Nanotubes: Metallic by a twist of fate

When a tailor mismatches the stripes in a fabric while sewing a shirt, the garment merely looks funny. However, when a single atomic layer of graphite rolls up into the minuscule cylinder known as a nanotube, the angle at which the edges join can have a dramatic effect on the tube's electric conductivity.

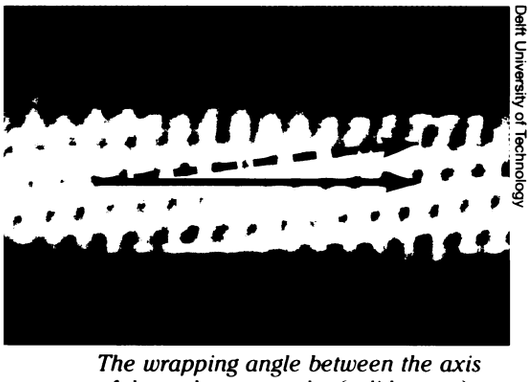
Even though they are made of exactly the same material, some carbon nanotubes conduct electricity as easily as a metal does, while others act as semiconductors, blocking the passage of low-voltage current. This variation, first predicted by three research groups in 1992, has now been observed by two teams of scientists, one from Harvard University and the other from Rice University in Houston and Delft University of Technology in the Netherlands. Their findings appear in the Jan. 1 NATURE.

Both teams used a scanning tunneling microscope to determine the diameter and spiral angle of a nanotube. They then measured the tube's conductivity with a miniature probe.

"It's remarkable how one small change in a nanotube's structure can make a tremendous difference in its electrical behavior," says Andrew G. Rinzler of Rice.

In graphite, each carbon atom links to three others, forming a hexagonal lattice that resembles a slice through an atomic-scale honeycomb. The regularity of this pattern allows the edges of a sheet of graphite, when rolled into a cylinder, to match seamlessly at several different angles.

"This landmark work goes a long way toward telling us that the basic theory is correct," says Mildred S. Dresselhaus, a physicist at the Massachusetts Institute of Technology. However, she adds, additional experimental and theoretical work needs to be done to link some of the teams' observations to the details of the theory. *—S. Perkins*



Delft University of Technology

The wrapping angle between the axis of the carbon nanotube (solid arrow) and the row of holes in the graphite's hexagonal atomic lattice (dotted arrow) affects the nanotube's electric conductivity.

Chimp brains show humanlike tilt to left

Human language abilities depend on tissue located mostly on the left side of the brain, or left hemisphere. A new study finds that the common chimpanzee, despite its inability to speak, shares with people one feature of this anatomical pattern—a structure called the planum temporale is larger on the left side of the brain than on the right.

In humans, a swath of neural tissue, known as Wernicke's area, encompasses the entire planum temporale and helps to orchestrate language comprehension.

A larger planum temporale in the left hemisphere also characterized the common ancestor of chimps and humans, a creature that lived around 8 million years ago, contends a scientific team headed by neurobiologist Patrick J. Gannon of Mount Sinai School of Medicine in New York. Whether the planum temporale fostered species-specific forms of communication or assumed other responsibilities during the course of evolution remains unclear, the group notes.

"This is a great contribution to the field [of comparative brain studies]," says neuroscientist Katerina Semendeferi of the University of California, San Diego. "Now we need to look more closely at brain organization in all apes."

Semendeferi suspects that the larger left-brain planum temporale exists in gorillas and orangutans, as well as in chimps. She directed a related study, published in the April 1997 JOURNAL OF HUMAN EVOLUTION, indicating that the brain's frontal lobe—often assumed to have expanded greatly in humans to support complex thought—is actually about the same size, relative to overall brain volume, in all apes.

Gannon's team conducted a microscopic analysis of the surface of 18 preserved chimp brains. The group identified anatomical landmarks delineating the planum temporale and then calculated its surface area in each hemisphere. A pronounced left-side size advantage appeared in 17 of the brains, the researchers report in the Jan. 9 SCIENCE.

An investigation reported 20 years ago found few signs of the planum temporale in chimps. As a result, some scientists thought this brain area was poorly developed in nonhuman primates.

