RANDOM SAMPLES

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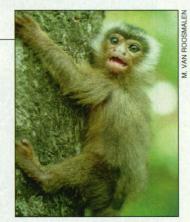
Anteing Up for a World War on Malaria

A plan to launch an international attack on malaria is beginning to pick up steam. The Multilateral Initiative on Malaria (MIM), an idea first proposed last January, can bank on \$2 million this year, an official with the World Health Organization (WHO) said last week at the 2nd Global Meeting on Parasitic Diseases in Hyderabad, India.

Before, only Harold Varmus,

director of the U.S. National Institutes of Health (NIH), had acted on the idea of coordinating malaria research efforts. He had promised to create a repository of research supplies-including purebred parasites and monoclonal antibodies—that would be available free to eligible researchers in developing countries (Science, 13 June, p. 1635; 18 July, p. 309).

Now \$1 million has been allocated by the NIH, another \$500,000 by the Tropical Diseases Research (TDR) Unit of WHO, and \$250,000 by the World Bank, says Tore Godal, head of TDR. The remainder comes from Britain's Wellcome Trust and other organizations. Godal calls the money "a step in the right direction," but argues that only a 20-fold increase in funds will match the real needs of the malaria researchers.



Puny primate. Fits in a teapot.

Brazil's New Monkey

The Brazilian Amazon is turning out to be a veritable treasure trove for new primate species. The latest one is a tiny monkey barely 10 centimeters long and weighing a scant 190 grams. The new species is close to the smallest monkey discovered to date, says primatologist Russell Mittermeier of the Washington, D.C.-based Conservation International.

The current record holder is the 9-centimeter-long pygmy marmoset, says Marc Van Roosmalen, a primate ecologist with the National Institute of Amazon Research (INPA) in Manaus, Brazil. Van Roosmalen has dubbed the new monkey the blackheaded dwarf marmoset. Based on the monkey's behavior and physical characteristics, he thinks it may be a link between the pygmy marmoset and full-sized Amazonian marmosets.

It's the seventh monkey to be discovered in Brazil in the last 7 years and probably won't be the last, says Van Roosmalen. He found this dwarf monkey in a 300,000-hectare region some 2100 kilometers west of Rio de Janeiro. That region was not previously thought to be biologically important, in part because of its proximity to populated areas. However, Van Roosmalen believes he has turned up a dozen or so new, unclassified primate species there during his search for black-headed dwarf marmosets.

A scientific description of the monkey is expected to be published later this year in the Brazilian journal Goeldiana.

Buckyballs Save Nerves

Nerve cells threatened by stroke or degenerative diseases may one day have an unexpected new ally—molecular spheres of carbon called buckyballs. Researchers have used a water-soluble form of these carbon spheres to soak up nerve-destroying chemicals and, in mice, to slow nerve decay.

Although normally of interest to organic chemists, buckyballs attracted the attention of neurologist Laura Dugan of Washington University in St. Louis after she learned of their ability

to get rid of naturally occurring molecules called free radicals. Free radicals have been implicated in nerve decay in Lou Gehrig's disease, multiple sclerosis, and Parkinson's disease.

Standard buckyballs dissolve only in highly toxic compounds such as benzene, so to make these free-radical sponges useful, Dugan's team attached six pairs of water-soluble carboxylic acid molecules to each carbon sphere.

Emulating conditions in the brain after a stroke, Dugan and her colleagues added these modified buckyballs to oxygen- and glucosestarved nerve cells, which build up free radicals. The expected nervecell death was cut by 75%.

They also treated mice bred to mimic victims of Lou Gehrig's disease. Untreated, these mice become progressively weaker and die after about 130 days, but the treated mice lived about 10 days longer, the researchers reported in the 19 August Proceedings of the National Academy of Sciences. The buckyballs "act as an effective antioxidant," sweeping up free radicals, says Jonathan Gitlin, a pediatric neurologist at Washington University.

Prions Up Close and Personal

Misfolded proteins, called prions, have been implicated in "mad cow" disease and in Creutzfeldt-Jakob disease in humans. Now researchers at the Swiss Federal Institute of Technology (ETH) in Zurich have taken an important step toward solving how prions can be pathogenic. They have determined the struc-

ture of the full-length prion protein-all 208 amino acids-in its normal, or healthy, shape.

"It's wonderful stuff," says Byron Caughey, a biochemist and prion expert at the National Institutes of Health's Rocky Mountain Laboratories in Hamilton, Montana. The new close-up, he says, should not only help researchers determine the as-yet unknown function of the normal protein but also provide clues about how it changes to cause disease.

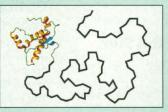
The new prion structure, reported in the 18 August FEBS Letters, follows up on work done over

the last year by the ETH team and, independently, by a group at the University of California, San Francisco. In recent months, both teams unveiled partial structures of the molecule. They had been unable to obtain sufficient quantities of the properly folded full-length protein for study.

The ETH researchers have now used chemical means to fold the full-length protein. They then determined the protein's structure with nuclear magnetic resonance, which identifies and locates the

> protein's atomic components by their specific magnetic fingerprint.

> The previous partial structures looked quite conventionalwith a trio of helices that look like molecular telephone cords-but 3 the new structure reveals, in addition, an unusual flexible tail, 97 \(\) amino acids long. "We were very surprised," says ETH molecular biologist Rudi Glockshuber, who # along with biophysicist Kurt Wüthrich led the Zurich team.



Prion portrait. New structure shows a rigid portion of the protein (left) connects to a floppy tail (right).

Previous research suggests that in prion diseases, this floppy tail adopts a more regular sheetlike structure, which then resists normal breakdown by enzymes, instead forming aggregates. The ETH team's approach yields large amounts of the normal protein, which should make understanding that transformation easier, says Caughey.