AAAS MEETING

SCIENCE NEWS of the week

Jupiter's Spot of Order in Chaos

For centuries, Jupiter's Great Red Spot has stared enigmatically back at earthbound observers and prompted many theories about its nature and origin. During the last decade, images from the Voyager space probes revealed the spot as an eye of stability in the midst of chaotic fluid flow within one of the wide, colored bands, or zones, that encircle the planet. Now, computer simulations are beginning to provide a better understanding of how and why Jupiter's atmosphere developed one (and only one of this size) spot within one (and only one) zone. These recent calculations show that a disordered, turbulent flow on a rotating body can spontaneously develop partial order in the form of a single, long-lived feature very much like Jupiter's ruddy eye.

"On Jupiter, one sees flow that is very chaotic, but nonetheless one also sees something well organized," says applied mathematician and astronomer Philip S. Marcus of Harvard University. "One sees the Great Red Spot, which has a lot of order and structure and which has been sitting around at least since Galileo looked at it through his telescope about 350 years ago. And that is a long time compared with other time scales on the planet."

Because of the difficulty in recreating Jupiter's rotating atmosphere in the laboratory and because the amount of data on the planet's dynamics is limited, Marcus has relied on numerical simulations using high-speed computers to do his experiments. "Computer simulation and modeling is a logical way to proceed," he says. Marcus presented his results last week at the annual meeting of the American Association for the Advancement of Science, held in New York.

Using a Cray computer and a simplified set of equations for fluid motion on a rotating surface, Marcus calculated and plotted how various sets of initial flow patterns would change over time within a particular zone. He found that regardless of the atmosphere's initial state, after a sufficiently long time, a zone's atmosphere ends up looking like a chaotic, east-west wind moving generally in a direction opposite to that of the planet's spin. Sometimes a large, stable vortex forms within this chaotic flow.

"What I've done is a numerical simulation that shows if one looks at a wide range of initial conditions, one gets either no spot or just one spot," says Marcus. "That's an empirical, numerically observed fact." The spot itself would have a vortexlike, anticyclonic flow.

Whether a spot shows up seems to depend on the distribution of energy and angular momentum within the planet's atmosphere, says Marcus. Near-equatorial zones like those where the Great Red Spot actually resides are more likely to provide the conditions that promote spot formation. There, variations in the Coriolis force, which are greatest at the equator and smallest at the poles, interact most strongly with the flowing fluid. "It's no surprise, then, to see a spot near the equator," says Marcus, "but you might not see one near the poles."

Jet Propulsion Laboratory

This mosaic, assembled from 12 Voyager 1 pictures, shows the red spot amid chaos.

Marcus also found that if a spot forms, it happens rapidly — within a single Jovian year—yet lasts a long time. If several spots somehow manage to organize themselves for a brief time, they rapidly drift and coalesce into one large spot.

"This simulation doesn't include all of the physics that's on Jupiter," says Marcus. "I'm assuming that whatever is causing the zones to remain where they are has a time scale that is much longer than [the time] it takes those spots to come together." This assumption allows Marcus to consider spot formation independently of zone formation. A zone is necessary as a "garden where red spots can grow," says Marcus. If the zone system were disrupted, the spot would disappear. Saturn has no spot, Marcus says, because the planet's zones of east-west winds have a different velocity profile than those on Jupiter.

"You can't see such things on earth because the zone system of the earth is also broken up by the continents," says Marcus. "As we know from experience, cyclonic and anticyclonic storms and other types of weather on earth last much less than 300 years." However, he speculates that large vortexlike features that have been observed within ocean currents such as the Gulf Stream may be analogous to the Great Red Spot. Such features may be further examples of "order in chaos."

—I. Peterson

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Drugs throw nutrition roadblock

Certain drugs commonly prescribed to treat psychosis and depression might interfere with vitamin metabolism, leaving even well-fed patients nutritionally deficient, a New York City endocrinologist told an AAAS symposium last week. Preliminary data from his laboratory also raised the possibility that damage to the heart in patients treated with the powerful anticancer drug adriamycin might be linked in part to a similar misstep in vitamin metabolism, says Richard Rivlin of Memorial Sloan-Kettering Cancer Center.

The findings stem from experiments in animals, Rivlin stresses, and are too preliminary to substantiate a change in the current treatment of human patients. Nonetheless, the scientist says he believes the anti-vitamin effect of certain drugs are of "clinical and physiological significance."

"We as Americans are obsessed with the idea that being well nutritionally means eating a good diet," Rivlin says. "But it isn't enough just to eat a good diet. It's important to consider the drugs, hormones and disease that [affect] metabolism as well."

Much of Rivlin's work has centered on vitamin B_2 or riboflavin, a coenzyme that plays key roles in a variety of processes

throughout the body, including blood formation, fat metabolism and energy production. In order to do its work, riboflavin must be converted to its active forms: flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD). Throw a few chemical roadblocks in the conversion process, and the nutritional value of the vitamin is lost. The anti-psychotic drug chlorpromazine, "remarkably similar in structure to riboflavin," presents just such a roadblock, Rivlin says. When he and co-workers John Pinto, Yee Ping Huang and Nicholas Pelliccione gave rats chlorpromazine in doses comparable to those given patients, the drug inhibited riboflavin conversion to FAD, particularly in the heart.

Tests with the tricyclic anti-depressants imipramine and amitriptyline showed similar results. The common link in the drugs, which are used therapeutically for very different purposes, is structure, Rivlin says. Each is similar enough in shape to configurations that riboflavin sometimes assumes in the body that the drugs bind to cell membrane receptors intended for the vitamin and valuable riboflavin is lost in the urine. Other drugs with similar functions, but different structures, "have absolutely no effect on vitamin metabolism," Rivlin says.

