AAAS MEETING

SCIENCE NEWS of the week

The Sophisticated Sounds of Simians

While the laboratory education of Washoe the chimpanzee, Koko the gorilla and other domesticated creatures is well documented, scientists have assumed that apes and monkeys in the wild do not communicate naturally with any "language." There is communication, to be sure — body movements in combination with various vocalizations are used to convey certain points — but nothing, it was thought, approaching the sophistication of the sign and symbol language assimilated by the famous lab primates.

Now, however, anthropologists at the University of California at Los Angeles (UCLA) report that wild vervet monkeys have "vocal repertoires [that] are far larger than originally believed." Moreover, computer analysis of the monkeys' specific "conversational" sounds reveals them to be surprisingly similar in some ways to human speech, according to the researchers.

"It's like watching humans in conversation," UCLA's Robert Seyfarth told SCIENCE NEWS. The monkeys, he says, have "gone some way along the road to language." He reported the findings last week in Los Angeles at the annual meeting of the American Association for the Advancement of Science (AAAS).

Seyfarth and UCLA colleague Dorothy Cheney say that the "elements of language" they have discovered among vervets are much more subtle and sophisticated than the alarm calls given off by the monkeys when threatened by predators. The researchers had reported previously that vervets sound specific alarms, depending on whether they are threatened by an eagle, snake, leopard or other predator (SN: 11/24/79, p. 357).

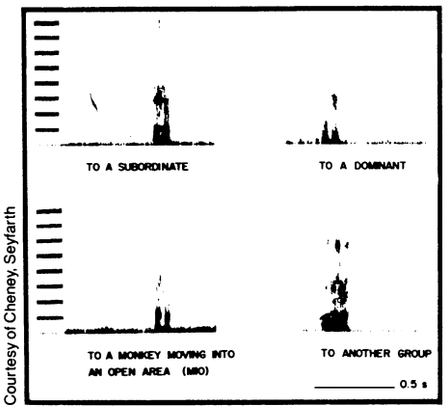
"That's what led us to investigate their grunts," Seyfarth explains. In contrast to alarm calls, which are more like screams, monkey grunts are uttered in all types of nonthreatening situations. And, Seyfarth says, they all seem to sound the same. "Even experienced observers can't tell the difference," he says.

But after years of study in Kenya, Seyfarth and Cheney thought they may have heard tiny differences in grunts made by monkeys in four specific situations: approaching a dominant monkey; approaching a subordinate; acknowledging a leader's call to move onto an open plain from a sheltered area; seeing another group of monkeys approaching.

As they had done in their alarm call studies, the anthropologists hid loudspeakers in the natural environment of six monkey groups in Kenya and played grunts recorded in each of the four situations. They filmed the responses and found that when the listener was addressed by the recorded grunt of a subordinate it looked "sharply" and confidently in the direction of the loudspeaker; when addressed by a dominant monkey, it moved away; when hearing the "open plain" grunt, it looked out toward that area; when hearing the "other group" grunt, it looked out even more strongly.

After viewing the films, Seyfarth brought the corresponding vocal tape recordings back to the UCLA phonetics laboratory for acoustic analysis. He used computer software that conducts "Fourier analysis" of human speech. The process, which analyzes speech waveforms, revealed that the grunt waves from each of the four categories differed in two respects: the placement of the strongest energy and the change of energy peaks over the duration of the grunt. The latter, Seyfarth says, is similar to how human speech distinguishes between vowels.

"There are definitely some elements of language," Seyfarth says. "They are using sound to represent features of their environment." However, he notes, while monkeys appear to have semantics, they lack syntax. "They don't combine two or more [sounds], they don't make sentences and there is no particular order with abstract structure," he says.



Courtesy of Cheney, Seyfarth

Computer analysis of these spectrograms reveals clear differences in grunt waves.

Still, he says, the findings "illustrate that you can't judge the size of vocal repertoire by ear alone." And Seyfarth adds that his research opens up the possibility that other animals, particularly apes, may have natural communication systems in the wild that are far more developed than is now believed. —*J. Greenberg*

AAAS

Dissection of the inebriated brain

The brain is the seat of alcohol's euphoric, intoxicating influence, as well as many of its long-term toxic consequences. But the mechanisms underlying alcohol's effects have been elusive, and no overall brain change has been observed that can explain all the striking effects of low doses of alcohol.

