Biomedicine

Joanne Silberner reports from Dallas at the American Heart Association Scientific Sessions

No free lunch

Since drugs, as well as exercise, can stimulate the heart, can swallowing a pill be as good for you as sweat and strain? Probably not, according to a study by Eugene E. Wolfel of the University of Colorado Health Sciences Center in Denver.

Wolfel and his colleagues set out to study the role of heart stimulation independent of exercise in attaining cardiovascular fitness. If physical movement isn't required, people with heart disease theoretically could be spared the effort.

The researchers gave dogs a drug used in treating heart failure, and compared the effects to exercise training. The exercised dogs showed various cardiovascular benefits not seen in the caged dogs given the drug, and subsequently were able to exercise longer. "This is saying you can't take a drug to attain cardiovascular fitness," Wolfel says.

Tis the season

While you're out there buying holiday gifts, feasting and resolution-making, consider this: A new study indicates that your cholesterol level may be about to go up by about 7.4 milligrams per deciliter of blood. (The median level in middle-aged Americans is 200 mg/dl.) Surprisingly, increased eating and decreased activity reported by the subjects accounted for only about a third of the cholesterol change, suggesting some other cyclical pattern is at work.

David J. Gordon and his colleagues at the National Heart, Lung, and Blood Institute found the change while looking for long-term trends in data collected for up to 10 years in 1,446 men with high cholesterol levels. To their surprise they found a yearly, cyclic pattern peaking Dec. 15 to Jan. 12 and hitting a nadir in June. Gordon suspects the winter increase holds for other men as well; for women he's less certain. The researchers were also surprised to find that among the 12 cities where data were collected, the two with the least seasonal changes, Houston and San Diego, showed greater cholesterol changes than cities as far north as Minneapolis.

The holiday cholesterol elevation is not enough to be health-threatening, but it could make a doctor think that a patient on a strict cholesterol-lowering diet is cheating. "I don't know what's really causing the seasonal effect," says Gordon.

Smoking, heart attack link

About 5 to 10 percent of heart attack victims don't have significant hardening of the arteries. But a disproportionate number of them, especially in the younger age groups, are smokers, says Michael J. Pecora of Emory University in Atlanta. While this association does not prove that smoking causes heart attacks in the absence of atherosclerosis, it is another reason not to smoke, says Pecora.

He and his colleagues studied 48 people who had little or no atherosclerosis yet had suffered heart attacks. Seventy-one percent had a history of smoking; the proportion was higher — 78 percent — for those under 60 years of age.

Salt balance and thyroid function

Atrial natriuretic factor (ANF), a hormone discovered in 1981, controls salt and water balance. High levels of the hormone are found in victims of congestive heart failure, and preliminary human trials have shown that a booster of the protein can reduce blood pressure in some cases. Now researchers say they've found the first metabolic abnormality associated with *decreased* levels of the protein.

Robert S. Zimmerman and his colleagues at the Mayo Medical School in Rochester, Minn., measured ANF levels in 11 patients with underactive thyroid glands. The levels were about two-thirds those of a healthy control group. ANF increased after the patients' thyroid conditions were treated.

Chemistry

New hazard in leaded gas?

Though ethylene dibromide (EDB) is best known today as the chemical fumigant banned from use as a pesticide in most U.S. agricultural and food operations (SN: 11/10/84, p.296), it is also an additive in leaded gasoline. Used alone or together with dichloroethane (DCE), it helps prevent deposition of lead compounds on engine parts. "The common understanding was that [EDB and DCE] are completely decomposed during combustion," say Markus D. Müller and Hans-Rudolf Buser of the Swiss Federal Research Station in Wädenswil. But a study they report in the November Environmental Science and Technology suggests otherwise.

Their mass spectra analysis of the exhaust of idling cars fueled with leaded gasoline revealed trace quantities of 44 unexpected halogenated compounds, most of them containing bromine. (Halogens are a group of elements on the periodic table that include bromine and chlorine.) In fact, the chemists note, the EDB-based brominated compounds prevailed even in the exhaust of gasoline that had initially contained equal quantities of both additives. None of these halogens was identified in the exhaust of vehicles fueled with unleaded gasoline.

Although the researchers note that the toxicity of most of the compounds, such as 2,4,6-tribromophenol, is not known, they do point out that several of the brominated ones are structurally similar to toxic chlorine-based herbicides.

Environmental contamination with these gasoline additives may be somewhat less of a concern for U.S. motorists since a dramatic phasedown in the use of lead in gasoline was completed earlier this year (SN: 3/23/85, p. 187).

Vitamin A effects of PCBs and dioxins

It has been observed that certain halogenated chemicals — among them polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs) and dioxins — can reduce concentrations of vitamin A circulating in the blood or stored in the liver, creating potentially serious deficiencies. What hasn't been understood is the mechanism. Now Dutch researchers working with the PCB known as 3,4,3′,4′-tetrachlorobiphenyl (TCP) have unraveled its effect in rats. Their work, reported in a recent issue of Toxicology and Applied Pharmacology (Vol. 85, No. 3), indicates that a metabolite of the PCB binds to a blood protein, transthyretin. This binding interferes with formation of the blood-protein complex used to transport both vitamin A and the thyroid hormone thyroxin, according to the researchers, A. Brouwer and K.J. van den Berg at the Radiobiological Institute in Rijswijk.

Like the Dutch team, Steven Aust, a toxicologist at Michigan State University in East Lansing, has found ties linking these chlorinated compounds, vitamin A and thyroxin. However, he largely discounts the applicability of the Dutch findings to most of the other related compounds of toxicological concern.

The 3,4,3′4′-TCP used in the Dutch study "is detoxified" when it is broken down in the body's metabolism process, Aust notes. "Such is not the case for dioxin" and many other PCBs, his own research indicates. He told Science News that "it appears that this particular chemical — 3,4,3′,4′-TCP — affects vitamin A differently than the other chemicals." In fact, he says, the TCP's nonmetabolizing relatives appear to raise, rather than lower, blood serum concentrations of vitamin A — perhaps an indication that they are pulling vitamin A from its storage in the liver.

Aust's newest findings also indicate that feeding thyroxin to animals that have ingested TCDD — the most toxic of the dioxins — will increase the TCDD's toxicity and magnify its effects on vitamin A, causing a more severe decrease in the body level of vitamin A. Similarly, he says, he's showing that rats whose thyroid glands have been removed — leaving them with less thyroxin — are far less severely affected by dioxins.