"If Gannon's group is able to see planum temporale landmarks in chimps, they're to be congratulated," comments anthropologist Dean Falk of the State University of New York at Albany. "Their finding fits with some previous research on asymmetry in chimp brains."

The greatest left-right asymmetry of the planum temporale has been reported in people with perfect pitch, Falk notes. In chimps, a similar left-hemisphere emphasis may help in processing melodic aspects of their vocal communication, she theorizes. *—B. Bower*

Hugo Van Lawick/Discovery Channel

Left turn for chimp brains.

Ebola virus vaccine protects guinea pigs

Few diseases have as fearsome a reputation as Ebola fever. Just to handle the Ebola virus, scientists must wear space suits and employ the strictest biohazard precautions. This rare illness is transmitted by close contact and most often kills humans swiftly. There is no effective treatment for Ebola fever and, so far, no way to prevent it.

In their efforts to make an Ebola vaccine, scientists have tried traditional methods—such as using an inactivated virus or a slightly modified version of a live Ebola virus—with some success in laboratory animals. These approaches can make researchers uneasy, however.

"I don't know that I'd be willing to take an injection of a purified, inactivated strain" of Ebola, says Anthony Sanchez, a virologist at the federal Centers for Disease Control and Prevention (CDC) in Atlanta. "And we can't predict what an attenuated live virus would do in a person."

Instead, Sanchez and other researchers are exploring the nascent field of gene vaccination in hopes of producing a riskfree inoculation that will protect against the Ebola virus. By injecting the genes that normally encode some of the virus' proteins, researchers at the University of Michigan Medical Center in Ann Arbor and the CDC have rendered 15 of 16 guinea pigs impervious to the Ebola virus when exposed to it less than 2 months later. Six unvaccinated guinea pigs died.

Of 10 guinea pigs exposed to the live virus 4 months after inoculation, 7 survived; all of the unvaccinated animals died. The team's findings appear in the January NATURE MEDICINE.

Although the intact Ebola virus seems to thwart the body's immune and inflammatory mechanisms when it infects a person, the treated guinea pigs pro-

duced antibodies and T cells to battle the virus, says molecular virologist and study coauthor Gary J. Nabel of the Howard Hughes Medical Institute at the University of Michigan.

An Army research team has used a slightly different technique to prevent Ebola fever and Marburg fever, a related disease, in mice and guinea pigs. In one promising series of experiments, the researchers generated a self-replicating RNA molecule from a modified Venezuelan equine encephalitis virus and used it to carry an Ebola or Marburg virus gene that encodes a glycoprotein.

Injected into a guinea pig or mouse, this vaccine stimulates an immune response against the Ebola or Marburg virus and protects the animal. The researchers, from the U.S. Army Medical Research Institute for Infectious Diseases (USAMRIID) in Frederick, Md., describe their technique in the Dec. 22, 1997 VIROLOGY.

This week, the group began testing the technique on 12 cynomolgus macaque monkeys, the first study of primates that has used an Ebola gene vaccine. Similar research on the Marburg virus in monkeys is already under way at USAMRIID, says Jonathan F. Smith, a virologist at the institute. "The real crunch issue is primate testing," Smith says. "This is crucial."

The Marburg virus was first identified in Marburg, Germany, in 1967. Ebola fever emerged near the Ebola River in the Congo, then Zaire, in 1976. Since then, researchers have identified other Ebola strains, but the Zairian virus remains the most deadly for humans, causing high fever and sometimes bleeding from every orifice.

In various outbreaks, Ebola fever has killed 50 to 90 percent of its victims. In a 1995 outbreak in Kikwit, Zaire, more than 280 people died; a spate of cases a year later in Gabon killed 43.

Because of safety protocols governing work with the Ebola virus, researchers have proceeded cautiously. For example, the protocols limit how many animals may be used, and extreme care must be taken in laboratories to avoid needle sticks or animal bites.

Some scientists are studying antiviral medications in hopes of treating Ebola victims. "Ideally, you have a vaccine and therapy," says James M. Meegan, a virologist at the National Institute of Allergy and Infectious Diseases in Bethesda, Md. "If you're the person in the [space] suit, you'd like both of those." —*N. Seppa*

A meaty answer to a nosy question

Ah, the sweet smell of . . . meat?

