agrees on the need for caution. To make the treaty work, he says, nations must adopt environmentally sound definitions of forest alterations—an unclear definition could backfire. For example, if "deforestation" leaves out destruction from fire, and "reforestation" is defined as planting trees where forest used to grow naturally, then some countries might encourage burning down forests and replanting them later for credit. This would result in a net carbon loss to the atmosphere because the new forest could take decades to store as much carbon as the original forest. Scientists will discuss possible definitions at the September workshop.

Experts agree that those definitions and

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the calculations for determining whether countries meet the Kyoto goals must take into account different forest types. "If you have a forest like part of the Brazilian Amazon that regrows slowly ... and is inefficient to harvest and use, then it may be best to leave it protected," says Gregg Marland of the Oak Ridge National Laboratory in Tennessee. But for fast-growing forests that can be harvested easily, such as in the southeastern United States, he says, "then it's best to make forest products" that sequester carbon or displace other carbonintensive materials such as cement.

The global picture may seem overwhelming, but sequestration efforts such as the Noel Kempff project in Bolivia suggest that it is possible to take steps now. Whether such schemes will count under the treaty is expected to be a hot topic at a treaty meeting in Buenos Aires in November, in part because most Latin American countries which already have forest conservation programs they want to bolster—are lobbying for it. But Nelson of the Nature Conservancy predicts the issue will not be resolved soon: "Working out how to do these projects is very complicated, because there's so much room for interpretation, so many agendas, and so much at stake."

-KAREN SCHMIDT

Karen Schmidt is a writer in Washington, D.C.

New Network Aims to Take the World's CO₂ Pulse

An expanding array of carbon dioxide monitoring towers around the world could help scientists pin down carbon sinks and enforce the Kyoto Treaty

This spring, a gleaming, 50-meter aluminum tower rose among the aspens and red maples in a Michigan forest, "almost like an extra tree," says Jim Teeri, director of the University of Michigan Biological Station near Pellston. Towers also sprouted in a California grassland, a Quebec wetland, and a Costa Rican rainforest. Fitted with devices for sensing faint whiffs of carbon dioxide, the towers are the latest tool for answering a key question in climate change models: how much carbon is sequestered by ecosystems.

Until now, researchers have picked at the edges of the carbon-cycle problem, either

modeling fluxes globally or looking at tree growth and other clues to carbon storage. But a worldwide network of 70 or so towers now running or about to come online will soon churn out a stream of data on how much CO₂ is socked away in various soil and plant types. That information should, over the long haul, help refine models of global warming as greenhouse gases continue to build up in the atmosphere. "To see what the terrestrial biosphere is going to do in the future, data

from these sites are crucial," says Dave Hollinger, a U.S. Forest Service ecologist.

The need for such data has become more pressing now that 38 nations have pledged to slash carbon emissions under the Kyoto treaty. Tracking CO_2 flux between land and air is "enormously important for what becomes of the treaty," says NASA ecologist Tony Janetos. Indeed, notes Riccardo Valentini of the University of Tuscia in Italy, a flux tower network "can be an independent way to verify [the cuts] that the Kyoto protocol requires." Others caution that the science of tracking CO_2 is still in its infancy. "It seems like a promising approach," says ecologist and climate modeler David Schimel of the National Center for Atmospheric Research (NCAR) in Boulder, Colorado, "but it's also exploratory."

The towers will help probe a long-standing mystery: the so-called "missing carbon." Only half the 7.1 petagrams of carbon released by fossil fuel burning and biomass



Spotty coverage. The global Fluxnet project will feature towers tracking the movement of carbon dioxide between various ecosystems and the air.

destruction each year stays in the atmosphere. The ocean absorbs 2 petagrams, leaving unaccounted for a whopping 1.8 petagrams—enough carbon to fill a soccer field with a pile of coal 230 kilometers high (see diagram on p. 504). When scientists feed data on atmospheric CO_2 levels collected worldwide into climate models and subtract fossil fuel emissions, the results point toward the Northern Hemisphere as a major carbon sink. But forest inventories and landuse studies fail to explain where all the carbon goes, says Schimel, perhaps because these approaches do not fully account for soils, which may absorb as much as twothirds of the missing carbon.

The towers will track carbon by measuring CO_2 breathed in and exhaled by plants and soils. The technique, pioneered in the 1970s, uses wind velocity sensors and infrared gas analyzers to measure CO_2 in air drafts. But "lots of funny things can happen," says Harvard University atmospheric chemist Steven Wofsy. For example, weak nighttime drafts and other factors can lead to underestimates of CO_2 release by 10% or more. Despite this drawback, Wofsy's team has shown that warm temperatures alone do not spur carbon storage at the Harvard Forest—abetting factors include a long growing season, cloud-free summer days, and less snow cover (*Science*, 15 March 1996,

p. 1576). And a study in Canada found that as Earth warms, boreal ecosystems may turn into major sources of CO_2 from thaving peat (*Science*, 9 January, p. 214).

Eager to find out what the towers might reveal about other biomes, researchers running 24 North American flux towers are now organizing a longterm network, called Ameriflux, to monitor ecosystems as diverse as tundra, cropland, and old-growth forest. The

towers will measure CO_2 about 10 times a second over the next 3 years. The Ameriflux team is also linking up with a 3-year-old network in European forests and other towers in Japan, the Amazon, Australia, Siberia, and Southeast Asia. Called Fluxnet, the datasharing project is funded by NASA, which wants to use the data to calibrate an Earth Observing Satellite (slated for launch next

year) that will estimate how much CO_2 plants absorb. Another aim is to pool data on the World Wide Web so that modelers can combine them with data from inventories, satellites, land-use studies, and CO_2 measurements from airplanes. They can then test predictions of how much carbon the different ecosystems now sequester and how much they will absorb as greenhouse gas levels rise.

