The satellite has been sought unsuccessfully in the last three NASA budget cycles, and the agency now ranks it behind such programs as the Venus Orbital Imaging Radar, a Saturn orbiter and double atmospheric probe, the beginnings of a late-1980s Mars mission and a comet program.

On the Soviet side, however, the picture looks different. Surkov told SCIENCE NEWS that a polar-orbiting lunar satellite *is* in the works, and that its mission is set to take place "within five years." □

Cosmonauts return, astronauts named

After a record-setting 96 days in orbit, Soviet cosmonauts Yuri Romanenko and Georgi Grechko left the Salyut 6 space station and returned to earth aboard the Soyuz 27 spacecraft on March 15. During the last week of their stay aloft, they followed an expanded exercise routine, intended to minimize the effects of the return to earth-normal gravity after the long period of weightlessness. Even so, back on the ground the cosmonauts found problems in otherwise easy tasks, such as turning a radio dial or lifting a cup of tea. Both crewmen have reportedly tried to "swim" out of bed in the morning, as was their custom in orbit. Soviet sources, however, have cited no serious readjustment problems.

The day after the Soviet spacemen landed, the U.S. space agency announced the selection of its first four two-man astronaut crews for the space shuttle, which is to begin orbital flights in spring of 1979. The first flight will be commanded by John W. Young, veteran of Gemini 3 and 10, Apollo 10 and the moon-landing Apollo 16. The pilot will be "rookie" Robert L. Crippen, who thus becomes the first of the astronauts who transferred over from the Air Force's canceled Manned Orbiting Laboratory program to get a prime-crew berth. The other selected crews have not been assigned specific shuttle flights, but one of them will fly the mission to raise the orbit of Skylab if it is decided to undertake the task. The crews are: Joe H. Engle and Richard H. Truly, who flew together in the shuttle's approach-and-landing tests; Fred W. Haise (lunar module pilot for Apollo 13) and Jack R. Lousma (pilot of the second Skylab crew); and Vance D. Brand (command module pilot for the Apollo-Soyuz mission) and Charles G. Fullerton (who flew shuttle tests with Haise).

The shuttle orbiter itself was transferred atop its 747 jet carrier last week from the West Coast to begin a series of tests at the NASA Marshall Space Flight Center in Alabama. Meanwhile, according to AVIATION WEEK, the Soviet Union is developing a reusable space shuttle of its own, a delta-shaped craft that has already been drop-tested from a Soviet Tupolev Tu-95 bomber. □

Strep vaccine for unborn children

A number of viruses and bacteria are known to threaten the health of the human fetus, either in the womb or as it makes its way through its mother's birth canal (SN: 4/12/75, p. 242). One of the villains is group B streptococcus. Although the danger of the bacterium to unborn children has been known for about 30 years, its prevalence and devastating effects have become more evident during the 1970s.

Group B strep appears to be transmitted to a woman's vagina during intercourse. She will experience no symptoms as a result of this infection. However, if the infection is present as a child passes through her vagina at birth, the child can also become infected. As a result of infection, the child may die shortly after birth or go on to develop blindness, deafness, spinal meningitis, mental retardation or epilepsy. Approximately 15,000 newborns in the United States suffer from group B strep infections each year.

It is now reported that a vaccine to protect unborn children against group B strep and its ravages is being developed by Carol Baker, assistant professor of pediatrics and microbiology at Baylor College of Medicine, Morven Edwards, a postgraduate fellow in pediatrics at Baylor, and Dennis Kasper of Harvard University. A report on their preliminary testing of the vaccine will appear in the April JOURNAL OF CLINICAL INVESTIGATION. □

Multiple sclerosis: A closer look

Multiple sclerosis (MS) is a slow, implacable killer. The central nervous system is only injured in patches as the disease sporadically destroys myelin, a fatty sheath insulating the nerve fibers. Scar tissue replaces the missing myelin and the flow of nerve impulses is interrupted or distorted. When this occurs, the resultant symptoms can include blindness, paralysis, numbness and loss of coordination.

MS seems an incredibly complicated disease, and scientists have yet to learn its cause, cure or treatment. Discoveries during the last few years suggest that MS results from a viral infection that precipitates an autoimmune disease in which the body's own immune defenses attack the myelin sheathing. But the disease is more complicated than that. Influences of genetics, environment and geography also figure into the risk formula.

