Self-Awareness: Humans Are Not Alone

An apparent hole detected in the mental evolutionary chain places chimpanzees and orangutans closer to human thought levels and further away than believed from other primates, a State University of New York at Albany psychologist reports. "It has been argued that there is a [mental] continuity from one animal to another; that they differ by a matter of degree," says Gordon G. Gallup Jr. Now, however, Gallup has pinpointed a "sudden change, a void, between great apes and others."

The critical factor, as Gallup describes in the May AMERICAN PSYCHOLOGIST, is self-recognition, an ability that until recently was thought to be inherent only in human beings. But experiments by Gallup and others since around 1970 yield "striking evidence" that chimps and orangutans indeed possess a sense of self. (Gorillas, the third member of the great ape catagory, have not yet been tested for self-recognition.)

Gallup's most recent research, soon scheduled for publication, provides further evidence that other primates do not possess the great ape's ability to conceive of their "selves." In that study, a wild-born, preadolescent macaque monkey was exposed to a mirror for 2,400 hours for a period of more than five months, but it failed to show any convincing evidence of self-recognition.

In contrasting studies over the last several years, chimpanzees and orangutans began to show signs of self-recognition after only two to three days of mirror confrontation. Gallup has shown that once exposed to mirrors, chimps move rather quickly from treating the image as if it were another chimp to recognizing it as themselves. "They used the reflection," he says, "to gain visual access to and to experiment with otherwise inaccessible information about themselves, [such as] grooming parts of the body that could not be seen directly, picking bits of food from between their teeth, blowing bubbles and making faces at the mirror."

Still Gallup sensed that his colleagues "might not be terribly convinced or enamored" with his conclusion that such behavior meant the chimps had really identified the source of the reflection. So, following an initial round of mirror exposure, each animal was anesthetized and painted around the eyebrow and ear with a bright red, odorless, nonirritating dye. Convinced that the chimps could not realize they had been so marked, Gallup placed them back into cages, first without a mirror, then with one.

He found that the chimps' attempts to touch a marked area on themselves increased by a factor of more than 25 times in the presence of a mirror. Not only did the incidence of such behavior increase, but so did the viewing time. In addition—even though the dye had long since dried and was indelible—the chimpanzees attempted to visually examine and smell the fingers used to touch the marked areas.

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Lab workers support head of anesthetized chimp after marking certain facial areas with red dye for self-recognition test (marks not visible in photograph).

The implications of such results, as Gallup suggests, are far reaching. "To the extent that self-recognition implies a rudimentary concept of self, these data show that, contrary to popular opinion, man may not have a monopoly on self-concept," he says. The psychologist is not sure of the cause of the apparent gap between great apes and other primates, such as monkeys and baboons (which have also been tested). He theorizes the discrepencies may be due to differences in the size of cortical mass among the animals or to an inherent "threshold phenomenon" that provides some animals, but not others, with the ability to self-conceptualize.

"Primate research poses one of the greatest contemporary threats to traditional notions about man," Gallup says. "Man may not be evolution's only experiment in self-awareness." □

Rat insulin gene spliced into bacteria

Manufacture of human insulin in bacteria has been perhaps the most obvious and the most tempting promise of the recombinant DNA techniques. As the number of diabetics increases worldwide, a shortage is developing of the beef and pig insulin that has allowed many diabetics to live nearly normal lives. Furthermore, a source of human insulin could help diabetics who become allergic to the animal hormones.

Now the first step toward insulin manufacture in bacteria has been accomplished. Researchers at the University of California at San Francisco announced this week that they have successfully placed a mammalian insulin gene into a bacterium. Although the insulin gene originated from a rat, rather than from a person, the researchers believe that the same techniques could be used for a human gene. However, putting the human insulin gene into bacteria would require, according to the National Institutes of Health guidelines, stricter safety measures in tightly sealed laboratories such as those being constructed at NIH and Ft. Detrick.

