critters' dead bodies," Cliver acknowledges. "So all we know is that by the best available means, we can't get them back after they go onto a board." The big concern is whether bacteria hiding deep within the wood might subsequently surface to contaminate the foods on the chopping block. "As best we can tell, that isn't going to happen," Cliver says.

The same is not true of knife-scored plastic cutting boards. The scientists found that bacteria lodged in the plastic's cut grooves not only survived a hot-water-and-soap wash, but could later surface to contaminate foods. By contrast, Cliver says, with wood "a good wipe will do fine – and if you forget to wipe the board, you probably won't be too bad off."

At one point, the Wisconsin researchers inoculated wood and plastic on three successive days, maintaining each board – without cleaning – at room temperatures and high humidity. By that time, "the plastic boards were downright disgusting," Cliver says, "while the wood boards had about 99.9 percent fewer bacteria than [Ak] had put on them."

"Wood is more forgiving – and perhaps user-friendly – than plastic is once it's been cut some," Cliver says.

Boards sold to homeowners typically come from the factory treated with mineral oil. "That treatment is intended to make the wood more impermeable – like plastic," Cliver says. "The bad news is that it does make wood more like plastic. . . . In every one of our tests, if the wood had been treated to retard the penetration of moisture, the bacteria survived longer."

Wood's presumed bactericidal activity does not depend on whether it is new – nor, apparently, on species. Cliver and Ak have already tested boards from hard maple, birch, beech, black cherry, basswood, butternut, and American black walnut. Tests on oak and ash are pending.

The microbiologists hope to submit their findings for publication within the next few months. One weakness, Cliver notes, is their inability to nail down a mechanism or agent responsible for wood's antibacterial properties.

Although no laws prohibit commercial establishments from using wooden cutting boards, the Food and Drug Administration's model codes for state agencies call for using only "nonabsorbent" and easily cleaned materials for surfaces that food contacts. The USDA also recommends acrylic or other nonporous materials to consumers asking about preferred cutting boards, according to Bessie Berry with its Meat and Poultry Hotline in Washington, D.C.

Cooks should never cut on glass, she says, because minute shards may chip off and become embedded in food.

Microbiologist Priscilla Levine of USDA's Food Safety and Inspection Service says she knows of no scientific studies demonstrating the advantages of one cutting-board material over another in inhibiting bacterial contamination. She told SCIENCE NEWS that her agency based its recommendations on "common sense."

Like state and local inspectors, these federal agencies have "bought the myth" that plastic is safer than wood, says food scientist O. Peter Snyder, a St. Paul, Minn.-based consultant to the retail-food industry. For at least two decades, he says, "sanitarians [sanitation inspectors] out there have been telling us to use plastic cutting boards, even though they had no evidence that plastic was better."

Indeed, Snyder contends, the little research done on the subject has failed to demonstrate plastic's superiority. He cited one study conducted about 25 years ago that showed wooden cutting boards were at least as good as plastic when it comes to cleaning off microbial contamination.

If others confirm the Wisconsin data, Snyder says, sanitarians may have to alter their advocacy in favor of wood. But, he adds, considering how slowly practices change in the food business, 10 years after such confirmatory data came in "sanitarians would probably still be requiring [retail establishments] to use plastic cutting boards." *– J. Raloff*

Dream sleep: A risk for heart patients?

To sleep: perchance to dream: ay, there's the rub/For in that sleep of death what dreams may come. . . .

William Shakespeare wrote of the dark side of sleep in *Hamlet*. Now, a team of Iowa researchers suggests that the dreaming stage of sleep poses a particular peril for people with coronary artery disease.

Humans spend about one-third of their life in sleep. Despite the popularity of sleep, scientists know very little about the physiological changes that occur during slumber. They do know that people typically pass through repeated cycles consisting of several stages of deep sleep followed by a bout of REM sleep, named for the rapid eye movements that take place at that time, when dreams occur.

In 1989, researchers at Harvard University Medical School discovered that people with arterial disease who wake up at night run the risk of ischemia, a reduction in blood flow to the heart (SN: 11/25/89, p.341). That finding fits with the observation that heart attacks often occur in the morning, just after a person wakes up. Indeed, scientists know that REM sleep occurs more frequently during the predawn hours and is associated with an activation of the sympathetic nervous system, which regulates involuntary bodily functions such as heart rate and blood pressure.

To get a detailed picture of what happens during the wee hours, Virend K. Somers and his colleagues at the University of Iowa College of Medicine in Iowa City recruited eight healthy people to spend the night in a sleep lab. The researchers hooked the recruits up to devices that recorded brain waves, heart rate, and blood pressure. By inserting electrodes into the volunteers' leg muscles, the team kept tabs on the sympathetic nervous system.

They found significantly lower heart rate, blood pressure, and sympathetic nervous system activity during non-REM sleep than during wakefulness. This is the first REM study of normal volunteers sleeping at night rather than during the day, Somers says.

The team also reports that REM sleep is associated with a surge of sympathetic nervous system activity. Indeed, these data extend the understanding of dream sleep by finding that sympathetic nervous system activity more than doubled during REM sleep. The researchers describe their findings in the Feb. 4 NEW ENGLAND JOURNAL OF MEDICINE.

"The most striking finding was the association between REM sleep and very, very high levels of sympathetic discharge," Somers says. "We think this may be a potential mechanism that explains the high incidence of heart attacks and strokes in the very early morning hours."

Of course, the Iowa group studied only healthy volunteers and thus cannot prove the link between REM sleep and heart attack risk. However, Somers and other scientists believe that the risk of heart attack starts as the sympathetic nervous system revs up – a process that makes platelets more likely to stick together and already clogged arteries more likely to spasm. During REM sleep, a blood clot may begin to form in the heart's coronary arteries. However, the heart attack doesn't occur until that clot grows large enough to shut off the heart's blood supply, perhaps several hours later, when the victim is rushing to get to work on time.

For people who think the solution to heart attack risk is to spend less time in bed, sleep researcher Richard L. Verrier of Georgetown University in Washington, D.C., warns that sleep deprivation results in a rebound effect. Sleepy people make up for lost time by going through heavy periods of REM sleep, he notes. People with artery disease may benefit from the commonsense advice to get plenty of sleep – and thus avoid heavy REM periods, he says. *– K.A. Fackelmann*