Virology: lectures, conferences, workshops.

[Madison, Wisconsin]: [s.n.], [s.d.]

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research news

From the University of Wisconsin-Madison / News Service, Bascom Hall, 500 Lincoln Drive, Madison 53706 / Telephone: 608/262-3571

Release: Immediately 07/27/84

CONTACT: David W. Kingsbury (901) 522-0410

By TERRY DEVITT
University News Service

TECHNOLOGY AVAILABLE TO ERADICATE MUMPS AND MEASLES

MADISON--One of the most remarkable of scientific achievements has been the eradication of smallpox.

The highly contagious and frequently fatal smallpox virus was tracked down, isolated and eliminated from its last hiding places in remote corners of the world several years ago. Now, according to one prominent researcher, mumps and measles may one day join the list of extinct viruses harmful to humans.

Addressing the annual meeting of the American Society for Virology at University of Wisconsin-Madison, David W. Kingsbury, a researcher at St. Jude Children's Research Hospital in Memphis, Tenn., said the technology is now available to exterminate the viruses that cause measles and mumps.

However, Kingsbury said the elimination of these diseases is not likely soon. Many developing countries, struggling to meet the everyday needs of their people, do not have the resources to mount extensive vaccination programs for diseases that are rarely life-threatening, he said.

"In industrialized nations instances of these diseases have fallen to negligible levels," said Kingsbury, "but the vaccines are relatively costly and developing countries cannot give them a high priority."

Kingsbury is an expert on a group of related viruses that cause measles,

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mumps and parainfluenzas. Parainfluenzas are respiratory ailments such as croup, acute bronchitis and pneumonia.

The nature of immune responses prompted by measles and mumps have enabled scientists to produce effective vaccines. However, despite this success the related parainfluenzas have proven more difficult to control, Kingsbury said.

"In principle we can get rid of mumps and measles because we have effective vaccines," Kingsbury said. "But vaccines haven't worked with these other diseases. They don't stimulate the kind of lifelong immunity that measle and mumps vaccines do."

Vaccines, by exposing people to a disarmed disease virus, spur the body's immune system to produce antibodies, proteins that confer immunity to the disease virus used in the vaccine.

"In terms of parainfluenzas, our approaches to vaccine production have not met with success," said Kingsbury. "We're going to have to try different approaches."

According to Kingsbury, more research into antibiotics may provide researchers with new strategies to control parainfluenza viruses.

"As we learn more about these viruses and the ways they work, the better our chances to pull something off," Kingsbury said. "We base our design of antibiotics on understanding the organisms we want to shoot at. Parainfluenza viruses have a lot in common and it's possible an antibiotic directed against a key process in one organism would be effective against the whole group."

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Release: Immediately 07/27/84

CONTACT: Wayne H. Thompson (608) 262-3937

By TERRY DEVITT
University News Service

OLD TIRES CARRY DEADLY HITCHHIKERS

MADISON--Although somewhat of a homebody, a woodland mosquito known to carry a strain of encephalitis seems to be doing quite a bit of traveling these days.

Known to scientists as Aedes triseriatus, the mosquito is found throughout southern Wisconsin and rarely travels far from its woodland home. It is of particular concern in southwestern Wisconsin's driftless area, where it harbors a form of viral encephalitis known as La Crosse Virus.

But according to scientists at the University of Wisconsin-Madison, the mosquito has a penchant for hitching rides in discarded tires and spreading the disease to parts of the state where it is not usually found.

The spread of the virus concerns scientists and health officials because the disease causes an inflammation of the brain and is sometimes fatal. It is especially threatening to children under 15.

Addressing scientists at the annual meeting of the American Society for Virology, Wayne Thompson, a UW-Madison professor of preventive medicine, said he was able to isolate La Crosse virus in mosquitoes from a Washington County woodlot known as Nob Hill. The area, 20 miles northwest of Milwaukee, is well outside the region where the virus is normally found.

"The Nob Hill isolate is the only one that we've obtained from the eastern part of Wisconsin," said Thompson. "All the other virus activity we've seen

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has been in southwestern Wisconsin."

