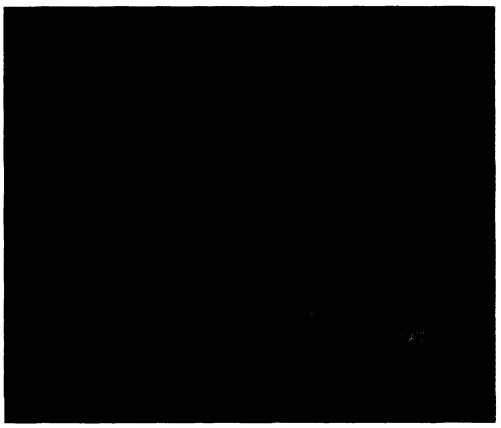
tion" engine can also segregate electrical charges into different parts of a cloud; the reuniting of those charges is lightning. Although Jupiter has no ocean or wet ground, moist convection seems to work much as it does on Earth. "We're using lightning as a beacon to tell us where the convection is" on Jupiter, says planetary meteorologist Andrew Ingersoll of the California Institute of Technology in Pasadena, who co-authored the *Nature* papers with Peter Gierasch and Donald Banfield of Cornell University and their colleagues.



Night lights. Jovian storms (reddish in false color, top) sport powerful lightning strokes (blue, bottom).

CREDIT: GIERASCH *ET AL.*, *NATURE*

By observing a thunderstorm hundreds of kilometers long just west of the Great Red Spot in daylight and in darkness, Gierasch and his colleagues could gauge how much heat thunderstorms are extracting from Jupiter's interior. In daylight, they watched the clouds carried by the air move up and away from the storm, which told them how much warm air the storm was raising. During the night, they measured the lightning associated with the storm. Then they surveyed about half the planet to determine the global abundance of lightning. They calculate that moist convection delivers a large part of the heat that is known to leak out of the interior. The Galileo data "is very good evidence the heat comes up localized in convective systems," says planetary meteorologist Conway Leovy of the University of Washington, Seattle, "just as it does in Earth's" lower atmosphere.

But that is where the terrestrial analogy stops, argue Ingersoll and his colleagues in the second paper. The Great Red Spot, they note, is not the hurricane-like storm that textbooks often describe. Neither it nor the whole hierarchy of smaller ovals on Jupiter draw their energy directly from their own moist convection, as hurricanes do. Instead, they argue, it's a two-step process. If jets don't tear apart the lightning-riddled jovian thunderstorms and take on their energy, the planet's rotation turns the thunderstorms into stable swirls of spinning air, or small eddies. They rotate in the same direction as the ovals without any more energy input from moist convection. This shared sense of rotation among small eddies and ovals seals the fate of the eddies, while feeding the large ovals.

The eddies that form near the same latitude as the Great Red Spot, for example, scurry westward as much as 400,000 kilometers before encountering the slower moving Great Red Spot, says Ingersoll. Being only a couple of hundred kilometers deep, jovian spots behave as if they are two-dimensional, notes Louisville's Dowling. In a two-dimensional fluid flow, he says, "everything that touches, merges." The opposing winds at the point where eddy and spot contact tend to nullify each other; a common, quiet center forms between them; and they and their stores of kinetic energy merge.

Such resupplying of energy ultimately derived from the interior needn't happen all that often, notes Dowling. Even without its weekly recharging, he notes, the Great Red Spot would keep on spinning for centuries. "The reason it can get away with it is there's no surface friction; there's nowhere to stand" on Jupiter, only an atmosphere that becomes more and more dense with depth.

Although moist convection of deep heat now appears to be a prime driver of jovian weather, "there's still a puzzle," says Leovy. Some jets, such as the one the Galileo probe fell through, seem to increase in strength the deeper they go. That renewed a long-standing debate: Is all the weather seen at the jovian cloud tops relatively shallow—rooted only a few hundred kilometers down—or does part of it penetrate thousands of kilometers, to near the planet's heart (*Science*, 12 September 1980, p. 1219)? Galileo won't solve all the mysteries, it seems. **–RICHARD A. KERR**

NEUROBIOLOGY

A New Clue to How Alcohol Damages Brains

It's common knowledge that drinking alcohol can cause birth defects. Even so, 20% of women who drink continue to do so while they are pregnant, according to a 1996 report from the Institute of Medicine. As a result, roughly 1 infant in every 1000 born in the United States has fetal alcohol syndrome, characterized by facial abnormalities, stunted growth, and learning and memory problems. New work now provides some surprising new insights into how alcohol may cause some of that damage.