Early results are exceeding expectations. For example, tower data appear to confirm that in temperate zones, landscapes nearer to the equator are likelier to serve as CO_2 sinks. In Italy, for instance, forests absorb as much as 5 tons per hectare each year. The amount stored drops off further north, and a Swedish boreal forest, where peat may have begun to thaw, actually releases about 0.5 tons of carbon per hectare a year. Thus, the towers may be more helpful than expected in terms of closing in on the missing sink, proponents say. Inspired by these results, NCAR's Schimel has suggested to a White House panel on climate change research that Ameriflux expand its network to perhaps 100 towers, if the cost per tower could be brought down.

Some are even more optimistic. At a Fluxnet meeting last month, scientists reported preliminary findings that European forests absorb a net total of up to 0.28 petagrams of carbon a year—a third of the continent's industrial emissions. According to Valentini, who directs Euroflux, the next step is to add data from grasslands and croplands and plug them into "more sophisticated models" of fluxes between soils, plants, and the atmosphere.

A global network of 250 towers coupled with satellite and weather data might allow monitors to see whether countries are living up to their Kyoto commitments. It "can be an independent way to verify what the Kyoto protocol requires," Valentini says. But such statements make some U.S. scientists antsy. If the towers are seen as "tools for the carbon police," says one Ameriflux researcher, Congress may set out to kill the flux program. Others worry that tower fever will lead agencies to underfund other methods needed to ferret out the missing sink. Even with a larger network, it will be difficult to extrapolate local CO₂ fluxes to a regional level, says land-use expert Richard Houghton of the Woods Hole Research Center in Massachusetts. "I don't think they can do it at the accuracy you'd need," he notes.

Flux tower scientists acknowledge that the program is still proving itself—but they say it is on the right track. Until recently, Wofsy admits he had doubts about how useful the towers would be for closing the carbon cycle: "A year ago, I would have said we're not trying to do that." Now, he adds, the data are more encouraging. "We might be able to make more progress than we thought." –JOCELYN KAISER

NEUROGENETICS

New Gene Tied to Common Form of Alzheimer's

Amutation in a protein that may help scour toxins from between neurons appears to increase the risk of late-onset Alzheimer's disease

Over the past half-dozen years, researchers hoping to pin down the cause of the devastating brain degeneration of Alzheimer's disease have seen their list of potential culprits grow. They've found, for example, that mutations in any of three different genes can cause some cases of early-onset Alzheimer's, which strikes in middle age. In addition, they've identified a variant of another gene that increases an individual's risk of developing the much more common form of the disease that occurs later in life. But researchers have been all too aware that none of these discoveries could fully explain the

late-onset Alzheimer's disease that afflicts so many families. Now, they have an important new suspect to add to their lineup.

Earlier this week, at the Sixth Annual International Conference on Alzheimer's Disease and Related Disorders, which was held in Amsterdam, neurogeneticist Rudy Tanzi of Harvard's Massachusetts General Hospital in Boston reported genetic evidence indicating

tion and nerve cell death.

Gene team. Among those tracking *A2M*'s role are, from left to right: Marilyn Albert, Deborah Blacker, Linda Rodes, and Rudy Tanzi.

that a common mutation in the gene encoding a protein called α_2 -macroglobulin ($\alpha_2 M$) makes the people carrying it more susceptible to developing the neurodegenerative condition as they age. (The results will also appear in the August issue of Nature Genetics.) At present, no one knows how many Alzheimer's cases might be linked to the mutation, but the number could be large, given that an estimated 30% of the population carries the mutation. "I think that [the new mutation] is probably the strongest risk factor for whether you get Alzheimer's late in life-as strong as or stronger than ApoE4," says Alzheimer's expert Sam Sisodia of the University of Chicago, referring to the only other gene currently linked to the late-onset form of the disease.

The new gene and its protein could also make sense of how several other proteins already implicated in Alzheimer's might conwell, for one way $\alpha_2 M$ may prevent β amyloid deposition is by binding the peptide and transporting it into cells for degradation—a step that uses the very same receptor that apoE uses to enter cells. ApoE4 or excess amounts of other apoEs might block the $\alpha_2 M$ – β amyloid complex from binding to the receptor, preventing the cleanup crew from removing its sweepings. All that makes the Tanzi team's discovery "scientifically very interesting," says Steven Hyman, director of the National Institute of Mental Health (NIMH)—and perhaps a clue to new Alzheimer's therapies.

tribute to the disease. Work by other re-

searchers suggests that the normal $\alpha_2 M$ pro-

tein acts as a kind of cleanup crew for neu-

rons by binding to several proteins that

could have toxic effects and sweeping them

out of the space between neurons. These include, for example, the small protein β amy-

loid, already notorious as a possible cause of

Alzheimer's. The mutation may put this cleanup crew out of commission, or at least

slow it down, leading to β amyloid deposi-

tein called apoE, may fit into this picture as

ApoE4, a variant of a lipid-carrying pro-

The apoE4 link was one of the clues that first alerted Tanzi and his colleagues to the gene encoding $\alpha_2 M$. They reasoned that if *ApoE4* is a risk factor for Alzheimer's, then other proteins that bind to the apoE4 receptor, a cell surface protein known as LRP (for low-density lipoprotein receptor–related