Although cases of La Crosse encephalitis have turned up in eastern Wisconsin before, Thompson said those instances are usually associated with exposure to mosquitoes in the southwestern portion of the state. However, in the Nob Hill area, Thompson's team turned up some old tires which he suspects might have been responsible for introducing infected mosquitoes into the woodlot.

Thompson said the mosquito normally lays its eggs in water-filled tree holes. However, old tires also serve as excellent breeding grounds.

"We found about 30 tires in this 30-acre woodlot," Thompson said. "Some of them had a history of being imported from an area near La Crosse and that could explain how the virus got to this Washington County woodlot."

Once introduced into the woodlot, the mosquitoes likely transfer the virus to their uninfected cousins already living there, Thompson said.

Isolating the virus in the Nob Hill area aroused Thompson's concern about the spread of the virus. However, he said it is not likely the La Crosse virus will spread to other woodlots around Nob Hill because wooded areas in that region are separated by open areas which serve as barriers to the mosquitoes.

"This particular mosquito doesn't fly as far as some other mosquitoes and it's very intolerant of sunlight," said Thompson. "It's not likely to spread quickly to other woodlots in the area. However, it could eventually spread to other woodlots by way of fencerows."

According to Thompson, about 20 or 30 cases of La Crosse virus are reported in Wisconsin each year. But he added that the incidence of the disease is probably higher because some cases go unreported.

The virus also is a problem in other parts of the country. Thompson, who first identified the virus in 1964, said the mosquito and the virus it harbors can be found in Iowa, Minnesota, Illinois, Ohio and New York.

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CONTACT: Paul Shapshak (213) 828-7726/824-4307 after Wed., Aug. 1

By SUE REYNARD
UIR Science Writer

NEW RESEARCH METHOD MAY PINPOINT CAUSE OF MS, SCIENTIST SAYS

MADISON--A new research method is helping scientists identify viruses associated with neurological diseases and may help pinpoint the cause of multiple sclerosis, according to Paul Shapshak of the University of California-Los Angeles and the Wadsworth VA Medical Center in Los Angeles.

"Almost any virus may cause neurological disease if it gets into the brain," Shapshak said at the annual meeting of the American Society for Virology at University of Wisconsin-Madison last week (July 22-26). "We want to link specific viruses with specific diseases. Our ultimate goal is to see whether multiple sclerosis is caused by a virus."

Multiple sclerosis (MS) is a disease that destroys the myelin sheath surrounding nerve cells. The process, called demyelination, leads to loss of brain function because nerve messages cannot be transmitted.

"Over 30 years ago, David T. Imagawa of UCLA suggested that MS might be caused by measles virus in the brain," Shapshak said. "But it was not until very recently that a research method was developed for detecting and identifying virus genes in the brain."

The technique, designed by Ashley T. Haase of the University of Minnesota in Minneapolis and Wallace W. Tourtellotte of the Wadsworth Center, relies on the ability of "molecular probes" -- made up of short sequences of genetic

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material -- to bind or "hybridize" only with complementary molecular sequences found in the genetic material of viruses. If a particular virus, living or dead, is present in a brain tissue sample, these molecular probes will be picked up by the virus; otherwise the probes will be washed off when the sample is rinsed. Shapshak said the probes are labeled with distinctive chemical compounds, so researchers can detect their presence and the presence of attached virus material.

Haase said his studies of brain tissue, using molecular probes, revealed that 60 percent of MS-afflicted brains contained measles virus. He added that 30 percent of normal brains also contained the virus.

Using the same technique but a different molecular probe, Shapshak and his colleagues, working with Tourtellotte, have found clear evidence that measles virus particles also occur throughout the cells of brains damaged by a deadly demyelinating disease called SSPE, subacute sclerosing panencephalitis. The probe studies also confirmed that a group of SV-40 type viruses are the cause of another chronic demyelinating disease called PML, progressive multifocal leukoencephalopathy, Shapshak said.

Shapshak's team recently began to search for viruses in tissue from 1,000 frozen brains held at the National Neurological Research Bank at Wadsworth, an institution founded by Tourtellotte and supported by the National Institutes of Health and private donations. Most of the brains in the research bank are diseased and the majority are MS-afflicted brains.