The insulin genes that Howard Goodman, William Rutter and co-workers inserted into the bacteria were not the actual genes dissected from a rat chromosome. Instead the researchers introduced a copy of a copy. The first copy is messenger RNA, the short-lived intermediate that carries genetic information from the cell nucleus to the protein-making apparatus in the cytoplasm. Because cells in the rat pancreas produce predominately one protein, insulin, most of the messenger RNA in those cells contains the information for producing that hormone. With a special enzyme called reverse transcriptase, the investigators made DNA copies identical to the functioning gene from the messenger RNA. This procedure has also been used for study of human genes (SN: 5/7/77, p. 294). The new DNA copies were then joined to rings of bacterial DNA, and those rings, or plasmids, inserted themselves into the bacteria *Escherichia coli*. The bacteria containing insulin genes were easily identified, because the plasmids also carried genes that make bacteria resistant to drugs.

Although the transferred insulin gene can be reproduced within the bacterium, it cannot actually produce insulin there. So far no mammalian gene has functioned to make protein after researchers relocated it into a bacterial cell. However, there is evidence that genes of yeast can function in bacteria (SN: 3/12/77, p. 165). Rutter and Goodman predict that, despite the complicated mechanisms of insulin production, within a year they will be able to persuade rat genes to function in bacteria and actually direct manufacture of rat insulin.

If bacteria cannot yet function as an insulin factory, they can already function as a gene factory. The UCSF researchers have discovered details about the insulin protein by carefully analyzing the abundant copies of the insulin gene produced in bacteria. Production of insulin in the

pancreas cells is a process of several steps. First a long protein chain is synthesized, then enzymes clip it into several pieces, two of which combine to form the final insulin molecule. From their analysis of the DNA, the researchers were able to deduce for, the first time, the amino acid sequence of several discarded parts of the precursor molecule. The investigators also determined the sequence of the DNA just beyond the insulin gene. That region of DNA does not code for any protein, but it may contain the specific signals that control when protein will be made from the gene. The researchers suggest that analyzing the DNA sequences may yield clues to some varieties of diabetes by indicating just what is amiss.

"These experiments with insulin really emphasize the benefits over the risks; they point out the possible practical application of recombinant DNA research," say Goodman and Rutter. □

More signs of a heavy lepton

One of the things enthusiasts for the new unified field theories of particle physics are looking for is evidence of the existence of so-called heavy leptons. Heavy leptons are unstable and more massive relatives of the known leptons—the electron, the muon and the two kinds of neutrino. Their existence (plus the new kinds of neutrino that ought to come along with them) will balance out the scheme of particles required by theories that attempt to unify the descriptions of all the forces that animate particle physics into a single unified theory.

Last week, SCIENCE NEWS reported evidence for possible heavy leptons from an experiment at Fermilab in Illinois. Almost two years ago (SN: 8/2/75, p. 68), Martin Perl of Stanford University reported evidence for a heavy lepton, which he called a U particle, having a mass around 2 billion electron-volts (2 GeV). Later he pointed out that this U might be alternatively explained as one of the newly discovered family of charmed particles. Now a report from the DORIS storage ring facility at the Deutsches Elektronen-Synchrotron at Hamburg strongly supports both the existence of the U particle and its heavy-lepton nature.

Reporting from Germany, the May 12 NEW SCIENTIST points out that the DORIS events occurred at a total energy of 4 GeV. This strongly suggests creation of pairs of U's (one electrically positive, one negative) that then decay into muons or electrons plus neutrinos, which go away unobserved. An important point is that the energy spectrum of the resulting electrons agrees with what theory expects from a heavy-lepton decay. The electron-energy spectrum from the decay of a charmed particle would be something quite different. □

How does vitamin E prevent aging?

Some of the more provocative aging research of recent years concerns vitamin E's ability to retard aging. Denham Harman of the University of Nebraska School of Medicine, one of the nation's leading gerontology researchers, found that vitamin E could extend the lives of rodents by some 30 percent (SN: 3/18/72, p. 188).

