

Puzzling pulses from a star cluster's core

Each discovery of a spinning neutron star, or pulsar, seems to bring with it new mysteries. The latest puzzling addition to the pulsar catalog is PSR 2127+11, located near the center of the globular cluster M15 in the constellation Pegasus. This recently discovered pulsar has no nearby companion star, meaning the pulsar ought to be slowing down. Instead, measurements made by Alexander Wolszczan of the Arecibo Observatory in Puerto Rico and his colleagues reveal that the pulsar's rate of spin appears to be increasing.

Normally, astronomers would attribute such an increasing rate to the transfer of matter from a companion star to the pulsar. In the absence of such a donor, the increasing rate "is probably the result of the pulsar being bodily accelerated in our direction by the gravitational field of the collapsed core of M15," Wolszczan and his colleagues report in the Feb. 9 NATURE. The motion of the pulsar within the globular cluster apparently overwhelms the slowing caused by "magnetic braking" as the spinning neutron star radiates energy in the form of radio waves.

Globular clusters, found near the fringes of our own Milky Way galaxy, consist of dense aggregations of old stars. M15, about 30,000 light-years away, is particularly dense. In such an environment, the pulsar could conceivably be orbiting the cluster's massive collapsed core or under the gravitational influence of stellar neighbors. However, the measured acceleration suggests that either the core contains substantially more mass than astronomers believe or the pulsar is improbably close to its neighbors.

Precise measurements of any changes in the timing of pulses over the next few years should help clarify the situation. "If the pulsar is influenced by nearby stars rather than the central gravitational potential, then in about two years we should see the effect," Wolszczan says.

The pulsar, spinning on its axis once every 110 milliseconds, also has an unexpectedly long period. "Initially, everybody thought that in globular clusters we would find predominantly rapidly spinning, millisecond pulsars," Wolszczan says. "This pulsar may be incredibly old. Even though it started out as a millisecond pulsar, it had enough time to slow down to its presently observed period."

Another possibility is that the pulsar was originally a member of a binary system, gradually increasing its spin by gathering matter from its partner. But an encounter with a third star could have removed the companion, interrupting the pulsar's cosmic meal and leaving it with a relatively long period. — *J. Peterson*

Boosting memory in the blink of an eye

Neuroscientists studying learning in rabbits have identified a drug that, with further testing, may yield a memory-enhancing treatment for humans afflicted with brain disorders such as Alzheimer's disease.

Rabbits shed their age-related difficulties in learning a laboratory task when injected with nimodipine, a drug that blocks the action of calcium in the brain, report Richard A. Deyo and his colleagues at Northwestern University Medical School in Chicago in the Feb. 10 SCIENCE.

Nimodipine is used to improve blood flow in the brains of elderly stroke patients. Of particular interest to the Northwestern researchers, memory problems created by a stroke often abate with nimodipine administration.

To study this effect more closely, they conducted a simple experiment on 36 rabbits. The sample included both young (about 3 months) and aging (about 3 years) adults; half received infusions of an inactive substance and the rest got nimodipine. Most of the rabbits heard a tone, followed by a puff of air aimed at one eye, causing them to blink. They received 80 such trials per day for up to 15 days, or until they learned to blink in response to the tone. Researchers randomly presented some

of the nimodipine-treated rabbits with the tone and the air puffs.

Aging rabbits given the inactive substance required an average of 1,000 trials to learn the task, twice as many as their younger counterparts. Not only did both age groups markedly improve on nimodipine, but aging rabbits learned the task over an average of about 360 trials — virtually the same rate as the younger animals. Randomly conditioned nimodipine rabbits, young or old, did not learn to blink in response to the tone.

In human experiments, substantial learning deficits in the same conditioned eye-blink response begin to appear among people 50 years of age and older with no evidence of brain disease, Deyo says.

He and his co-workers plan to study the eye-blink response in Alzheimer's patients before and after nimodipine treatment.

Nimodipine may somehow improve learning by inhibiting calcium flow in the smooth muscle of the blood vessels feeding the brain, Deyo says. He suspects, however, that nimodipine and several similar drugs block calcium transmission in the hippocampus, a brain structure crucial to memory.

— *B. Bower*

Docking site decoy, antibody fragment wed

Researchers this week reported engineering a new class of hybrid molecules they say may significantly improve a promising, experimental AIDS therapy. The antibody-like molecules, dubbed immunoadhesins, not only sop up circulating AIDS viruses but also contain protein fragments with the potential to trigger an immune response against the virus, HIV.

The approach adds an offensive component to an essentially passive form of AIDS protection now under investigation by the National Cancer Institute in Bethesda, Md. In those trials, researchers inject patients with a soluble variety of CD4, the "docking site" molecule on the surface of cells to which HIV normally binds. Preliminary results suggest soluble CD4 can slow or prevent infection by saturating the CD4 binding sites of circulating HIV before the viruses can attach to and infect immune system cells (SN: 8/20/88, p. 124).

Daniel J. Capon of Genentech, Inc., a South San Francisco biotechnology company, and his colleagues sought to add a one-two punch to soluble CD4. They engineered the genetic machinery inside cultured human kidney cells to mass produce a new product — a CD4 molecule fused with a string of amino acids com-

mon to many antibody molecules. In intact antibodies, that amino acid portion — called the Fc fragment — normally mediates two powerful immune responses: It helps killer white blood cells recognize and ingest antibody-bound foreign invaders and it triggers a process known as complement activation that destroys those invaders by chemical dissolution. The researchers tested immunoadhesins' ability to protect cultured cells exposed to HIV. They report in the Feb. 9 NATURE that the Fc fragment, when linked to CD4, loses its ability to activate complement. But the novel molecule appears to retain its ability to rally immune cells against sopped-up HIV.

The researchers say further tinkering may produce an "optimal molecule" that passively attracts and more actively attacks HIV. Significantly, they add, animal experiments indicate immunoadhesins will remain in the human body nearly 200 times longer than run-of-the-mill CD4, which can disappear from circulation within a few hours. Moreover, the molecule's structure suggests it can cross the placenta from mother to fetus, suggesting immunoadhesins may prove useful in preventing perinatally acquired HIV infection. — *R. Weiss*