tion associated with coronary heart disease in humans can be triggered in laboratory animals merely by putting them on a copper-deficient diet.

And he recently did. Using mice, he attempted to repeat a study performed 20 years ago, which had reported finding a link between dietary fat and coronary heart disease-type effects. But there was a "hidden variable" in that earlier diet, Klevay says, because it had been low in copper. When he compared the disease effects reported with factors linked elsewhere to copper deficiency, he says, it became apparent that nearly all the factors attributed to fat could be accounted for by the copper deficiency alone. To test that, he repeated the study with copper being the only variable. And his results, published in the February ATHEROSCLEROSIS, matched those of the earlier study — serious heart disease.

In another previous study, 13 of 15 animals on copper-deficient diets had dropped dead in the fifth week of a six-week study. "Limited autopsies showed ruptured hearts and aneurysms," Klevay says. In later, similar tests, he identified major electrocardiogram (EKG) changes as well. While puzzling over the EKG changes, Klevay ran across a reference to EKG changes in human participants in the famous Framingham study (SN: 1/24/81, p. 55) that were deemed predictive of an individual's potential for developing heart disease. Klevay's animals had exhibited two of those predictive EKG changes.

Harold Sandstead, former director of the Agriculture Department's Human Nutrition Research Center on Aging at Tufts University in Boston, described his research showing elevations in one man's blood cholesterol — from 206 mg/deciliter of blood to 235 mg/dl — after the man was put on a diet having only 0.8 mg of copper for 105 days. The man's cholesterol levels dropped back to 200 once he resumed a copper-adequate diet.

Copper is found in such foods as beef liver, nuts and seeds, dark chocolate, breakfast cereals and goose breast. But eating 2 mg worth of copper daily won't ensure a sufficiency of the mineral. Sandstead cites animal studies by Sheldon Reiser at the Agriculture Department's Carbohydrate Nutrition Lab in Beltsville, Md., that suggest that diets high in fructose — a simple sugar contained in ordinary sucrose, or table sugar — reduce the body's ability to absorb copper. In fact, Reiser says, copper-deficient animals fed fructose will start dying in five weeks of catastrophic heart disease — such as ruptured hearts — while similarly copper-deficient animals whose sugar source was cornstarch survive comfortably. Reiser says that in the fructose group, "every index of unfavorable metabolic effect was magnified."

Fructose appears to be only one of a group of chemicals that raise cholesterol and inhibit copper metabolism, Klevay says. Moreover, there is also a group that lowers cholesterol and enhances the body's uptake of copper; it includes aspirin, calcium and carbonates.

Walter Mertz, director of the Agriculture Department's Beltsville Human Nutrition Research Center, focused on chromium. In humans, he says, this is one of the few trace elements "that consistently declines with age." In the United States and other industrialized countries, he notes, glucose tolerance — the rate at which the body metabolizes excess sugar — also declines with age. Since chromium is known to help bring the hormone insulin together with insulin receptors on a cell's surface, there's growing suspicion that some element of mature-onset diabetes may be fostered by a chromium deficiency. Animals with chromium deficiency will metabolize sugar at almost half the normal rate, Mertz says, "which means, at least in animals, that chromium is necessary for maximum effectiveness of insulin." Currently, he is trying to find out "in what cases and under what conditions we can improve the glucose metabolism of middle-aged people." He's also trying to identify what conditions, such as exercise, influence human chromium requirements.

New data from Japan suggest a second major role for chromium, Mertz says — the stimulation of DNA transcription within cells. While it's still too early to be sure, he says, chromium seems to be a part of a very specific protein that influences a cell's nucleic acid metabolism.

Work by Herta Spencer is showing that through their dietary choices, many people risk developing serious zinc deficiencies. New data by Spencer, chief of the metabolic section at the Veterans Administration Hospital in Hines, Ill., on obese men hospitalized to lose weight, show that weight loss generally correlates with losing zinc. Zinc deficiency can cause skin rashes, appetite loss, poor wound healing, mental lethargy, hair loss and taste disturbances.

