



STAR WITH FLARE: Artist's conception of the December 2004 gamma-ray flare, the brightest burst ever seen, expanding from SGR 1806-20.

tial explanation. In an analysis to be published in *Nature*, he and his colleagues conclude that at least a few percent of all short bursts are quite likely to be explained in this way. Based on the observed luminosity and expected frequency of giant magnetar flares, a few dozen of these events per year would occur in other, relatively nearby galaxies. This amount is not enough to explain all short gamma-ray bursts, but, Palmer says,

“5 percent is a good approximation.” He quips that this number “is probably not off by more than a factor of 20, which is actually pretty good in this business.”

As for the cause of the other short gamma-ray bursts, Chryssa Kouveliotou of the NASA Marshall Space Flight Center says that the leading explanation is the violent merger of two neutron stars orbiting each other. But Palmer notes: “With the December 27 event, we now know that neutron-star mergers are not responsible for *all* short gamma-ray bursts. Whether they are responsible for *any* of them is still an open question.” Wijers agrees that it remains unclear whether a neutron-star merger can produce this type of gamma-ray burst.

The answer may come soon, though. Astronomers expect that the Swift satellite, which became fully operational in early April, will accurately pinpoint sky positions and distances for a number of short bursts, enabling scientists to finally get a grip on these enigmatic phenomena. Palmer, for one, is optimistic: “The next gamma-ray burst we see could bring enlightenment.”

Govert Schilling writes about astronomy from Amersfoort, the Netherlands.

NASA

HEALTH

Snoring Suspects

FREE RADICALS MAY SET OFF SLEEP APNEA'S CARDIO DANGERS BY LISA MELTON

Snooring is not just a recipe for marital discord; it can be life-threatening, too, when it is a part of sleep apnea. This disorder, in which breathing stops many times a night, can detonate dangerous cardiovascular stress. But scientists have long puzzled over why we should respond so fiercely to dips in the oxygen supply. Now a new study has identified the tissue and chemical changes that stir up the problem, a finding that could lead to novel drug treatments.

In North America as many as 24 percent of adults suffer from sleep-disordered breathing, a problem exacerbated by obesity. People with obstructive sleep apnea cease breathing for about 15 seconds, every few minutes, hundreds of times a night. Besides feeling drowsy and exhausted the next

day, people with sleep apnea face high blood pressure and risk heart attacks and stroke. Indeed, they are about three times as likely to die from a heart attack in the middle of the night as the general population, according to a study in the March 24 *New England Journal of Medicine*. “The consequences of this intermittent [oxygen deprivation], if it persists for years, can be very drastic,” says physiologist Nanduri R. Prabhakar of Case Western Reserve University.

Prabhakar has long been mystified by sleep



BAD SLEEP: Health dangers lurk in snoring.

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VITAMIN
NONTHERAPY

Free radicals may help trigger sleep apnea, but is targeting them the answer? Lately antioxidants to target these reactive molecules have flunked almost every therapeutic test. "There have been a lot of studies using antioxidants to prevent disease—vitamin C, vitamin E and beta carotene—with not a huge degree of success. The main reason is that antioxidants don't get to the right place and don't decrease free radical damage," says biochemist Barry Halliwell of the National University of Singapore. According to Halliwell, the best solution for sleep apnea is to tackle the problem at the root. "The antioxidants will never do as much good as would be done by controlling your cholesterol levels and losing weight," he points out.

apnea: Why does a brief shutdown of oxygen intake spark an extreme cardiovascular response? After all, people living at high altitude—for example, in the Andes—adapt perfectly well to a low-oxygen environment without developing hypertension.

To pursue this question in molecular detail, he re-created sleep apnea in rats by cutting the oxygen to their cages with a frequency similar to that experienced by human sufferers. At the same time, other rats breathed continuously in a low-oxygen atmosphere that replicated conditions in mountainous areas. Within 10 days, only the rats exposed to oxygen in fits and starts developed hypertension. The most dramatic difference between both groups, Prabhakar announced at a Novartis Foundation meeting in London this past January, showed up in the carotid body, an oxygen-sensing tissue located in the main artery in the neck.

Normally, when oxygen levels drop, the carotid body tells the nervous system that blood pressure must rise to deliver more oxygen to compensate for the shortfall. These urgent signals are prompted by oxygen free radicals acting as messengers. But

when oxygen levels plummet repeatedly, as they do in sleep apnea, free radicals overwhelm the carotid body. The excess jams the carotid body into the "on" mode, so that even when oxygen levels return to normal, blood pressure continues to surge.

Prabhakar speculates that free radical scavengers might counter the devastating effects of sleep apnea. He has tested one such compound—a superoxide dismutase mimetic—in his rat model and found that the chemical averted hypertension. Could a humble antioxidant vitamin supplement do the same for human patients? An antioxidant pill would be an ideal solution, because the only existing therapy is cumbersome: it involves wearing a face mask connected to a positive airway pressure machine during the night to maintain a constant oxygen level.

"Sleep apnea is a much neglected problem," says Prabhakar, whose findings have enhanced our knowledge about the perils that lurk behind these broken nights. He hopes human trials of antioxidant therapy will be able to start soon.

Lisa Melton is a writer based in London.

HOLDING ON TO
HYDROGEN

Besides studying cryoadsorption and storing hydrogen chemically in hydrides, researchers are investigating sorbent materials that can hold hydrogen atoms on surfaces physically. Among the more promising (and the organizations working on them):

- **Carbon nanotubes** with single walls (Air Products and Chemicals, Allentown, Pa.)
- **Organo-metallic fullerenes**, or "buckyballs" (carbon 60), containing iron or scandium atoms (National Renewable Energy Laboratory, Golden, Colo.)
- **Porous silica frameworks** infused with ammonia borane and related compounds (Pacific Northwest National Laboratory, Richland, Wash.)

ENERGY

Solid (State) Progress

HYDROGEN-FUEL STORAGE FOR CARS GETS A MATERIALS BOOST BY STEVEN ASHLEY

Motorists expect cars to go at least 300 miles between fill-ups. That's not a concern for autos that burn gasoline or diesel, but for a future in which vehicles run on nonpolluting hydrogen, adequate driving range remains a real roadblock [see "On the Road to Fuel-Cell Cars," by Steven Ashley; *SCIENTIFIC AMERICAN*, March]. Despite considerable effort, engineers have so far failed to find a way to cram enough hydrogen—the lowest-density substance in the universe—onboard cars.

Conventional approaches to compact hydrogen storage—compressing the gas to up to 10,000 pounds per square inch (psi) or cooling it down to cryogenic temperatures so that it liquefies (around -252 degrees Celsius)—can attain only about half



FILL 'ER UP WITH H₂. Any future hydrogen economy will require compact onboard storage as well as a nationwide distribution and refueling system.