On page 1056, a team led by neuroscientist John Olney of Washington University School of Medicine in St. Louis and his former postdoc, pediatric neurologist Chrysanthy Ikonomidou (who is now at Humboldt University in Berlin), reports that alcohol works through the receptors for two of the brain's neurotransmitters, glutamate and GABA, to kill brain neurons in rat pups. The animals were exposed to one episode of high blood alcohol during the first 2 weeks after birth—a time when rat brains are going through developmental stages that occur in human brains during the third trimester of pregnancy. Although researchers have known for some time that alcohol exposure late in development causes brain damage in rodents, the new work provides the first possible explanation of how that damage occurs.

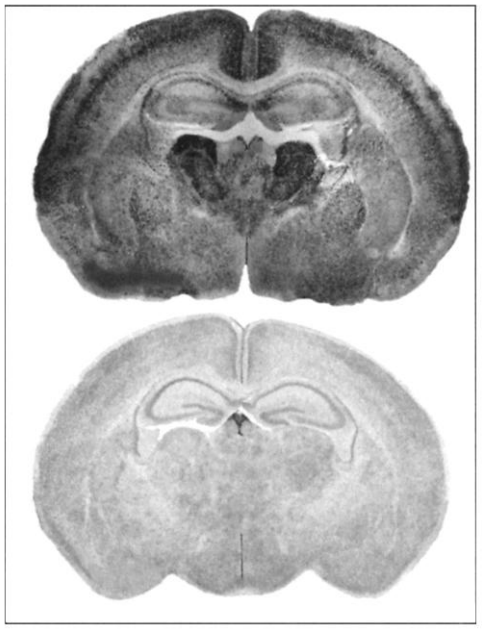
In doing so, says pharmacologist Boris Tabakoff of the University of Colorado Health Sciences Center in Denver, it provides "the first step to understanding how you might control that damage," possibly with drugs that block alcohol's effects on the receptors. But the work carries an even more important message for the public, says neurobiologist David Lovinger, who studies alcohol's effects on neurons at Vanderbilt University School of Medicine in Nashville, Tennessee. Late-pregnancy drinking "is really unsafe for the brain," he says.

Olney and Ikonomidou did not set out to investigate the effects of alcohol on the brain. They were following up on studies they performed last year on chemicals that block the so-called NMDA receptor for glutamate, an excitatory neurotransmitter in the brain. The team's earlier work showed that chemicals that block the NMDA receptor cause brain neurons to die by programmed cell death, or apoptosis, during the phase of development when the brain's neurons are forming connections with each other. The group went on to look for other drugs that trigger apoptosis and found that barbiturates and benzodiazepines, both of which suppress neural activity by activating the receptors for the inhibitory neurotransmitter GABA, caused similar neuronal die-offs.

Because ethanol blocks NMDA receptors and activates GABA receptors, "we thought it would be wise to test ethanol," Olney says. Indeed, the pattern of neuron death they saw with ethanol "seems to be a composite" of what they saw with GABA enhancers and NMDA blockers. That, Olney says, suggests that alcohol's effects on the brain are carried out through these two receptors.

Other groups had previously noted that alcohol exposure late in development causes widespread neuron death in rat brains, and there had been hints that NMDA receptors are involved. For example, pharmacologist Paula Hoffman of the University of Colorado Health Sciences Center and others have shown that blocking NMDA receptors kills

neurons from rat cerebellum maintained in lab cultures. The new work confirms that the same happens in whole animals, and in more areas than just the cerebellum, says Hoffman. What's more, she adds, the suggestion of a role for GABA receptors in the killing effects of alcohol "is quite novel."



Deadly consequences. The black staining shows massive cell death in a brain section from an 8-day-old alcohol-exposed mouse (top). The bottom section is from an untreated mouse.

Indeed, those results raise the possibility that benzodiazepines (Valium and its relatives)—which are sometimes given to newborns as anticonvulsants—may also kill neurons in the developing brains of human infants. "The benzodiazepines are considered extremely safe drugs," says Tabakoff. "If this study is correct, one might need to reassess their safety in [infants] while the brain is still developing."

Ikonomidou points out, however, that the rats were given higher doses of benzodiazepines than those usually given to infants, and it will require more studies to say whether the drugs as they are typically used present a danger to infants. "Prolonged seizures themselves ... can lead to irreversible brain damage, and it is imperative to try and stop them," she notes, adding, "we do not have good alternative drugs to use."