For one group of investigators, the odor of success is octanal, a molecule that most human noses perceive as a meaty smell. In the first case where a specific odor and its mammalian receptor have been definitively shown to work together, this team has identified a cell surface protein that enables rat nasal cells to perceive the octanal molecule.

Several years ago, scientists discovered a large family of genes, numbering as many as a thousand, all of which encode cell surface proteins made by the sensory nerve cells within the mammalian nose.

While investigators believe that these proteins act as receptors for odorants, the free-floating molecules sensed by the olfactory system, they have had trouble linking odorants to specific receptors.

To study a putative receptor called I7, a research group headed by Stuart Firestein of Columbia University engineered viruses to carry extra copies of the gene for I7 as well as a gene that encodes a fluorescent marker.

After infecting the rats' nasal cavities with the viruses, the scientists identified fluorescently labeled sensory cells and sprayed them with various odorants, one at a time. A device called an electro-olfactogram, which measures electric impulses generated within cells, enabled the researchers to determine whether the cells recognized any of 65 sprayed odorants.

For 64 of the odorants, the electro-olfactogram detected similar responses from both infected and uninfected nasal cells. For octanal, however, the response of infected cells was significantly quicker and stronger, presumably because the cells were binding the chemical with the additional copies of I7 on their surface.

The investigators then tested odorants that are structurally related to octanal. The infected cells responded to smells that had a meaty or waxy smell, but not to those that smelled more like grass or fruits, thus demonstrating the receptor's ability to distinguish small differences among odorants, Firestein and his colleagues report in the Jan. 9 SCIENCE.

By connecting specific odorants to receptors, researchers may learn which features of receptors are crucial to recognizing smells and how a thousand or so receptors can distinguish among the estimated 10,000 odorants, says Firestein.

"Every receptor is going to bind more than one thing, and every [odorant] is going to bind more than one receptor," notes olfactory researcher Glenn D. Prestwich of the University of Utah in Salt Lake City. —*J. Travis*

Sulfur speeds oil formation in lab

Once, a popular rags-to-riches scenario involved stumbling upon an unknown oil deposit under the backyard—a discovery valuable enough to turn an ordinary citizen into an oil baron. In reality, petroleum companies spend a lot of money and effort trying to predict before they drill whether a spot will yield a gusher or a dry well.

Oil deposits build up from layers of organic matter that decomposes—over enough time and at sufficiently high temperatures—into a complicated mix of hydrocarbons. Oil companies use mathematical models to calculate whether enough time has passed for a particular set of geologic conditions to yield a pot of black gold.

The roil of chemical reactions that turns dead plants and animals into oil can thwart even the best prognosticators. One ingredient, a reactive form of sulfur, appears to be critical in determining how quickly oil forms, says Michael D. Lewan of the U.S. Geological Survey in Denver.

In their painstaking search for hidden deposits, petroleum companies occasionally find oil in surprising places. These unexpected deposits, which tend to be rich in sulfur, prompted Lewan to propose in 1985 that sulfur-containing organic material turns into oil more readily than standard models predict.

He suggested that decomposition might go faster simply because carbon-sulfur bonds split more easily than carbon-carbon bonds, but the explanation didn't account for the diversity of components in petroleum, he says. He then theorized that sulfur radicals could accelerate breakdown.

Lewan's recent experiments show that a reactive form of sulfur speeds up oil formation by stimulating the breakdown of hydrocarbon molecules. He baked a hydrocarbon—chosen to mimic a partially decomposed precursor to oil—in a closed capsule at 350°C for 3 days, with and without a sulfur compound. The compound, known to create radicals, increased hydrocarbon breakdown by more than 20 percent, Lewan reports in the Jan. 8 NATURE.

The presence of these sulfur radicals could help explain the composition of petroleum and offer a new way of estimating the time it takes for oil deposits to form in the earth, he says.

Alan Burnham of the Lawrence Livermore (Calif.) National Laboratory disagrees with Lewan's analysis, saying that the results don't reveal the real-world relationship between time, temperature, and sulfur content. "Everyone agrees that sulfur lowers the temperature of oil formation," he says, "but the question is, by how much?" —*C. Wu*