Recently developed methods are allowing scientists to examine alcohol's effects on individual groups of cells in the brains of laboratory animals. These effects are "highly specific to certain nerve pathways," reported Floyd E. Bloom of Scripps Clinic and Research Foundation in La Jolla, Calif., at the AAAS meeting. They comprise the first elements in what scientists expect eventually to add up to a biochemical scenario of intoxication.

The brain area that coordinates nerve cell activity to produce fine motor control, balance and muscle tone is currently spotlighted in such research. This area, the cerebellum, uses "Purkinje" cells with complex, branched structures to gather information from incoming cells and carry output to the rest of the brain.

In one line of research, these cells were examined in mice that had been inbred at the University of Colorado in Boulder for about 25 mouse generations to be extremely sensitive or extremely insensitive to alcohol. The Purkinje cells react differently to alcohol in the sensitive and insensitive mice. In the most sensitive mice, alcohol depresses the activity of the Purkinje cells more markedly and for a longer period than in the least sensitive mice. This differential activity is not seen with depressant drugs other than alcohols, and it is not seen in the hippocampus, the other brain area examined.

Alcohol sensitivity is a property of the cerebellar tissue, says Barry Hoffer of the University of Colorado Alcohol Research Center in Denver. In recent research he has transplanted pieces of cerebellum into the eyes of mice of the donor strain and those of different strains. The alcohol sensitivity of the Purkinje cells always reflects the donor, rather than the recipient, strain. Therefore, the cell itself, rather than its input, determines at least in part the response to alcohol.

But Bloom and his colleagues report that some of the cells that carry signals into the cerebellum also play a role in alcohol's effects. The Scripps group finds that in normal rats an intoxicating dose of alcohol increases the activity of one major source of input to Purkinje cells, the nerve processes called climbing fibers. This is a particularly important input, explains Bloom's co-worker Steven Henriksen, because it preempts the Purkinje cell, interrupting the cell's other activities. Thus, in

the presence of alcohol, the climbing fiber activity overwhelms the cerebellum's normal output.

The climbing fibers arise from cells in the area of the brain stem called the inferior olive complex. This area gathers information from other areas of the brain to send on to the cerebellum. Bloom speculates that ethanol may activate the cells of the inferior olive by reacting chemically with a normal neurotransmitter to create an unnatural, stimulatory compound.

A laboratory study of long-term exposure to ethanol has revealed another specific site of alcohol's action, Bloom reports. He and his colleagues acclimatized rats to alcohol vapors that produce a blood alcohol level associated with intoxication. They used this procedure to avoid a frustrating problem of animal research on alcoholism: Animals generally refuse to drink a solution containing alcohol unless it is the only liquid available and they are quite dehydrated; thus the animals are generally in poor health. In contrast, animals exposed to alcohol vapor remain healthy and continue to gain weight, Bloom says. And they become tolerant to the alcohol, so that after three weeks of exposure, cells of the inferior olive no longer show increased activity. But all is not normal. If the flow of alcohol vapor is interrupted, there is a "profound shift" in activity. Bloom has traced this postwithdrawal change to another set of brain cells, the locus ceruleus.

The locus ceruleus offers a tempting explanation of the clumsiness of an intoxicated person. This brain stem area, which sends processes to the cerebellum as well as to other brain regions, normally shows a response with a fixed latency to novel events in an animal's environment. But in the presence of alcohol, the latency becomes variable. Thus, the nerve cell signal loses its time relationship with the triggering event. This discrepancy may make the difference between catching or missing a dropped plate, for example, Bloom speculates. "This may be the start of a biochemical description of the alcohol effect."

—J.A. Miller

AAAS

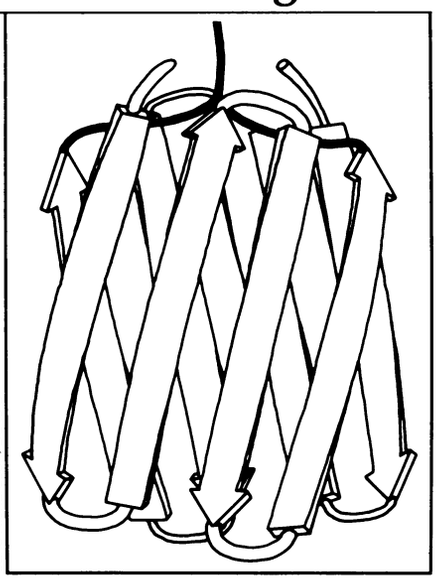
Beyond the limits of protein building

Some children may use their Tinker Toys to model the structures printed on the box, perhaps embellishing them with an extra hub or two. But other children may go beyond the toymakers' imaginations, creating structures all their own and adding pencils, forks, twigs and mousetraps to their edifices.

