centipedes and even in onychophorans—worm-like creatures that are often described as "living fossils," the closest living relatives to the group that gave rise to the arthropods, including insects. (The work is also described in the 1 August issue of *Current Biology*.) Because ancestors of the two groups diverged from insect ancestors long before the insect body plan subdivided, says Grenier, the finding implies that "the gene duplications didn't happen during insect evolution. They were much more ancient."

Indeed, says geneticist and Nobel Prize winner Ed Lewis of the California Institute of Technology in Pasadena, one of the earliest proponents of idea that *Hox* gene duplication brought about insect segment diversity, the Grenier team's work "quite nicely" puts that theory to rest. As an alternative, Grenier proposes that segment diversity arose from changes in *Hox* gene activity.

To analyze the *Hox* genes of centipedes and onychophorans, Grenier, fellow Carroll lab members Theodore Garber and Robert Warren, and Australian collaborator Paul Whitington first purified the organisms' DNA, and then used the polymerase chain reaction to amplify their homeobox regions. The researchers then sequenced the regions and compared them with fruit fly sequences. They found that each fly *Hox* gene has a related or "orthologous" gene in the centipedes and onychophorans.

Having ruled out the simplest theory of insect segment diversification, Grenier and her co-workers went on to explore whether changes in gene regulation, either of the *Hox* genes themselves or of the genes the *Hox* genes control in turn, might explain it instead. The group found major differences in the ways embryonic fruit flies, centipedes, and onychophorans deploy orthologous genes. For example, the *Hox* genes *Ubx* and *abd-A* are expressed primarily in the abdominal segments of fruit fly larvae, but in the centipede embryo the two genes are active in all seg-

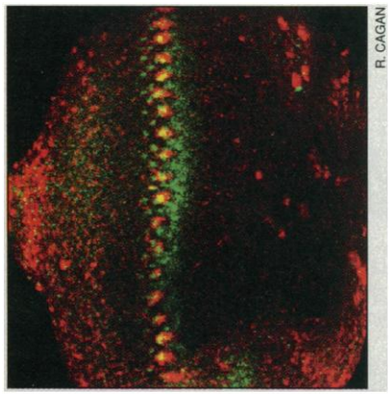
ments but the head, and in the onychophoran embryo they are turned on only in the hindmost segment.

To understand exactly how these organisms came to have different body plans, says Carroll, researchers will have to compare the complex regulatory regions of the *Hox* genes and the genes they regulate in many different species, reconstructing shifts in the timing and location of gene expression that may have altered different lineages' development. While this form of evolutionary tinkering is "more complicated" than simply duplicating existing genes, Grenier says, "it's also more exciting, because you can see the huge potential for generating diversity."

Arraying the Fly Eye

A fly's compound eye is one of the marvels of development. Because each of its hundreds of independent photoreceptor units, called ommatidia, sees in a slightly different direction, they have to be assembled in an absolutely uniform hexagonal array in order for the fly's brain to piece together a coherent, wide-angle view of the world. But just how this design is imposed on the eye imaginal discs, the clumps of undifferentiated cells in the fly larva that give rise to the ommatidia, has long puzzled biologists. In Utah, however, developmental researcher Ross Cagan reported studies of the fruit fly *Drosophila melanogaster* that may finally explain how the tidy array arises.

Researchers have known for years that during fly eye formation an indentation known as the "morphogenetic furrow" sweeps across the imaginal disc from back to front, leaving behind rows of perfectly spaced ommatidia. "The furrow is the transition from no pattern to pattern," says Cagan. "But how do you get the pattern? That's one of the Holy Grails of fly genetics." Patricia Powell and Susan Spencer, postdocs in Cagan's lab at Washington University in St. Louis, have now gained a handhold on that prize.



Compound Interest. Lingering *atonal* activity (green) marks the cells that will be ommatidia.

They have shown that as the furrow moves, cells destined to create a new ommatidium supply a protein signal that prevents the cells immediately ahead from forming another ommatidium. Instead, they form the narrow interommatidial spaces, while the cells between these spaces, which have not received the inhibitory signal, differentiate into ommatidia. These in turn release the inhibitory signal, and the process repeats until the furrow fully traverses the disk. "Periodic spacing patterns are everywhere in development, but we know very little about how the placement of one pattern element affects the positioning of the other," says Don Ready, a developmental biologist at Purdue University. "Cagan's results open [this] to molecular genetic attack."

