sionally hit the earth.

The first suggestion for periodic disturbance of the Oort cloud is that the sun has a dark companion star, variously called Nemesis or Death Star, with an orbital period of 28 million years. At its perihelion passage, Nemesis is close to the Oort cloud and perturbs comets. Smoluchowski points out that for a period of 28 million years, its orbit would have to be so elongated and its aphelion so far from the sun that its attraction to the rest of the galaxy would equal its attraction to the sun. Thus each aphelion passage would so perturb Nemesis that after a billion years it might fly off. "We have never seen this star," Smoluchowski says. "Where it came from and how stable it is bothers me."

Both the second suggestion and Smoluchowski's own depend on the periodicity of the solar system's passage through the midplane of our galaxy. The collective gravitational field of the galaxy is strongest in the middle plane of the galactic disk. It tends to attract everything toward the midplane, but stars, as they reach the midplane, are carried through by momentum. Then they are attracted back, and momentum carries them through again. This results in a periodic, pendular motion, "like sewing," Smoluchowski says. Or one might call it a galactic yo-yo. The sun passes through the midplane about every 33 million years.

One suggestion is that passage of the solar system through the clouds of dust and molecules that lie thickly in the midplane knocks comets loose from the Oort cloud. Smoluchowski agrees that the clouds could do this, but he points out that the clouds do not lie flat in the midplane, but in a "fluffy distribution" above and below. The sun never really leaves the region where they lie, so they might trigger comets almost any time.

Smoluchowski would use instead simply the gravitational field in the midplane. This peaks in a relatively well-defined location. Its pull would be enough, he calculates, to give objects in the Oort cloud a kick equal to 30 to 40 meters per second in velocity, about their velocity in the Oort cloud orbit. That's a severe perturbation, and it could knock them out. However, they could go around the Oort orbit a couple of times before coming loose. Thus, this scheme provides the basic periodicity with a leeway that could be several million years depending on when the perturbed comets come loose from the Oort cloud. Also, there is no guarantee that any such comets will hit the earth, so the occasional omission of an extinction is provided for.

Where are we now? Smoluchowski says he consulted Gerard de Vaucouleurs, also of the University of Texas at Austin, "the granddaddy of these things." De Vaucouleurs told him that the solar system is moving toward the constellation Virgo, that is, on the northward swing of the pendulum. But exactly how far the solar system is from the midplane is not known. However, it seems likely that we passed the midplane about 11 million years ago. There was no extinction then, and Smoluchowski concedes this may be a flaw in the theory, but given the uncertainties as to when perturbed comets come loose, we may be still waiting for it.

—D. E. Thomsen

Oral acyclovir puts genital herpes on hold

In its most dramatic success to date, daily doses of the antiviral drug acyclovir have cut the rate of recurrent genital herpes, or blocked flare-ups completely, for up to four months in patients suffering from the painful, sexually transmitted disease. The findings, reported in two papers in the June 14 New England Journal of Medicine, are the first to show that oral acyclovir can suppress the disease for long periods in otherwise healthy people, says Stephen E. Straus, co-author of one of the papers. "We have had nothing in the past, nothing at all, that has been able to do what we accomplished in this study," he says.

Speaking at a news conference held at the National Institutes of Health, NIH's Straus emphasized that "this is not a cure," and added that the data suggest "there is no long-term benefit of this treatment" once it's discontinued. In both studies, on the average, patients had recurrences less than a month after they stopped taking the drug.

Researchers at the Centers for Disease Control estimate that there are between 5 million and 20 million cases of genital herpes in the United States, and at least 300,000 new cases each year. When the virus is active, it can cause sores on and near the genitals. But between outbreaks it retreats up sensory nerves near the blisters and sits dormant in the nerve ganglia. Acyclovir is powerless to eliminate the latent virus, which explains why the drug can suppress but not cure herpes.

Straus and his colleagues at NIH and Burroughs Wellcome Company in Research Triangle Park, N.C., studied the drug's effect on adults with unusually frequent recurrences—12 to 16 episodes a year instead of the three or four that most infected people have. Of the 32 patients who completed the double-blind trial, which lasted 125 days or until a flare-up occurred, half took capsules five times a day for the first five days and then three times a day thereafter, and half took a placebo. All 16 in the placebo group suffered recurrences during this time while only one fourth of those on acyclovir had recurrences. Immediately following the recurrences, these patients began a second round of therapy, all of them receiving acyclovir. Only two of them had flare-ups during this phase.

The second report, by John M. Douglas and his colleagues at the University of Washington and the Children's Orthopedic Hospital in Seattle and the University of California at San Diego, carried equally striking results. In this double-blind study, which also involved people with frequently recurring herpes, 96 patients were given acyclovir capsules either twice or five times a day, and 47 received a placebo. During the four-month experiment, 94 percent of those receiving placebo had outbreaks while less than one third of those on acyclovir had recurrences.

Both studies show that in those cases in which acyclovir failed to prevent recurrences it did stave off flare-ups for an average of nearly 120 days while patients on placebo usually had their first outbreak in less than a month after "therapy" began. When blisters did appear despite the acyclovir treatment, "the recurrences ... [were] very mild, even hard to document in some cases," Straus told Science News.

Oral acyclovir isn't available for general use in the United States, but the Burroughs Wellcome Company expects the Food and Drug Administration's decision on their application for prescription sales "within the next couple of months," says a company spokesman.

Even if the drug's oral form is approved, many researchers who work with the drug feel that too little is known about its long-term effects to justify its indiscriminate use. "Acyclovir is a fantastic anti-herpes agent, but we have to be very prudent in our use of the compound," says Rein Saral of Johns Hopkins University in Baltimore, who leads research on acyclovir therapy for patients with weakened immune systems. "I don't think we have any idea what the drug will do in [an otherwise] normal population over an extended period of time," he says. Straus says that further safety studies are needed, but observes that "the short-term data seem to show a remarkable lack of any side effects."

Concern that acyclovir therapy might encourage resistant strains by blocking more sensitive viruses prompted both groups to screen viruses collected from their patients. Sandra Nusinhoff Lehrman, who collaborates with Straus, says that of 1,500 isolates, fewer than 50 had "decreased sensitivity" to acyclovir, a ratio that suggests less of a resistance problem than is found in some difficult-to-treat bacteria.

—G. Morse