tube of graphite capped on the ends by two hemispheres of a buckyball, while the other, slightly larger form consists of 10 to 20 concentric cylinders of carbon. Lieber used the latter as a template for the carbide nanorods.

The key to making such small fibers was starting with nanotubes, says Richard E. Smalley of Rice, a codiscoverer of buckyballs who currently works with carbon nanotubes.

"To make a nanometer structure . . . that will survive in the real world, you must construct a surface where the atoms are happy," Smalley says. "We're beginning to learn about nanotubes and how to use them as a . . . template for other elements. Carbon is teaching them how to do it."

Lieber's carbide nanorods were between 2 and 30 nm in diameter and over 1,000 nm long. Although many of the rods were smooth and straight, some of them had unusual shapes. Lieber observed sawtooth titanium carbide nanorods and niobium carbide ones with a helical structure.

The properties and shapes of the nanorods open up many possible applications. Lieber found the niobium carbide nanorods to be superconducting and the iron carbide ones ferromagnetic. Hongjie Dai, a physical chemist who works with Lieber, speculates that passing a current through a corkscrew-shaped nanorod would produce "a very interesting magnetic field profile."

Lieber and his colleagues are continuing to characterize the properties of the nanorods, not an easy task considering the difficulty of handling such small structures. They plan to continue studying possible applications and making nanorods out of different carbides. "In principle, you can make all the carbides that exist," Dai says. — *Corinna Wu*

Lieber

Carbon nanotubes about 10 nm in diameter.

Diet, exercise, genes strengthen bones

Study after study these days touts the benefits of eating right and exercising. But the value of that advice may depend on your genetic inheritance.

A study of a gene associated with the development of strong bones indicates that the importance of exercising and drinking milk varies with the type of vitamin D receptor *(VDR)* gene your cells harbor.

"We found that the combination of dietary calcium intake and exercise can overcome a poor genetic predisposition," says epidemiologist Loran Salamone of the University of Pittsburgh team that conducted the study, "while for those with a beneficial genetic makeup, little activity and low calcium intake didn't adversely affect [bone strength.]"

The finding may help identify women who need to work at developing strong bones.

Through early adulthood, people continually add calcium and strength to their bones, which reach a peak density around age 35. Women rapidly lose bone density after menopause, and their ability to avoid osteoporosis—severe bone loss that can lead to life-threatening fractures—depends upon their bone density before menopause.

In 1994, researchers discovered that the vitamin D receptor is vital to achieving high bone density. But not all vitamin D receptors are the same. The *VDR* gene, which carries the blueprint for making the receptors, comes in two varieties. Both produce functional vitamin D receptors, but one stores calcium in bone a little more efficiently.

People who inherit two copies of the more efficient form of *VDR*—known as *b*—develop high bone densities. Those who inherit two copies of the less efficient gene—known as *B*—have somewhat less strong bones.

Salamone knew that a person's *VDR* gene complement would help determine bone strength, but she figured that factors such as dietary calcium and exercise would also play a role. She and her colleagues studied 470 premenopausal women age 44 to 50. They interviewed the women about their diets, exercise habits, and hormone usage, then analyzed the women's bone density and genetic makeup.

As Salamone reported at a meeting of the Society for Epidemiologic Research in Snowbird, Utah, in June, women with two *B* forms of *VDR* who exercised the most developed 7 percent higher bone density than women with the same genes who exercised the least. Women harboring *B* who had high calcium intake and who exercised enjoyed 10 percent more bone density, but a calcium-rich diet without exercise had no beneficial effect.

Women with the *b* genes developed strong bones regardless of exercise and calcium intake.

"This is a wonderful study," says epidemiologist Marian T. Hannan of the Boston University Arthritis Center. Salamone "has shown that it is possible to make modifiable changes that can have an impact on osteoporosis."

Salamone points out that her study population is white, so she isn't sure how the genes affect black women, who ordinarily have much higher bone density.

— *L. Seachrist*

Gene for early, aggressive Alzheimer's

An international team of scientists has discovered a gene for a rare, but very aggressive form of inherited Alzheimer's disease. The gene may be responsible for the majority of familial cases of Alzheimer's that strike before the age of 60.

Alzheimer's usually sets in after age 65, but this early-onset form ravages its victims in midlife, affecting some people as early as their thirties. Even though this gene accounts for only a small percentage of all cases, its discoverers hope their work will aid understanding of the disease in all its forms.

"The gene may not be mutated in other forms of Alzheimer's disease," says team member Peter St. George-Hyslop of the University of Toronto. "But it probably does play some role."

Previously, scientists had noted that mutations in the gene for apolipoprotein E, found on chromosome 19 (SN: 8/14/93, p.108), played a role in some familial cases that begin after age 65. And mutations in the gene for beta-amyloid precursor protein, found on chromosome 21 (SN: 2/23/91, p.117), account for some early-onset cases of Alzheimer's.

To find this third Alzheimer's gene, the team gathered genetic information from 21 families afflicted by the early-onset form of the disease.

In six of those families, the researchers report in the June 29 NATURE, mutations in the *S182* gene on chromosome 14 account for up to 70 percent of all early-onset cases of Alzheimer's. What's more, the mutated gene amounts to a ticking time bomb; virtually all who inherit it will be stricken during midlife.

How the mutation leads to Alzheimer's remains a mystery. The researchers speculate that the protein produced on instructions from the mutated gene may not process beta-amyloid precursor protein correctly.

The finding will enable researchers to explore the function of the gene in animal models, St. George-Hyslop says. And because mutations in *S182* occur in only 6 of the 21 early-onset families, he believes that research will uncover a few more genes associated with Alzheimer's.

— *L. Seachrist*