Planarian regeneration does have clear differences, however. The most significant seems to be that the worms do not dedifferentiate cells to create the blastema. Instead, planaria turn to cells called neoblasts.

Scattered within the planarian body, neoblasts apparently remain in an unspecialized, stem cell state, which enables them to differentiate into any cell type. Wherever planaria are cut, the neoblasts migrate to the site and form a blastema by themselves.

Sánchez' colleague Phillip Newmark has kept planarian neoblasts alive in petri dishes for several weeks, an advance the researchers hope will allow them to genetically alter the animals. The investigators will try to slip genes into the neoblasts or mutate existing genes, then add the cells to worms whose own neoblasts have been destroyed with radiation. When such worms regenerate, any new cells should derive from the genetically engineered neoblasts, says Sánchez.

Efforts to identify planarian regeneration genes have already offered some promising leads, Sánchez' group reported at last summer's International Congress for Developmental Biology in Snowbird, Utah. With a technique called subtractive hybridization, the researchers collected fragments of genes turned on in blastemas when a planarian regenerates its tail, its head, or both and compared them to genes normally active in the head and tail. Genes active only in the blastemas presumably participate in regeneration, notes Sánchez.

The investigators detected dozens of DNA fragments specific to regenerating blastemas, some belonging to genes that become active in the initial hours of the process. In several cases, the researchers have found that the fragments are parts of genes found previously in other animals.

One such gene encodes an enzyme that degrades the extracellular matrix, a mesh of proteins and other molecules that surrounds cells. This enzyme may trigger regeneration by releasing growth factors bound to the matrix or by eliminating obstacles to a cell's proliferation and movement, says Sánchez.

Is the notion of regenerating human limbs or diseased tissue a pipe dream or a realistic expectation for the 21st century? It's far too early to know whether there are insurmountable differences between animals that can regenerate and those that cannot, says Jeremy P. Brockes of University College London.

He and his colleagues have recently shown that some factor present in blood serum, newt or any other kind, can induce newt muscle cells to dedifferentiate. The muscle cells of nonregenerating vertebrates don't respond to the serum, however, indicating that they are not sensitive to this still unidentified factor.

If feasible, overcoming this insensitivity might prove invaluable. Cardiac specialists might then learn to trigger damaged heart muscle to replace itself. Such an advance could be possible even if scientists never obtain sufficient expertise to regenerate complex structures like limbs, suggests Brockes.

Given the potential value of regeneration in medicine, Stocum finds it puzzling that so many scientists choose to study cell and organ transplantation or work on building artificial hearts, livers, and limbs rather than investigate animals that can regrow parts of themselves.

"With regeneration, you don't have to worry about [finding] donor cells, and you don't have to worry about immune rejection," he says. "Regeneration is clearly superior to any kind of transplantation or artificial tissues." □

To find a link to an animated version of limb regeneration, visit SCIENCE NEWS ONLINE *at* ***http://www.sciencenews.org***.

Nutrition

Getting older—and a little rounder?

A person's body fat typically doubles between the ages of 20 and 50. While this middle-aged spread is usually attributed to eating too much and exercising too little, there may be more to it than that. The body's ability to break down and use large quantities of fat drops with age, a new study finds. As a result, more of the fat eaten at large meals will become body fat instead of being burned as energy, explains Susan B. Roberts of the Department of Agriculture's Human Nutrition Research Center on Aging at Tufts University in Boston.

On four different days, her team analyzed how 16 women—half of them in their twenties, the rest over 60—metabolized various portions of peanut-butter-and-jelly sandwiches and milk. The four test meals ranged from a 250-calorie snack to a 1,000-calorie feast that represents two-thirds of the average 60-year-old woman's daily energy intake.

The two groups processed the fat in the smaller meals with equal efficiency. However, the older diners burned only 70 percent as much fat from the big meal as those in their twenties, the researchers report in the October AMERICAN JOURNAL OF CLINICAL NUTRITION. One explanation may be the fact that older women possess more glucagon, a hormone that instructs the body to put more sugar into the blood. "That's not good," Roberts says, because the more readily used sugar discourages the body from burning fat. In their thirties, people begin to lose skeletal muscle (SN: 8/10/96, p. 90), the site of most fat metabolism.

To cope with these age-related changes, Roberts suggests, older diners should not only eat less at a sitting but also exercise more—to strengthen and retain fat-burning muscle. —*J.R.*

Boning up on fish oil

For decades, nutritionists have extolled the virtues of fish oils and other foods rich in omega-3 fats as a means of keeping the heart healthy. Studies in animals now suggest that this type of fat also aids bone. If the same holds for humans, physicians may one day add omega-3 fats to the dietary regimen they prescribe to help stave off osteoporosis.

Most polyunsaturated fats fall into one of two groups: the omega-6 fats found in corn oil and the omega-3s found in ocean fish, flaxseed, green vegetables, and some nuts.

In recent years, the omega-6 content of Western fare has been climbing—often at the expense of omega-3, which now constitutes around 7 percent of polyunsaturated fats in the typical diet, notes Bruce A. Watkins of Purdue University in West Lafayette, Ind. At an international omega-3 conference in Bethesda, Md., last month, he reported on his studies of chicks and rats. As omega-3 dropped from 50 percent of polyunsaturates to some 12 percent, bone formation also fell—by about 20 percent.

The fat's apparent stimulation of bone growth seems to arise from its effect on at least two hormonelike compounds. When omega-3 makes up only 12 percent of polyunsaturates in the diet, production of prostaglandin E (PGE_2), a "local hormone," is high, limiting bone growth, Watkins says. Increasing the omega-3 share of polyunsaturates to 50 percent halved the bone-making cells' production of PGE_2, bringing it to a level more conducive to bone formation.

The same dramatic increase in omega-3 fats enhanced the activity of insulinlike growth factor (IGF) by 50 percent and increased significantly the production of a protein that ferries IGF around the body, Watkins' data show. Production of IGF, which fosters continual growth and remodeling of bone, typically falls in old age, precipitating the bone loss underlying osteoporosis.

"I think the message here," Watkins told SCIENCE NEWS, "is that when the diet supports more responsive remodeling, you can shape the bone to be stronger." —*J.R.*