If the results are substantiated in human studies they should spur doctors to identify patients vulnerable to clinically induced malnutrition, Rivlin says. "Just as there are risk factors for developing heart disease, there really ought to be risk factors for developing specific vitamin deficiencies."

Boosting consumption of riboflavin might be one way to counteract the effect, Rivlin says, though he cautions against the common assumption that if small amounts are good, megadoses must be better. A study he recently completed of healthy elderly women all taking daily riboflavin supplements showed that the vitamin was not stored by the body—even tiny increases over daily requirements were excreted.

Rivlin has just begun to look at the effects of adriamycin and other anti-cancer drugs in its class on riboflavin metabolism. A potent weapon against solid tumors, adriamycin is not without problems. In the short run, it alters the heart's rhythmic beating. In the long run it can lead to irreversible cardiac scarring. Most other organs, however, are spared. Rivlin has found that even very low doses of the drug given to rats interrupts the usual conversion of riboflavin to FAD in heart tissue. Riboflavin is known to be especially important to energy production and electron transport in the heart.

While the work is still in its very early stages, the approach might eventually lead to a method of both prevention and treatment of the toxic effects of the drug through dietary supplementation, the scientist told SCIENCE NEWS. — *D. Franklin*

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Breast tumors are prevented in mice

A group of New York researchers reports it has been able to prevent the formation of a specific type of virus-triggered breast tumor in a strain of mice. And while the results appear to have no immediate implications for women, scientists say there may be certain similarities between this animal tumor and the human variety.

"This is a good model," says Vinay Likhite of the Champlain Valley Immunology Foundation in Plattsburgh, N.Y. There is "no proof" that human mammary tumors are caused by a virus, he says, such as the virus endogenous to the C3H mice he studied. But both the mouse and human breast tumors are similar, he says, in their spontaneous onset.

It is known that female C3H mice develop breast cancer spontaneously between 4 and 15 months of age; the tumors are caused by the Bittner virus, which is transmitted by the mother's milk, and males generally are unaffected. Likhite and his colleagues injected 48 1-month-old female mice with testosterone. The researchers reported at the AAAS meeting that none of those mice developed breast cancer, while all 209 of the controls were afflicted with the characteristic tumors by 15 months. Testosterone does *not* affect tumors that have already formed, they report.

"Just how [testosterone] prevents Bittner virus from inducing tumors is under study," say Likhite and co-researchers Claire Klausner and Antonio Rottino, both of the Hodgkin's Disease Research Foundation in New York City. "Also the potential of androgens for preventing human breast cancer, e.g. in women with a history of breast cancer, remains to be determined."

One key element, which Likhite is currently pursuing in followup studies with various drug combinations, would be to achieve the same degree of prevention without causing the accompanying masculinization.

Unless that can be accomplished, Likhite's results so far are not particularly remarkable, says William DeWys, associate director of the National Cancer Institute's prevention program. However, DeWys does concede that although most human breast cancer is not thought to be caused by a virus, it may be similar "epidemiologically" to the C3H model. Moreover, he says that a Columbia University group several years ago discovered that members of the Parsi religious sect in India appeared to pass on the risk of breast tumors through viruslike particles in the mother's milk.

Likhite himself says he doesn't "want to get too excited" about his findings. But if his followup experiments are as successful, he says, "I will agitate for human testing." — *J. Greenberg*

Depression and the lymphocyte link

The wish for healing is half of health, the Roman philosopher Seneca observed in the 1st century. Some researchers are studying the reverse of his observation, probing how depression or similar mental malaise may contribute to ill health.

Links between depression and illness have long been noted. Viral infections, particularly herpes simplex, are more common in depressed people, who also have higher death rates. Moreover, some studies suggest a higher cancer risk, says Steven Schleifer of the Mount Sinai School of Medicine in New York City.

Schleifer recently released data showing that lymphocytes — the main immunocompetent cell — proliferate "significantly" less in severely depressed patients than they normally would when stimulated to reproduce.

"The data indicate that the immune system is altered in hospitalized patients with depressive disorders," says Schleifer. But reasons remain unclear: It could be prompted by behavioral factors or by changes in the hypothalamic-pituitary-thyroid axis, he adds. Schleifer's on-going studies on specific immune responses to bacterial or viral antigens by lymphocytes.

In related but separate research, Steven Maier of the University of Colorado in Boulder recently found "substantially lower" T-cell lymphocyte proliferation and less effective "killer" cell lymphocyte action in rats with the psychological malady called "learned helplessness."

Learned helplessness occurs when animals are faced with inescapable situations, such as the stress of uncontrollable electric shocks. Once taught they cannot escape their fate, they become helpless to take advantage of escape routes later offered in response to stress, Maier says.

"They eat less, drink less, lose weight, develop ulcers. None of these follow if the stress is controllable. So the dimension of control proved more powerful than exposure to the event per se," Maier says. Learned helplessness also causes the physical release of analgesia-inducing opiates, and lymphocytes have opiate receptors, he adds. His current study looks at that possible link. — *A. Rowand*

Gene-splice ruling upheld

Field tests of a genetically engineered bacterium designed to prevent frost damage to crops were once again postponed as the U.S. Court of Appeals on May 25 upheld a District Court decision against the University of California (SN: 5/26/84, p. 325). Unless a further appeal overturns the decision, the injunction remains in effect until the District Court holds a full hearing on the case. □