The recommended daily allowance (RDA) for zinc is 15 mg. However, many diets — even those where weight reduction is not a goal — are deficient in this essential metal. One analysis Spencer performed on hospital diets showed that zinc levels in supposedly well-balanced, calorie-adequate diets could range from 4.6 mg to 19 mg per day. Zinc levels tended to vary with dietary levels of animal protein, especially fish and red meat (though legumes, wheat germ and cheese are also good sources). Because weight-reducing diets are frequently low in animal protein, they risk being low in zinc, Spencer says. However, it's not just the inherent zinc level that makes such diets a threat to zinc sufficiency. Preliminary data suggest that protein is needed to carry zinc across the intestinal lining so that it can be absorbed, she says. What's more, certain chemicals like EDTA — used for removing lead from the body — will pull huge quantities of zinc, her research shows. In persons who had been excreting 0.4 mg of zinc daily in urine, three days of EDTA treatment was enough to pull 64 mg of zinc from their body stores.

Ironically, the use of concentrated zinc supplements to counteract this problem may only create another — a calcium deficiency. In a just-completed study, Spencer found that high levels (140 mg) of zinc reduced the body's ability to absorb calcium — from a normal 69 percent to only 39 percent — when the diet contained only a quarter of the RDA for calcium. Spencer notes that for women whose diets are already seriously low in calcium — as most U.S. women's diets are — such zinc supplementation might increase their risk of postmenopausal bone loss. *— J. Raloff*

A new look at arthritis origins

People with rheumatoid arthritis generally have hot, swollen, inflamed joints, and this process of inflammation is generally considered to be the root of the disease.

But according to evidence presented this week at the meeting in Anaheim, Calif., of the American Rheumatism Association, and in the May ARTHRITIS AND RHEUMATISM, the inflammation may be secondary to proliferation of the synovial cells lining the joint.

Researchers from World Health Organization arthritis centers at the University of Alabama at Birmingham (UAB) and Mainz, West Germany, have found that, at least in the mouse model they are studying, the earliest change in tissues destined to become arthritic is not inflammation but an uncontrolled reproduction of synovial cells. Using a strain of mice that spontaneously develop arthritis, they observed a "clear-cut dissociation" between tissue destruction and overt inflammation.

"It suggests an alternative mechanism of joint destruction other than classical inflammation," says UAB's William Koopman. "Whether it will hold true [in humans] is speculation."

One factor supporting the theory, Koopman told SCIENCE NEWS, is that anti-inflammatory drugs, while relieving some of the symptoms of rheumatoid arthritis, don't prevent joint destruction.

"There are patients who don't seem to have a lot of inflammation — their joints are not warm, swollen or tender — yet there is evidence of joint destruction," notes Koopman.

Hans-Georg Fassbender of the arthritis center in Mainz looked at more than 20,000 tissue biopsies from people diagnosed with rheumatoid arthritis, and found that some of these people had little inflammation. Fassbender's pathological evidence, combined with the clinical evidence of "symptomless" patients, suggests that inflammation is not the whole story in hu-

mans as well as in mice.

Comments arthritis expert James Klinenberg of Cedars-Sinai Medical Center in Los Angeles, "We have simplistically said the proliferating [synovial cells] come from an inflammatory stimulus." But a cause-and-effect relationship has never been proved, he notes. The UAB work "is reasonable and exciting — something to speculate about."

If the problem does prove to be proliferating synovial cells, "It could point the way to the development of agents to interrupt the process," says Koopman. But it also raises a key question: If inflammation doesn't kick off the cell proliferation, what does?

Researchers from the Scripps Clinic and Research Foundation in La Jolla, Calif., suggest that one cause may be Epstein-Barr virus, which may stimulate the immune system into marshaling an attack against the body's cartilage and joints.

—J. Silberner

A fresh start at Three Mile Island?

After almost six years of hearings, investigations and legal disputes, the Nuclear Regulatory Commission (NRC) last week voted 4-1 in favor of allowing the undamaged unit (TMI-1) of the Three Mile Island nuclear power plant to begin operating again as soon as June 11. TMI-1 has been shut down since a catastrophic accident struck its nearby twin, TMI-2, in March 1979.

The decision, however, was immediately challenged when the state of Pennsylvania and Three Mile Island Alert, a Harrisburg-based citizens' group, filed separate petitions with the federal appeals court in Philadelphia. Both petitions contend that NRC did not have enough information to make a proper decision and that further hearings are necessary. The chief doubts center on whether General Public Utilities Corp. (GPU) has the "character and integrity" to operate a nuclear power plant safely.

Says Pennsylvania Governor Richard Thornburgh, "Until we can be assured that the plant can be safely and competently operated, no action to open the plant should be taken."