These results prompted investigators to ask whether vitamin E could extend human lives as well. But because aging studies in people require years, two other respected gerontology researchers, Lester Packer of the University of California at Berkeley and James R. Smith of the Veterans Administration Hospital in Martinez, Calif., undertook to see whether vitamin E could extend the lives of human lung cells in the test tube. The vitamin did appear to double the usual lifespan of these cells, compared with cultures of control human lung cells. In fact, vitamin-treated cells were still dividing when the experiment ended (SN: 9/28/74, p. 199).

But three years and 19 experiments later, Packer and Smith (who is now with the W. Alton Jones Cell Science Center in Lake Placid, N.Y.) have still not been able to reproduce the results of their first experiment. They report this failure in the April PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. Other investigators, they add, have also had trouble confirming their results, notably Leonard Hayflick, another veteran gerontologist at Stanford University.

Rather than conclude that their initial study was erroneous and the later ones correct, Packer and Smith contend that there might have been something special about the first experiment that made it work. What could the singular, crucial factor be? They suggest that there was probably some chemical difference between the calf serum (fluid component of blood without blood cells and clotting factors) used in the first experiment and that used in subsequent experiments. Such a suggestion is plausible. Calf serum is the nutrient medium of choice for cell culturing, and each lot of serum that biologists buy from biological and chemical companies is nonidentical.

So it is quite possible that the calf serum Packer and Smith used in their first experiment was sufficiently different from that used in subsequent experiments to produce disparate results. Another group of researchers, in fact, has found that dissimilar serum lots resulted in varying test-tube lifespans of human lung cells. They are Edward S. Schneider, Youji Mitsui and Karen Braunschweiger of the Gerontology Research Center in Baltimore (the in-house research program of the recently formed National Institute on Aging).

Assuming that Packer and Smith's suggestion is valid, it would alter the going explanation of how vitamin E retards aging. Past evidence, supplied by Harman, Packer and Smith and some other researchers, suggests that vitamin E increases the lifespan of cells by protecting them from free radicals. Free radicals are highly reactive chemicals. All cells produce them in small amounts in the course of their everyday reactions. Free radicals are also produced by many environmental chemicals. More specifically, vitamin E's effect in countering aging has appeared to be due to its antioxidant abilities—it protects cells from excess oxidation produced by free radicals. Indeed, diet supplementation with some other antioxidants besides vitamin E—Santoquin (a quinoline derivative), 2-MEA (2-mercaptoethylamine) and BHT (butylated hydroxytoluene)—can also increase the lifespan of animals.

But vitamin E does not always increase cellular lifespan while working as an antioxidant, Packer and Smith have found. So vitamin E's antioxidant properties were probably not alone responsible for the increase in cellular lifespan initially observed. However, because the lifespan of control cultures in the original lot of serum was not unusually long, serum alone could not be responsible for the increased lifespan observed among the vitamin-treated cells. Thus Packer and Smith conclude that vitamin E probably extended the lives of cells in the first experiment by interacting with some chemical or chemicals in the batch of calf serum used. In analyzing the little bit of serum they had left from their initial experiment, however, they were not able to identify any compounds that might have been responsible for the effects. □

Cancer immunization: Tempered progress

One of the more innovative approaches to cancer treatment and prevention is to exploit the body's immune system against cancer viruses in hopes that, while viruses have still not been proven causes of human cancers, the techniques might have some value for people. One of the more promising avenues in this area was reported two years ago by a team of German and American scientists—notably Werner Schäfer of the Max Planck Institute for Virus Research in Tübingen, West Germany, Dani P. Bolognesi of the Duke University Medical Center and Fernando de Noronha of Cornell University.

For the first time, viral-induced leukemia was successfully prevented in mammals (mice) by using purified viral antigen as a vaccine and was cured in mammals (mice) by using antiserum to this purified