Protein engineering, an activity still in its early stages, attempts to improve upon nature by remodeling existing enzymes to make them more stable or more active or to change the type of reaction they catalyze. But Bruce Erickson of Rockefeller University in New York and his colleagues are taking a more radical approach. They are constructing from scratch a molecule that does not look like any protein in nature, and they plan to add binding and catalytic sites to its basic framework. Erickson reported at the AAAS meeting the successful chemical synthesis of the framework for a new class of molecules, which he calls betabellins.

"We are opening up a new vista," Erickson says. "With this approach we are not limited to the 20 L-amino acids in natural proteins. We can put in D-amino acids [the mirror images of the L-amino acids found in nature], and we can use any of the 2,000 non-genetically-coded amino acids."

The basic plan for betabellin's structure comes from a natural structure, idealized for chemistry. Several different proteins, such as immunoglobulins and the enzyme superoxide dismutase, have bell-shaped, slatted regions made up of an arrangement of amino acids called beta sheets. Erickson, in collaboration with Jane and David Richardson of Duke University in Durham, N.C., devised a sequence of amino acids that comprises a structure far more regular than any "beta barrels" seen in nature. Betabellin has two identical flat regions. Each of these sheets is made up of three rows of eight amino acids, with a sharp turn between them, and one row of seven amino acids attached to a special synthetic cross-linker.



Jane Richardson, Duke U.

The basic shape of betabellin.

The structure of this small, globular protein includes a variety of special features that appeal to organic chemists. Because its two halves are symmetrical, they can be synthesized simultaneously. Betabellin contains internal sites where it can be snipped into pieces appropriate for analyzing the amino acid sequence. And it contains end sites to which binding sites and active sites can later be added. Betabellin also can be crystallized easily.

Erickson found many constraints on the choice of amino acids in the framework. To form the beta sheets, he had to alternate small hydrophobic (water-shunning) and hydrophilic (water-loving) amino acids. At the tight turns, he used proline and asparagine—originally the L forms, but later he replaced them with the D forms, which fit better in the available space. He included one cysteine in each half of the molecule, and in his most advanced structure he has those amino acids attach to each other in a disulfide bond, thus holding together the two sheetlike sections.

Even the most carefully planned chemical scheme can generate surprises. When Erickson purified the betabellin structure containing the disulfide bond, he obtained two distinct materials with identical amino acid sequences. Erickson and his colleagues are now trying to determine the specific structural difference.

The next step will be to further modify the framework and to add binding and catalytic sites modeled after those found in nature. "We think the present technology is adequate to start evolving proteins with new functional groups at active sites," Erickson concludes. This research, like much of the protein engineering work, is sponsored by the Office of Naval Research.

—J.A. Miller

AAAS

Tracing disease to trace minerals

Trace mineral deficiencies in the diet may play a bigger role in human health than most physicians now realize, according to a series of papers presented last week at the AAAS meeting. Many of these reported links between trace minerals and health problems — such as heart disease and diabetes — are still only suggestive. However, the deficiency levels discussed at the meeting are common in the typical American diet, the researchers warn, and if such subtle deficiencies are definitively found to jeopardize health, they may be affecting millions of people in the United States alone.

For example, while the safe and adequate amount of copper is generally thought to be 2 to 3 milligrams per day, "probably 75 percent of daily diets in the United States contain less than 2 mg of copper," reports Leslie Klevay, acting director of the Agriculture Department's Human Nutrition Research Center in Grand Forks, N.D. Several papers reported data suggesting that copper-deficient diets may increase one's risk of developing a host of health-threatening conditions, including coronary heart disease.

When fed a diet deficient in copper, animals have developed bone fragility, anemia, defects of the connective tissue, arteries and bone, infertility, heart arrhythmias, high cholesterol levels, heart attacks and an inability to control blood sugar levels. Klevay notes that any condi-