To come up with this model, Cagan's group started by looking at the expression of a gene called *atonal* (*ato*), shown by previous researchers to provide a signal crucial to ommatidium development by instructing one cell to become the central, so-called "R8" photoreceptor. As the furrow moves forward, other studies had shown, *ato* is expressed in the cells just in front of it. When the furrow engulfs the *ato*-expressing cells, *ato* expression is switched off again, but Cagan's team noticed that while this shutdown occurs immediately in the cells destined to become interommatidial spaces, it takes longer in the future R8 cells.

Because these results indicated that the timing of *ato* expression is key to ommatidia differentiation, the group went looking for the signals that control it. They eventually found that as the furrow approaches, the activity of *ato* is turned down by a signal sent through the Ras pathway, a well-known intracellular signaling cascade. This doesn't happen right away, however, in cells destined to be ommatidia, because the Ras signal they receive is apparently weakened when it meets up with another protein called Rhomboid. The combination of Ras and Rhomboid also induces the expression of a protein called Argos, which interferes with the Ras pathway and further slows the shutdown of *ato*.

Argos, however, seems to prevent this same process from unfolding in the next row of cells. By tagging Argos with a fluorescent marker, the group saw that each nascent ommatidium squirts the protein forward into the pending row. Earlier research had shown that Argos blocks the expression of Rhomboid, so with that observation, a model for the entire patterning pathway clicked into place.

When it reaches the cells of the unpatterned region immediately ahead of the furrow, Argos inhibits Rhomboid there. That blocks the signals that slow the shutdown of *ato*, resulting in an interommatidial space. Without Rhomboid, however, the Ras pathway can't make new Argos, so more distant cells in the unpatterned region don't receive Argos early, allowing them to produce a new ommatidial field—which in turn activates new Argos to continue the cycle. Thus, each ommatidium clears the region ahead of it, ultimately resulting in a hexagonal pattern. Cagan's group is now attempting to verify each of these steps, by studying how the pattern is affected when various components of the system are knocked out.

Organs Made to Order

A developing organism's cells are like highly trained orchestra members: Each carries the full genetic score but has to listen to cues from the cells around it to know when to play its own specific portion. Now, scientists from Japan have learned to administer a key cue to undifferentiated cells from newt and frog embryos, enticing them to play whole movements by growing into finished organs such as a liver or even a beating heart. If a similar approach can be made to work for mammals, it might aid efforts to construct replacement human organs from embryonic and fetal tissues.

For the work, developmental biologist Makoto Asashima and his colleagues at the University of Tokyo used cells taken from a part of the early amphibian embryo called the animal cap, a region that normally expands to form all the embryo's mesodermal (middle) tissues such as muscle and internal organs. Several years ago, the researchers noticed that low concentrations; about 0.5 nanogram per milliliter (ng/ml) of activin—a protein known to be important for organ formation in the intact embryo—in the culture fluid caused animal cap cells to develop into red or white blood cells, while slightly higher doses induced formation of muscle tissue. Once initiated, these tissues' developmental programs seemed to run without external input. "This led me to wonder whether it would be possible to create a complete, functional organ in vitro," Asashima says.

He has accomplished just that. A 50 ng/ml dose of activin produced a notochord, a rod of cells along the embryo's dorsal surface that gives rise to much of the nervous system. A dose of about 75 ng/ml gave a heart, complete with heartbeat, while 100 ng/ml yielded a liver. By adding other substances such as retinoic acid and insulin-like growth factor at various points in development, Asashima also made a pronephros—a precursor to the kidneys—and rudimentary eyes and ears. "The question of what purified factors can do, exemplified by the work of Asashima and colleagues, is very interesting," says Hazel Sive, a molecular biologist at the Whitehead Institute for Biomedical Research in Cambridge, Massachusetts. "Maybe if you get the right tissues and the right factors, you could get [human organs] to regenerate in a dish, which is what everybody really wants."

–Wade Roush