calories for the average person rebuilds the fat layers of at least some of the formerly obese.

"It's looking as though obesity may turn out to be a chronic illness for some people," says Rudolph Leibel, a researcher at Rockefeller University in New York City. "It's probably wishful thinking for these people to believe that if they can just get the weight off they can go back to a 'normal' diet."

Leibel studied 12 men and 14 women before and after they lost an average of 100 pounds each. By feeding each volunteer a liquid diet of known quantity and composition, and precisely adjusting the amounts given, Leibel and co-workers were able to determine exactly the caloric intake each person needed to maintain a stable weight. They then induced a weight loss by reducing consumption to roughly 600 calories per day, and compared the now-slimmer subjects with persons of the same size who had never been overweight.

"The results were striking," Leibel says. The number of calories an average person needed to maintain his or her weight was quite similar to the number an obese person used *when obese*. After weight reduction, the formerly obese needed 28 percent fewer calories. From an energy metabolism perspective, obese subjects were more "normal" in obesity than after weight loss.

After their weight loss, Leibel told Science News, the formerly obese patients in his study often complained of persistent feelings of fatigue, mental depression, intolerance of cold and irregular menstrual periods — many of the same symptoms suffered by women with the eating disorder anorexia nervosa. The similar symptoms were found to persist as long as patients maintained their weight at the reduced level. This situation might reflect a similarly deranged metabolic setpoint in the two conditions, Leibel says.

It's still not clear whether the weight loss induced the metabolic change, or simply unmasked an underlying condition. Leibel and colleagues are currently searching for biochemical markers that might help them detect persons with an artifically high metabolic setpoint before they become obese.

Whose ape is it, anyway?

One sometimes wonders whether orangutans, chimps and gorillas ever sit around the tree, contemplating which is the closest relative of man. (And, would they want to be?) Maybe they even chuckle at human scientists' machinations as they race to draw the definitive map of evolution on earth. If placed on top of one another, all these competing versions of our evolutionary highways would make the Los Angeles freeway system look like County Road 41 in Elkhart, Ind.

As was inevitable, a number of these anthropological architects met head-on at the AAAS intersection. And the results either opened up some new lanes or added some new road blocks, depending on one's perspective. Jeffrey H. Schwartz, associate professor of physical anthropology at the University of Pittsburgh concludes — after analyzing data on numerous fossils — that humans are closest, evolutionarily, to the orangutan, rather than to gorillas or chimps. Schwartz's theory appears to mesh with a previously reported finding that *Ramapithecus*, once widely viewed as the earliest known hominid (member of the human family), is actually an ancient ancestor of the orangutan (SN: 2/6/82, p. 84).

Disputing Schwartz's findings, however, was a report by Yale University scientists that genetic comparisons of humans and a number of primates demonstrate that chimpanzees are humans' closest relatives. Other groups have argued that gorillas indeed evolved on the closest track to humans.

Schwartz says that "a simple solution" to the question of why hominids, orangutans and *Ramapithecus* are so similar to one another is that all three "inherited these features from a common ancestor not shared with any African ape."

Biology

Gardiner Morse reports from Beltsville, Md., at a USDA symposium on biomembranes

Kinky membranes

When people get cold, they put on warmer clothes. When cells get cold, they often change their membrane structure. Cells have a variety of ways to do this. Researchers studying these processes hope to find new ways to protect plants against low temperature and other stresses, reports Guy A. Thompson Jr. of the University of Texas in Austin.

All biological membranes share a common basic structure: They are sandwiches of phospholipid molecules with their hydrophilic (water-loving) heads on the outside and hydrophobic (water-fearing) tails pointing inward. The tail's structure helps determine the membrane's fluidity. One common way membranes stay fluid in the cold is by increasing the number of lipid tails that are unsaturated (kinked with double bonds) Thompson explains. This prevents the tails from locking as easily with one another into a frozen lattice.

Many chilled cells depend in part on a "desaturase" enzyme to keep their membranes fluid. This enzyme converts single to double bonds in the lipid molecule tails. Thompson and others would like to know how this enzyme is regulated. One explanation Thompson favors derives from the tendency of enzymes to lose activity as they are cooled. There is evidence that desaturase enzymes embedded in the membrane slow down *less* when the cell is cooled than do enzymes which supply new saturated lipids to the membrane, he says. The result is that the ratio of unsaturated to saturated lipids in the membrane increases — that is, more tails are kinked by double bonds — and the chilled membrane stays fluid.

Anti-aluminum plant acids

Aluminum is toxic to many crops. One way it does its damage is by binding with calmodulin, a protein in plant cell membranes that plays a key role in regulating a variety of enzymes, says Alfred Haug of Michigan State University in East Lansing. "Aluminum puts specific lesions on calmodulin," Haug explains, and these can cause the malfunction of enzymes under its direction.

Some plants are more tolerant of aluminum-tainted soil than others, says Haug, and their high concentration of organic acids may be what protects them. He has shown that organic acids such as citrate shield calmodulin from aluminum by combining with the metal. One obvious strategy for making plants resistant to aluminum damage is to breed them for high organic acid content, Haug suggests.

Stop that sperm

Sperm may look determined swarming toward an egg, but once they get there, hours can pass before one of them finally fertilizes it. This is because sperm can't penetrate an egg until they've been "capacitated" — given the chemical go-ahead to release enzymes that breach the egg's membrane. Brian Davis of the Research Foundation of Southern California in La Jolla reports that microscopic membrane vesicles in the seminal plasma of various mammals can keep rabbit sperm on hold by apparently donating cholesterol to the sperm's membrane. This maintains the membrane in a state that prevents the release of its egg-piercing enzymes. Inactivated sperm have "tough outer skins," Davis explains.

Once the sperm are on their way to the egg, the inhibiting vesicles, which have accompanied them so far, are stopped in the cervical canal. The sperm swim unescorted into the uterus where uterine fluid depletes the sperm's membrane cholesterol and capacitates it over a period often lasting hours, Davis says.

Why such an elaborate system? One reason is that "the male is producing cells which are like little time-bombs," Davis told Science News. "If those reactions go off prematurely, the male tract just undergoes autolysis" — self-destruction by the sperm's powerful hydrolytic enzymes.