The probe method works well in identifying viruses present in both frozen and formalin-preserved tissue, said Shapshak, and should be applicable to any organ in the body. He believes the probe method will become a common research procedure in the coming decades, and may prove particularly useful in establishing the cause of slow chronic diseases such as MS.

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CONTACT: Enzo Paoletti (518) 474-6389, Bernard Moss (301) 496-9869

By JEAN LANG
UIR Science Editor

SMALLPOX VACCINE TAKES ON NEW DISEASE-FIGHTING CHORES

MADISON--The vaccine that virtually eliminated smallpox from the world is being re-harnessed by genetic engineers to take on new disease-fighting chores.

According to scientists attending the annual meeting of the American Society for Virology at University of Wisconsin-Madison last week (July 22-26), it is now possible to use recombinant DNA techniques to change the common smallpox vaccine into a vaccine that immunizes against several diseases simultaneously.

So far, recombinant vaccines have been made that immunize laboratory animals against herpes simplex, hepatitis B, influenza A, and Plasmodium, the organism that causes malaria.

"The beauty of this system is that is provides a generic approach for engineering all kinds of immunizing genes into vaccines for humans and animals," said Enzo Paoletti of the New York State Department of Health's Laboratory of Immunology.

He added that the recombinant vaccines are simple and inexpensive to make, remain stable for long periods when freeze-dried and are effective with a variety of animals and cell types.

Bernard Moss, of the Laboratory of Viral Diseases with the National Institute of Allergy and Infectious Diseases in Bethesda, Md., suggested that

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a recombinant hepatitis B vaccine should be ready for clinical trials on humans in a couple of years. The cost of each unit of recombinant vaccine should be only pennies, he said, making it an affordable vaccine for Third World countries in which hepatitis B is a serious disease.

The gene recombination has been successful, said Moss, because vaccinia -- the non-virulent pox virus used to generate immunity to smallpox -- is a large virus with an unusually large, easily engineered ring of genetic material. The versatile vaccinia virus also has a wide host range and is able to infect and convey immunity to a variety of animals.

A recombinant vaccine is created, said Moss, by first removing non-essential genes from the vaccinia virus and replacing them with carefully selected non-virulent genes from other disease-causing agents.

The inserted gene from herpes virus, for example, tells the infected cell to make a harmless piece of the herpes virus protein coat. The presence of this foreign protein, called an antigen, stimulates the body's immune system to make antibodies against the herpes coat protein and subsequently confers the body with immunity to infection by an intact herpes virus.

To make room for inserted genes, researchers have commonly deleted the so-called tk region of vaccinia's DNA. Paoletti and his colleagues have been looking for additional non-essential proteins that can be deleted.

"The more regions we can delete from vaccinia DNA, the more room we have to insert other immunizing genes, and the safer the vaccine becomes," said Paoletti.

He explained that vaccinia is already considered a very safe vaccine, but that because it is a live virus, the more limited its function, the better. The recombinant vaccinia, in fact, has proven even safer than normal vaccinia, he said.

Moss added that it is important not to delete portions of vaccinia DNA that enable the virus to infect cells and reproduce itself. It is only when

the vaccine virus actually penetrates and infects a cell that it can multiply and do its work of generating immunity. Moss and the other scientists are still refining their maps of essential and non-essential regions of vaccinia's DNA.

Paoletti's group is also trying to select ideal "promoters" to accompany inserted genes. Promoters are sections of DNA that lie upstream from the gene and control the gene's activity or "expression."

"In a multi-target vaccine," explained Paoletti, "you may want different disease antigens expressed at different levels. Carefully selected promoters provide that control and enhance the vaccine's properties."

He added that recombinant techniques make it possible to insert a variety of promoters from other viruses into vaccinia.

Laboratory tests with a herpes virus that causes cold sores in humans have been encouraging, said Paoletti. Mice immunized with the vaccine and then exposed to herpes virus, which is lethal in mice, all survived. Immunized mice did not do as well when exposed to the genital herpes virus, however.

"Ideally, for total immunity, we want to make recombinant vaccinia that contain the unique antigens of each virus strain," said Paoletti.

