Biomedicine

A heart made of iron—not!

Hawaii's Ironman Triathlon requires athletes to swim 2.4 miles in the ocean, bicycle for 112 miles, and then run a marathon of 26.2 miles—one right after the other. That punishing workout resulted in biochemical evidence of heart damage for some athletes, according to a study reported at the 69th Scientific Sessions of the American Heart Association in New Orleans. None of the 23 male and female athletes in the study had earlier evidence of heart disease.

Nader Rifai of Harvard Medical School in Boston and his colleagues collected blood samples from the participants before and after the triathlon. The researchers discovered that immediately after the race, 6 of the 23 athletes had elevated concentrations of troponins—proteins produced by injured heart cells—in their blood. Two of those six participants had troponin concentrations as high as those found in the blood of heart attack patients. None of the athletes had had abnormally high concentrations of these proteins before the competition, Rifai says.

The researchers also took ultrasound pictures of the athletes' hearts before and after the race. All prerace tests were normal, but immediately after the race, ultrasound images of five of the six athletes with elevated troponins showed abnormal heart function. The abnormality was most pronounced in the athletes with the highest troponin concentrations.

These findings suggest that prolonged and extremely arduous exertion may injure the heart. No one knows at what point the damage occurs or how long it lasts. Until further research clarifies that potential risk, says Gerald F. Fletcher, a cardiologist at the Mayo Clinic in Jacksonville, Fla., routine moderate exercise may be better for the heart than trying to cram in a grueling workout on an occasional basis.

Is buck fever a heart hazard?

You don't need to struggle through a triathlon to endanger your heart. Deer hunting can put men with clogged arteries at risk of a heart attack, hints a second study reported at the 69th Scientific Sessions of the American Heart Association in New Orleans.

Susan Haapaniemi of William Beaumont Hospital in Royal Oak, Mich., and her colleagues wondered whether the stress of a hunt could trigger hazardous changes in heart rate. They decided to find out by giving 25 men portable heart monitors to wear during a deer hunt. Seventeen of these hunters had previously been diagnosed with atherosclerosis, a condition in which fatty plaque clogs the arteries.

The researchers' data revealed that deer hunting puts rigorous demands on the cardiovascular system. For some men, the adrenaline surge occasioned merely by sighting a deer pushed their heart rate into the danger zone. One man's heart rate soared from 78 beats per minute to a whopping 168—while he was sitting in a tree, Haapaniemi told SCIENCE NEWS.

Success in hunting also proved exceptionally strenuous. The American Heart Association recommends a target zone for exercising that puts the heart within 50 to 75 percent of its desirable maximum rate. Shooting a deer sent heart rates up to 118 percent of that level, Haapaniemi says. Dragging a dead deer back to the road sent some hunters' heart rates up to 116 percent of the desirable maximum, Haapaniemi noted, adding that hunters often drag a deer for about an hour.

High heart rates strain the heart. For people with already clogged arteries, that strain could lead to a heart attack, Haapaniemi says.

The researchers urged hunters with heart disease to avoid dragging a deer, the hunting activity that makes the most sustained demand on the heart.

Gerald F. Fletcher of the Mayo Clinic in Jacksonville, Fla., says that people who love to hunt should condition themselves ahead of time so they avoid triggering a high heart rate.

Hunt intensifies for Parkinson's gene

Researchers seeking a genetic cause of Parkinson's disease owe thanks to the descendants of a couple who lived in southern Italy during the 18th century.

This lineage is remarkably prone to the neurodegenerative disorder. In the Nov. 15 SCIENCE, investigators report how DNA samples taken from a few dozen living members of the family revealed that a gene causing the disorder resides on the long arm of chromosome 4. Mihael H. Polymeropoulos of the National Center for Human Genome Research in Bethesda, Md., and his colleagues localized the gene by identifying other genetic sequences that were frequently inherited by family members with the disease but not by those without it.

The eventual identification of the chromosome 4 gene may help investigators understand what triggers this familial Parkinson's disease and perhaps the much more common noninherited cases that afflict more than a million people in the United States. Since treatment options for people with Parkinson's are limited and unsatisfactory, scientists also hope the gene's discovery will suggest new therapies.

Can a cold virus slay cancer cells?

It's a clever idea with an uncertain future. In the Oct. 18 SCIENCE, Frank McCormick and his colleagues at ONYX Pharmaceuticals in Richmond, Calif., describe a mutant virus that can kill cancer cells. In one experiment, they implanted human cervical cancer cells into mice and let tumors grow. They then showed that the virus could reduce the size of those tumors and sometimes eliminate them.

The cancer-killing mutant is an adenovirus, one of the many viruses that can cause colds in humans. This particular virus has a mutation that allows it to replicate only in cells lacking a protein called p53. Almost all normal cells synthesize p53, which guards against uncontrolled cell growth. Yet in more than half of all cancers, the tumor cells don't make p53. Thus the virus can reproduce in tumors and destroy them.

Physicians have begun clinical trials with the mutant virus, but one of the scientists who first described it in 1987 doubts that the treatment will prove successful. "The virus has to grow inside the patient, but there will probably be a very effective immune response against it because most everyone was infected with this kind of virus in childhood," says Arnie Berk of the University of California, Los Angeles.

Huntington's disease strikes mice

Huntington's disease, a fatal neurodegenerative disorder, stems from an unusual mutation in which a gene becomes too big for its own good. In people with the disease, a small bit of the gene's DNA sequence repeats an abnormally large number of times (SN: 6/10/95, p. 360). Following the mutant gene's instructions, cells create a protein that has extra amino acids.

A collaboration of scientists from the United Kingdom and Germany has now raised mice whose genomes include a small portion of the repeat-laden human Huntington disease gene. As they age, the mice begin to suffer symptoms of Huntington's disease, including tremors, epileptic seizures, and neurodegeneration. "Their brains are 20 to 30 percent smaller than [the brains of] their normal siblings," says Gillian P. Bates of Guy's Hospital in London, who headed the research effort.

Bates suggests that the mice will help investigators unravel the mystery of how the mutant proteins cause Huntington's disease. "It's beautiful, very important work. The mice show the classic signs of the disease," says Richard Myers of Stanford University, who has recently added a much larger portion of the disease gene to mice. Myers and his colleagues are still waiting to see if their mice exhibit symptoms.

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