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*