"GPU is not fit to hold a license to operate TMI-1," insists Ellyn R. Weiss, general counsel for the Union of Concerned Scientists, based in Washington, D.C. She points to evidence of reactor operators who cheated on their qualifying exams, allegedly false testimony from GPU officials at congressional hearings, harassment of workers involved in the TMI-2 cleanup, and the conviction of Metropolitan Edison Co. (the GPU subsidiary that originally operated TMI) for falsifying leak-rate data.

GPU officials say that TMI's new operator, another subsidiary called GPU Nuclear, "is ready and able to operate TMI-1 safely." Says GPU Chairman William G. Kuhns, "It has not been a quick fix, rather a deliberate, thoughtful program of developing what we want to be the finest nuclear operation in the country."

NRC's General Counsel Herzel Plaine, in his report to NRC, agrees that management faults have been corrected. GPU Nuclear "represents a significantly improved organization over Metropolitan Edison Co.," he says. But critics say that GPU has merely shuffled individuals around.

Moreover, the path NRC followed to come to a decision is itself controversial. "The NRC's administrative process on the restart of TMI-1 has often appeared tangled and confused with partial initial decisions leading to appeals, resulting in reopened and remanded hearings at every turn," says Rep. Don Ritter (R-Pa.).

At the time of the TMI-2 accident, TMI-1 had been shut down for routine refueling. That summer, NRC issued two orders that kept TMI-1 closed and specified which issues had to be resolved before the plant could restart. Most of the work fell to NRC's Atomic Safety and Licensing Board, which looked at the problems one by one and issued "partial initial decisions" as each problem was cleared up to its satisfaction.

Last February, a majority of NRC's five commissioners decided that all of the major issues had been settled and that no further hearings were necessary. But two commissioners were sharply critical of that decision.

Commissioner James K. Asselstine, who voted against the restart order, argues that the NRC either ignored or discounted important issues. "The Commission's decision-making process and its refusal to allow further hearings has not promoted public confidence," says Asselstine.

Commissioner Frederick M. Bernthal also criticizes what he calls NRC's "unwise and ill-considered path," although he voted in favor of restart on technical grounds. "I suspect that only the lawyers may delight in the decision," he says, "because their future is assured."

And many residents of Dauphin County, where TMI is located, are worried. "Doesn't the NRC understand that keeping TMI closed is the only way we can gain the peace of mind that we lost over six years ago?" asks County Commissioner Larry Hochendoner.

Nevertheless, unless the federal court delays implementation of NRC's restart order and if two NRC-imposed conditions are met, at noon on June 11, TMI-1 operators will have the authority to start producing power again. *—I. Peterson*

Court blocks Army testing laboratory

A U.S. district court has enjoined the Army from continuing the construction of a proposed aerosol toxin laboratory at the Dugway Proving Ground in Utah. The court ruled that the Army has not adequately considered and disclosed the potential environmental impact of the facility.

The suit was brought by the Foundation on Economic Trends, a Washington, D.C.-based public-interest organization, and two retired military officers (SN: 12/22 & 29/84, p. 397). The foundation, led by Jeremy Rifkin, has also won cases blocking the deliberate release of genetically engineered microorganisms and requiring the National Institutes of Health to prepare an environmental assessment of such experiments (SN: 3/9/85, p. 148).

The Dugway Proving Ground, located 87 miles southwest of Salt Lake City, is a Defense Department installation to assess the "military value of chemical warfare and biological defense systems," according to the defendants. Congress approved funds in 1984 for a modernization, which would include construction of an aerosol toxin laboratory. The Army says the laboratory was designed to meet the most stringent safety regulations for work with genetically engineered organisms, in case the Army decided to test such organisms at some future date.

The plaintiffs argued that the Army should be required to prepare a formal environmental impact statement on the grounds that the laboratory would likely be used for work with recombinant DNA organisms. Judge Joyce Hens Green decided that these grounds were not the most important aspect of this case, but that the quality of the environmental assessment was. She points out that pathogenic agents and toxins will be used in the proposed facility, even if genetically engineered organisms are not. "Indeed," she says in her written opinion, "the attention riveted on [the genetic engineering] issue has to some extent masked a more important one.... a proposed federal action — regardless of whether it involves new or different technology—must be accompanied by an environmental impact statement or an environmental assessment that comports with applicable standards of judicial review."

Green agrees with the plaintiffs that the assessment published by the Army is "clearly inadequate.... [It] represents but an amalgam of conclusory statements and unsupported assertions of 'no impact.'" Finally Green weighed defense concerns in her decision, and concluded, "With the mighty power and resources of our government, the defendants... can determine the urgency of the situation and move accordingly." *—J.A. Miller*