A symposium at the recent meeting of the American Society for Neurochemistry focused on advances in the understanding of MS. Cedric S. Raine of the Albert Einstein College of Medicine in New York reported that the rare occurrence of a death from acute multiple sclerosis has permitted a direct look at myelin destruction as it happens. In most cases death occurs only after a decade or so of the disease. The mechanisms that led to the cumulative damage of the tissue are usually no longer working.

Raine reported that the affected brain areas of a woman who died two weeks after the disease became apparent were inflamed. On the periphery of the lesions was evidence of attack of the myelin by antibodies and by the scavenger cells of the immune system, the macrophages. This finding supports the hypothesis that an autoimmune disease is a major component of MS. It also suggests that experimental allergic encephalitis (EAE) may be a good animal model of MS.

Studies on MS have been hindered by the lack of a suitable animal model that mimics the pathology of MS in humans. EAE is an autoimmune disease that quickly leads to death. It produces inflammation around blood vessels and much less destruction of myelin than seen in tissues from chronic MS patients. Raine's findings suggest that the early stages of MS may be like EAE.

Shirley E. Podulso of Johns Hopkins University School of Medicine in Baltimore has isolated the cells of the central nervous system that make myelin, the oligodendroglia. Both oligodendroglia and myelin disappear in MS lesions, and oligodendroglia are proposed to be the target cells in the disease. Podulso reported that the isolated cells from rats make whorls of myelin in culture, and may be capable of remyelinization. She is now studying the largely unknown properties of these cells and trying to learn exactly how myelin is made.

Progress is rapidly being made in understanding the components of MS, and the hope is that a breakthrough in one of the smaller areas will provide ways to halt the spread of the disease and, perhaps, alleviate some of its symptoms. □

Drug effects: Longer lasting than thought

Compared with what is known about the immediate dangers of drug use and abuse, data on the possible long-term effects of substance use—even with narcotics such as heroin—are sorely lacking. There have been indications of permanent or slowly reversible neuropsychological impairment among polydrug (multiple) users: Researchers at the University of California at San Diego observed two years ago that a substantial number of multiple drug takers showed such deficits, including brain wave abnormalities, for as long as five months after beginning treatment. But few systematic studies have been able to document these preliminary findings.

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... Drug effects

Now, a nationwide collaborative study, headed by the San Diego group, reports that long-term and perhaps irreversible organic changes from polydrug use appear to be a concrete danger. The researchers report in the February AMERICAN JOURNAL OF PSYCHIATRY that they examined 151 multiple drug users (some for up to 10 years) and compared them with control populations of other psychiatric patients and nonpatients. The drugs involved included alcohol, depressants, stimulants, heroin and other opiates, cocaine, marijuana and hashish. Patients were rated as either neurologically impaired or unimpaired.

Initially, 37 percent of the polydrug users tested as impaired. After three months of treatment and abstinence or sharply reduced drug intake, 34 percent still showed impairment, reports the research team headed by Igor Grant and Lewis L. Judd. The control psychiatric population's level of impairment also remained essentially constant, but other measures indicated that unlike the drug users, the control group's organic problems were not drug induced. The nonhospitalized group dropped from 8 percent to 4 percent in impairment level after three months.

"Although there is some evidence that such impairment is reversible" among the affected polydrug users, say the researchers, "the condition appears to be of at least intermediate duration and may be long lasting."

Drug types that appear most responsible for undiminished difficulties appear to be central nervous system depressants, including barbiturates, non-barbiturate hypnotics and minor tranquilizers, and opiates. "Specifically, both extensive lifetime and intensive short-term use of CNS depressants and opiates may represent the most dangerous conditions," say the investigators. Particularly surprising, they say, is the apparent relationship between heavy opiate use and neuropsychological impairment. The researchers believe this is the first such link ever reported.

Other high-risk factors for organic impairment identified in the study are increasing age, poorer education, certain historical medical factors and a diagnosis of schizophrenia (about half such patients in the study exhibited neuropsychological deficit). But the most significant findings deal with the correlation between polydrug use and long-term impairment.

"Sedatives and opiates might produce more long-term toxicity than has previously been suspected," the researchers suggest. "If this is so, we need to rethink practices that have led to exceedingly widespread use of sedatives and minor tranquilizers." They also call for a reexamination of "the potential long-term risks of prescribing [opiates] for years, as can occur in rehabilitation programs using the longer-acting narcotics as antagonists." □

Self-styling and the thymus

"Know thyself," counseled Plutarch. His maxim turns out to be a cardinal rule of nature, the cornerstone of immunology. The immune system of mammals is so fine-tuned that it can distinguish a native body component, the self, from a foreign invader, the non-self, when the differences are seemingly infinitesimally small.