Moss and his colleagues have also tested vaccinia recombinants containing genes for antigens of hepatitis B, influenza A and herpes simplex and reported that the vaccines stimulated good immune response in mice, rabbits and chimpanzees.

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research news

From the University of Wisconsin-Madison / News Service, Bascom Hall, 500 Lincoln Drive, Madison 53706 / Telephone: 608/262-3571

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CONTACT: Fred Rapp (717) 534-8253

By SUE REYNARD
UIR Science Writer

SCIENTISTS MUST CHANGE THINKING TO CURE HERPES, EXPERT SAYS

MADISON--A cure for herpes is not yet in sight and won't be until scientists change their thinking about viruses, according to a Pennsylvania State University virologist.

Fred Rapp of PSU's College of Medicine, speaking at a national virology conference held at University of Wisconsin-Madison last week (July 22-26), said to understand and cure herpes and other long-term repetitive infections, scientists must change the way they approach virus research.

Rapp said current knowledge of viruses is based largely on work with disease agents such as smallpox and polio that cause rapid onset of a short-term illness and then leave the body.

But he said many viruses, such as oral and genital herpes, produce diseases that progress slowly and steadily, or disappear for long periods only to re-emerge and cause a repeat infection.

Because current attempts to create vaccines against these viruses are based on the short-term infection model, the vaccines have been largely unsuccessful in humans, said Rapp.

Traditional vaccines, he explained, operate by causing the body's defense system to make antibodies against the virus. However, in chronic infections, like herpes, the viruses periodically become inactive and hide from the body's

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immune system in ways that still are not understood.

Remedies for long-term infections also are impeded by a poor understanding of what causes the latent viruses to become active, as in the recurrent eruptions of herpes. Many investigators simply cite "stress" as the inciting agents, said Rapp, but most recognize the indefinite nature of such explanations.

Rapp's own studies of herpes in cultured cells suggest that changes in temperature may play a role in reactivating the virus. Infection of the body by another virus also prods the herpes gene into activity, he said.

To illustrate the complexity of the herpes problem, Rapp described studies in which a drug called acyclovir reduced the incidence or duration of herpes outbreaks, but did not provide a cure: as soon as patients stopped taking the drug, the outbreaks continued at pretreatment levels. Why the drug can temporarily suppress herpes but not eliminate it is unknown, he said.

Rapp said control of the disease will come eventually, but only when scientists can either prevent the virus from getting into cells, or can destroy the virus' genetic elements without harming the genes of the host cells.

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From: University of Wisconsin-Madison / University News Service, 19 Bascom Hall, 500 Lincoln Drive, Madison, Wisconsin 53706
Telephone: 608/262-3571

7/27/84

NOTE TO EDITORS: Enclosed is a packet of stories that represents coverage of the annual meeting of the American Society for Virology, held this past week (July 22-26) at the University of Wisconsin-Madison. Many important advancements in the field were reported during the meetings, including breakthroughs in developing new vaccines to fight disease and new uses of viruses in the production of drugs by using genetic engineering techniques. These stories were produced by science writers working under the direction of Jean Lang, Science Editor for the UW-Madison and editor of the University-Industry Research Program's magazine, Touchstone. If you have any general questions about the meeting or about the enclosed releases, Lang can be reached at (608) 263-7274.

Linda Weimer, Director
University News Service

uw news

From the University of Wisconsin-Madison / News Service, Bascom Hall, 500 Lincoln Drive, Madison 53706 / Telephone: 608/262-3571

Release: Immediately 8/1/84

MADISON CONFERENCE REVEALS NEW FINDINGS ON INTERFERON

CONTACT: Sidney Grossberg, Medical College of Wisconsin, Milwaukee
(414) 257-8253

MADISON--A new finding that interferon binds to the membrane that surrounds the cell's nucleus promises to open new avenues of research into the ways in which interferon affects the body's cells.

"Until now, there has been no suspicion that interferon molecules were transported into the cell for possible action on the nucleus," according to microbiologist Sidney Grossberg of the Medical College of Wisconsin in Milwaukee. "For more than a decade, we thought interferon bound to the cell surface and somehow triggered reactions within the cell to achieve virus resistance."