Some alcohol researchers point out that understanding how alcohol kills neurons could spur the development of antidotes that would block its effects in pregnant women. But others see that as a long shot. The Olney group observed the dying neurons 24 hours after alcohol exposure, and the time window for preventing that damage may be too narrow for a "morning after" pill to work. And fetal alcohol researcher James R. West of Texas A&M University's Health Science Center in College Station points out that alcohol doesn't just kill neurons; it has other negative effects, such as causing neurons to grow incorrectly. Because of these multiple effects, there will be "no one silver bullet," West says. "The key thing," he adds, "is to find some way to keep mothers from drinking when they are pregnant." But Tabakoff notes that some alcohol-addicted pregnant women may find it virtually impossible to quit, making progress toward developing a drug that could block even some of alcohol's damaging effects "very, very important."

–MARCIA BARINAGA

STEM CELLS

Wisconsin to Distribute Embryonic Cell Lines

Since the 1998 announcement that scientists had managed to grow human embryonic stem cells in lab culture, researchers have been clamoring for access to them. But they have been blocked on two fronts. One is proprietary—the biotechnology company Geron, which funded much of the work to derive the cells, has kept a tight rein on them. The other is regulatory. In the United States and many other countries, research is restricted because these cells are derived from early embryos or aborted fetuses.

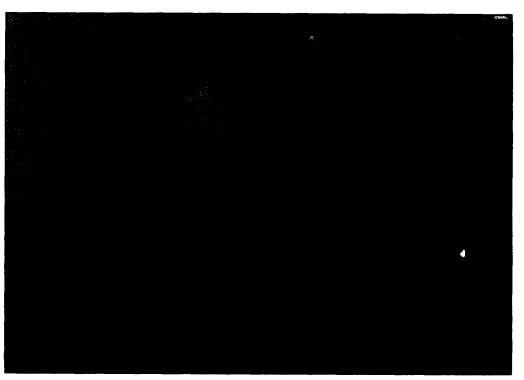
On 1 February, the University of Wisconsin (UW), Madison, took a major step toward lowering the first hurdle. In a move welcomed by many biologists, the university announced the creation of a nonprofit research institute to distribute the embryonic stem cell line derived by UW researcher James Thomson. But the legislative and regulatory limbo persists and may not be resolved before this summer.

What makes these cells so desirable is their potential to develop into any tissue in the body, such as insulin-secreting pancreas cells or dopamine-producing neurons. Researchers envision using these cells to treat a variety of ailments, from diabetes to Parkinson's disease. Not surprisingly, private sector interest in stem cell research is high.

Thomson's work at Wisconsin was supported by Geron Corp. of Menlo Park, California, and by the Wisconsin Alumni Research Foundation (WARF). Although Geron retains rights to certain commercial and therapeutic uses of the cells, WARF retained the right to distribute them to other researchers. WARF has now created the WiCell Research Institute, which will begin distributing stem cells to academic and industrial scientists late this spring. Thomson will serve as WiCell's scientific director but will also continue his research.

Scientists will be asked to submit a confidential summary of their research plans to WiCell, which will review it "to make sure the cells are used appropriately and with adequate respect," says WARF director Carl Gulbrandsen. For example, researchers will not be allowed to use the cells for reproductive cloning experiments or to mix the cells with intact embryos. Academic researchers will pay a one-time fee of $5000 for two vials of the cells, which should provide a virtually unlimited supply of cells in culture. The fee will cover quality control and technical support for the care of the fickle cells. Gulbrandsen says WARF's goal is to create a "not-for-profit subsidiary. We're not intending to make a profit off [academic] researchers, but we're also not intending to lose money."

WiCell will require researchers to certify each year that they are complying with the original agreement. In addition, the license for academics will apply only to research uses. If academics want to use the cells for profit, they will have to renegotiate with WiCell. Geron has commercial rights to "therapeutics and diagnostics with certain cell lines," Gulbrandsen says, but is keeping the details of its license agreement under wraps. WARF has a preliminary agreement with Geron to notify WiCell's stem cell recipients of the areas in which the company has exclusive rights.



Cell promoter. James Thomson's cell lines will be available to researchers.

Industrial researchers, on the other hand, will pay "a significant up-front fee" and a yearly maintenance fee, Gulbrandsen says. WARF will also collect a "flow-through royalty," a percentage of any revenue that results from the use of the cells. Those revenues will have to come from cell uses not already licensed to Geron.

Stem cell researchers welcome the new initiative. "It's an excellent idea to share these cells," says Oliver Brüstle of the University of Bonn Medical Center in Germany.

WiCell's first customers may come from overseas, because U.S. policy is unlikely to be settled by the time WiCell is ready to dis-

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