How does the body so successfully sort out viruses ensconced in its cells or distinguish between its own berserk cancerous cells and the normal cell cadre? How does it reject skin grafts and tissue transplants? Researchers at the Scripps Clinic and Research Foundation (SCRF) in La Jolla, Calif., and at the Southwestern Medical School (SMS) in Dallas, Tex., have found that the thymus, a pyramid-shaped organ located beneath the breastbone, teaches the immune system how to be self-aware.

More than 15 years ago scientists found that if you remove a mouse's thymus, the mouse loses its cellular immunity. It can no longer reject skin grafts, fight off viruses or destroy tumors. Its bone marrow stem cells no longer mature into killer T lymphocytes that recognize and kill abnormal cells in the body. (T is for thymus-derived.) T lymphocytes are the chief perpetrators of the cellular immune response. Rolf M. Zinkernagel of SCRF has investigated the connection between the thymus and the T lymphocytes.

When a patch of skin or organ is transplanted from one person to another, the graft usually doesn't survive. The killer T cells, recognizing molecules on the grafted cells as foreign, attack and kill the graft. These molecules, histocompatibility antigens, or H antigens, are somewhat like cellular fingerprints — they differ from individual to individual.

Although there are many H antigens — 30 have been detected in the mouse — some are more important than others in mobilizing the killer T cells. In mice one set, the H-2 system, is by far the greatest stimulus signaling the T cells to attack. The researchers have discovered that the bone marrow stem cells must learn from the thymus what their H antigens are before they can become able T cells.

Another wrinkle must also be considered. The body rejects organs and tissue transplants by recognizing foreign histocompatibility antigens. But virus-infected cells or cancerous cells don't have foreign H antigens, they have the afflicted individual's own antigens. So how does the body recognize deranged cells?

In 1974, Zinkernagel and Peter Doherty discovered that killer T lymphocytes could not destroy cells infected with a virus unless the lymphocyte and the cells had at least one H-2 antigen in common.

The researchers infected inbred mice with viruses. After a time they removed the T lymphocytes from the immunized mice and tested *in vitro* what type of infected cells the T cells would attack and kill. They readily killed infected cells from mice of the same inbred strain, but not those of another strain. The researchers concluded that the T cells not only had to recognize the non-self, the antigen to which they were immunized, but the self, their own H antigens, as well.

Now, in reports in the Jan. 19 NATURE and the March JOURNAL OF EXPERIMENTAL MEDICINE, Zinkernagel, Gerry Callahan, Alana Althage and Sue Cooper of SCRF, along with Jan Klein and Wayne Streilein of SMS and Gunther Dennert of the Salk Institute in La Jolla, have discovered more about this programming. They present compelling evidence that T lymphocytes are immunologically incompetent without a thymus because they can't recognize the self.

The researchers irradiated mice with doses large enough to destroy T lymphocytes and bone marrow cells, but not so large as to destroy the thymus gland. They then reconstituted the immune system of the irradiated (A-strain) mice. The mice were injected with stem cells from mice that were the offspring of an A-strain parent and a parent from another (B) strain. Thus, the transferred stem cells have both A- and B-strain histocompatibility antigens on their surfaces.

After infecting the reconstituted A-strain mice with viruses, the investigators removed their "AB" lymphocytes and tested them against virus-infected cells from both A- and B-strain mice. The AB-genetically equipped lymphocytes only killed the cells from the A-strain mice. Virus-infected cells from the B strain were unharmed. Nor did the T lymphocytes act against normal, non virus-infected cells from either strain. Apparently, the lymphocytes couldn't recognize the B part of themselves because the resident A-strain thymus couldn't program them to do so.

Other experiments are even more conclusive. The researchers irradiated AB-strain mice to kill their native lymphocytes and then removed their thymuses. Next, they transplanted A-strain thymuses into the AB mice, and reconstituted their immune systems with immature lymphocytes from other AB mice. Thus, these mice were AB, except their thymuses.

The mice were infected with virus. After the virus-sensitized T lymphocytes had time to mature, they were removed and tested against virus-infected cells. The lymphocytes only attacked the A-strain infected cells, the genetic type of the transplanted thymuses. Since everything about the mice was genetically AB except the thymuses, this seems conclusive evidence that the thymus determines, and in this case limits, the T lymphocytes' ability to recognize self H antigens. □