The finding was revealed in a report given by Grossberg and colleagues at the American Society for Virology's meeting at University of Wisconsin-Madison.

Grossberg explained that interferon, a family of proteins made by our bodies, plays many roles: it stimulates cells to engulf foreign materials and to kill tumor cells, it stimulates antibody production, and it inhibits the growth of many kinds of cells.

"We don't know how interferon regulates growth and stimulates cells," he said. "But this demonstration -- that interferon not only gets into the cell but also that the nucleus is a target -- may point the way to explorations we never dreamed of undertaking."

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By Jacqueline Kelley, University News Service, (608) 262-8282

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Add 1 -- Interferon research

AIDS AND INTERFERON

CONTACT: Olivia Preble, (202) 295-3491

MADISON--AIDS, the disease that curtails the effectiveness of the body's disease-fighting white blood cells, also may affect the action of interferon, the substance used by cells to combat virus infections.

Olivia Preble, a researcher at Bethesda's Uniformed Services University, has found that the white blood cells of AIDS victims do not respond normally to interferon treatments. She reported on the findings of her research team at the recent meeting of the American Society for Virology held at the University of Wisconsin-Madison.

When a cell is invaded by a virus, it first produces interferon. The chemical doesn't attack the invader but does warn neighboring cells and prevents the viruses' reproduction by keeping them from commandeering the cells' protein-producing machinery.

For this reason, medical professionals have tried using interferon to treat the often-fatal virus infections that accompany AIDS, said Preble. But recent studies by Preble and others have shown that the white blood cells of male, homosexual AIDS victims, in the presence of interferon, did not produce an enzyme crucial to the prevention of viral reproduction.

Preble's continuing research is aimed at determining the reason for interferon's ineffectiveness in AIDS victims. One possibility, said Preble, is that disease-fighting white blood cells are unable to function normally because they themselves are infected by viruses.

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-- By Sue Reynard, UIR Science Writer, (608) 263-2876

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Institutes, Etc.
Virology

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CONTACT: Roland R. Rueckert, (608) 262-6949

MARKEY TRUST AWARDS UW-MADISON $3 MILLION FOR VIRUS STUDIES

MADISON--The University of Wisconsin-Madison's Institute for Molecular Virology has been awarded a grant of $3 million over five years to expand its work in the area of basic virology.

The award, made today by the Lucille P. Markey Charitable Trust of Miami, Fla., will enable the institute to expand its programs of research on virus replication, pathology and control, according to Roland R. Rueckert, chair of the institute.

The Molecular Virology Institute was founded in 1964 by Professors W.W. Beeman and Paul Kaesberg, and was known as the Biophysics Laboratory until it was renamed in 1987. It is a research and training center devoted to the basic understanding of viruses, infectious agents that cause numerous diseases in plants, animals and humans.

The Markey Trust grant, Rueckert said, will enable the institute to add new faculty and staff, develop a center for computer graphics and support, and bolster the recruitment of top graduate students.

"The Markey Trust award is very important from the standpoint of letting us do new things," Rueckert said. "It comes at a very critical time in the fiscal situation. It sets up a new enthusiasm."

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The new computer graphics capabilities, for example, will allow UW-Madison virologists to speed up their work on the development of new drugs and strategies for combating viruses such as those that cause the common cold.

By using a computer to build a picture of a virus and its surface, scientists can see how antibodies and drugs interact with the virus.

"You can get the whole virus on the screen and see where the changes are," Rueckert said. "That guides us in studies of how antibodies, how drugs neutralize viruses."

That ability, Rueckert said, will help scientists design new control measures, including drugs and vaccines against the many kinds of common cold viruses as well as other viruses that affect people, animals and plants.

The grant will also enable the institute to recruit a new faculty member and as many as six post-doctoral researchers.

In addition, the Markey funds will be used to aid in the recruitment of top-flight graduate students to a new UW-Madison doctoral program in virology.

The Lucille P. Markey Charitable Trust was established in 1983 under the provisions of the will of the late Lucille P. Markey. Its assets are devoted exclusively to supporting and encouraging basic medical research.

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-- Terry Devitt, (608) 262-8282