The FDA's proposal was in response to a recent National Academy of Sciences report (SN: 9/18/76, p. 180). The NAS panel concluded that fluorocarbon propellants are depleting the ozone layer in the earth's stratosphere, but that more research is needed to accurately determine the extent of depletion, which could range from 2 to 20 percent in the next hundred years. The NAS report recommended no regulatory action be taken before up to two years of further research.

But FDA Commissioner Alexander M. Schmidt said knowledge of the exact rate of ozone depletion won't change the ultimate regulatory situation. "Without remedy, the result [of fluorocarbon use] could be profound adverse impact on our weather and on the incidence of skin cancer in people," Schmidt wrote to the Council on Environmental Quality. "Even a 2 percent ozone depletion from 'unessential' uses of fluorocarbons is undesirable."

The details of the FDA plan will be announced in mid-November. □

# The Kirlian effect: A flash in the pan?

Multicolored flares, sparks, twinkles, bubbles and flashes emanating from the human body—and they can be recorded by the process known as Kirlian photography, one of the most colorful phenomena to be investigated in recent years. The process is simple. In a typical Kirlian device, the object to be photographed is placed between two metal plates. A photograph is obtained when a large electric potential is applied. The explanation for the weird-looking photographs may be just as simple, but numerous exotic explanations have been put forward. Kirlian photography has been linked to almost everything from astral projection and acupuncture to pheromones and extrasensory perception (SN: 9/29/73, p. 202).

More mundane explanations have also been offered but were often overshadowed by the exotic. William Tiller of Stanford University, for instance, suggested that the Kirlian phenomenon is related to corona discharge—a luminous, low-current gaseous discharge occurring in the atmosphere at electric field strengths below the threshold for spark breakdown. John O. Pehek, Harry J. Kyler and David L. Faust, working at Logical Technical Services Corp. in New York, have followed up on this line of research. Their lead article in the Oct. 15 SCIENCE concludes: "Photographic images obtained by the Kirlian technique are principally a record of corona activity during an exposure interval. Most of the variations in the images of the corona of a living subject who is in contact with the photographic film can be accounted for by the presence of moisture on or within the subject's surface." A number of subjects were photographed under a variety of conditions and differences were recorded. But, say the researchers, "By controlling the availability of moisture at the fingertips . . . we have been able to replicate many of the changes in images of corona whose significance has been debated." Not a colorful explanation, but one that may throw water on Kirlian photography. □

# Huntington's disease: An animal model

Huntington's disease is one of the most devastating neurological disorders. The celebrated folksinger Woodie Guthrie was one of its victims. In the early stages of the disease he started walking lopsided, and as it progressed he flew into rages. Gradually he lost his ability to talk, read and walk. He could communicate with his wife and children only by flailing an arm at printed cards marked yes or no. He died in 1967, after 15 years of such agony.

Equally cruel, the disease is inherited and usually does not show up until age 30 or 40. This means that the victim may have reproduced by the time the disease is evident and has already passed on a 50-50 chance of inheriting it to the children. Two of Woodie's children have since developed Huntington's, and three more live in fear of getting it—29-year-old Arlo, a folksinger in his own right and already the father of three, 27-year-old Joady and 26-year-old Nora. The shadow of Huntington's also looms over 5,000 other families in the United States.

At present, no treatment can halt the progression of Huntington's, and even tranquilizers can provide symptomatic relief for only a third of patients. However, three Johns Hopkins researchers—Joseph T. Coyle, Robert Schwarcz and Robert Zacazk—do offer a glimmer of hope to Huntington's victims and potential victims. They have found an animal model for the disease, a model that has not been available before, since the disease only occurs in humans. This finding sets the stage for unlocking the secrets of the disease and finding an effective treatment for it. Without a model, researchers have had to rely on human autopsy material to study the disease, and they have been reluctant to try new drugs on patients that have not been tested in animals first.

A major site of Huntington's disease damage is the basal ganglia of the cerebrum. This is also the major site for Parkinson's disease, which leads to rigidity of muscles, tremor and difficulty moving—effects that are the opposite of the uncontrollable limb movement of Huntington's. Back in the 1960s, researchers identified the neurological basis of Parkinson's. Brainstem nerve axons that innervate the basal ganglia die, and the axons no longer provide the basal ganglia neurons with the neurotransmitter, dopamine. This discovery gave researchers the idea of giving L-Dopa, a precursor of dopamine, to Parkinson's patients in hopes that it would compensate for the dopamine deficiency in their basal ganglia and thereby eradicate the symptoms of the disease. L-Dopa provided dramatic improvement and is today the major treatment for Parkinson's.

Huntington's chorea, however, has long been known to consist of another basal ganglia abnormality—death of the neurons that comprise the basal ganglia rather than of the brainstem axons that innervate the basal ganglia. Two years ago, E.D. Bird and L.L. Iverson of the University of Cambridge and T.L. Perry and co-workers of the University of British Columbia found that this loss in basal ganglia neurons is accompanied by a dramatic loss in the two neurotransmitters that these neurons make—acetylcholine and gamma aminobutyric acid (GABA). Coyle and his co-workers set out to find a drug that, when injected into the basal ganglia of experimental animals, would produce the same kind of damage seen in the basal ganglia of Huntington's victims—death of the basal ganglia neurons and a decrease in the neurotransmitters they make.

The drug they tried was kainic acid. It has the ability to kill neurons, and receptors for it are limited to neuronal cell bodies and not axons. By injecting it into the basal ganglia of animals, they hoped to kill the neurons comprising the basal ganglia, but not the brainstem nerve axons innervating the basal ganglia, thereby reducing acetylcholine and GABA in the basal ganglia, but not dopamine, present in the basal ganglia courtesy of brainstem axons. Basal ganglia neuronal death and a decrease in acetylcholine and GABA, they anticipated, would then lead to the neuropathological characteristics of Huntington's.

They injected the drug into the basal ganglia of rats. It met their expectations beautifully, triggering neuronal death, a decrease in acetylcholine and GABA but not in dopamine or the behavioral changes resembling Huntington's chorea. "This procedure," they conclude in the Sept. 16 NATURE, "could provide an animal model for the study of the disease."

Now that researchers have a model for Huntington's, Coyle says, they can test different drugs on animals and can use the model to better understand the degeneration underlying the disease. Scientists can also use the model to look for some biological marker of the disease. If such a marker is found, it might then be used to identify Huntington's victims before they reproduce, or even to identify human fetuses with the disease, thereby giving parents the option of aborting a baby with Huntington's. □