was an association between cholesterol levels and death at age 50, but not by age 60. They point out that the over-50 data are complicated by dropping levels of cholesterol seen in many subjects (perhaps, say the researchers, as a sign of other diseases). The NHLBI analysis also did not show either increased mortality at low cholesterol levels or an overall relationship between cholesterol and cancer mortality. (Other studies had suggested one or both relationships.)

In 1984, directors of another NHLBI study predicted that lowering cholesterol through diet and drugs might eliminate one-fifth of the 500,000 fatal heart attacks in the United States each year, based on a 10-year look at men with high cholesterol levels (SN: 1/21/84, p.38). The following year, results from a six-year study of more than 350,000 men by Northwestern University Medical School in Chicago indicated that the risk of heart disease increases steadily as cholesterol levels rise (SN: 11/30/85, p.344). Several national associations have released diet guidelines aimed at lowering cholesterol intake. But for scientists the message remains a mixed one.

Using data from the 1984 and 1985 studies, as well as Framingham data, to build a statistical model, William C. Taylor of the Harvard Institute for Health Research and his co-workers at several other Boston research centers now suggest a somewhat different picture.

The Boston group looked at life expectancy and cholesterol levels, as well as smoking and high blood pressure, both considered risk factors for heart disease. In the April Annals of Internal Medicine, the scientists predict that, for persons aged 20 to 60 years, a "lifelong program" of cholesterol reduction would increase life expectancy only three days to three months among those who have normal blood pressure and do not smoke. For persons at high risk (smokers with high blood pressure and high cholesterol levels), added life expectancy ranged from 18 days to 12 months,

Two editorials, by scientists at the Harvard Medical School and the University of Michigan School of Public Health in Ann Arbor, which accompany the Taylor article, echo cautionary statements by Taylor and his coauthors that life-expectancy calculations are a type of educated guess, not certainties. But the report illustrates the confusion over cholesterol reduction as disease prevention. Both editorials suggest that public health would be better served by reducing smoking and high blood pressure, rather than lowering cholesterol intake in the general public. — *D.D. Edwards*

Winning and losing in the AIDS fight

Which groups in the population are at risk for developing AIDS and why some individuals infected with the AIDS virus do not develop the fatal disease are among the key unanswered questions in the spread of AIDS. More possible answers came last week in two published reports — one saying that the proportion of female AIDS patients who acquired the disease through heterosexual contact more than doubled from 1982 through 1986, the other suggesting that a substance involved in cellular protein production may be the trigger that turns a latent viral infection into a case of AIDS.

In a study reported in the April 17 Journal of the American Medical Association, Mary E. Guinan and Ann Hardy of the Centers for Disease Control (CDC) in Atlanta considered all reported cases of AIDS in women as of Nov. 7, 1986. (Reporting of AIDS cases to CDC began in mid-1981.) While the development of AIDS in nearly half of the 1,819 female cases was due to intravenous drug use, the proportion of women with AIDS due to heterosexual contact with men considered at risk for AIDS increased from 12 percent in 1982 to 26 percent. Guinan and Hardy describe high-risk men as being primarily intravenous drug users or bisexual.

This dramatic increase, say the authors, has implications for the spread of both heterosexual AIDS and pediatric cases caused by infected mothers. The prevalence of heterosexually spread AIDS has been an issue of some controversy (SN: 1/4/86, p.11).

It's a matter not merely of which groups are at risk, but also of which individuals. Scientists are actively seeking one or more cofactors that might explain why AIDS viruses that lie quietly inside the body's cells for perhaps as long as 15 years suddenly burst forth with a deadly vengeance (SN: 4/4/87, p.220). One factor activating virus production may be a cellular protein called NF-κB, according to a report in the April 16 Nature by Gary Nabel and David Baltimore of the Whitehead Institute for Biomedical Research in Cambridge, Mass.

The researchers found that NF-κB, which has a role in DNA's control of protein production in cells, increases in concentration when the body is challenged by infection. Based on their study, Nabel and Baltimore suggest that increased levels of NF-κB, formed in response to other viral or bacterial attacks, may activate the genetic mechanism of cells already containing the genetic code for the AIDS virus — resulting in a flood of AIDS virus being produced. — *D.D. Edwards*

Netting a better sales route

A hypothetical salesman must visit customers in a number of cities scattered across the country and then return home. The problem is to find the shortest possible route. This classic situation is one of many similar mathematical problems that turn out to be difficult to solve, not because a method isn't known, but because brute-force procedures, such as trying every conceivable combination, take too long. Even the fastest computers available would require years to handle a 30-city itinerary.

Recently, scientists have focused considerable effort on finding efficient methods that — short of a definitive solution — close in on a near-optimal answer to the traveling salesman problem within a reasonable time. Molecular biologist Richard Durbin of the King's College Research Centre in Cambridge, England, and zoologist David Willshaw of the University of Edinburgh in Scotland propose the latest shortcut — an "elastic net" to snare a potential answer. Their report appears in the April 16 Nature.

Durbin and Willshaw take a geometrical approach. They start with a small circle in the midst of a group of points, representing cities, randomly scattered across a certain area. Points on the circle correspond to cities in the plane. Under the influence of "forces" exerted by the cities, the circle is gradually stretched in various directions until it forms an irregular loop that eventually passes near every city. One "force" moves a point on the circle toward cities that are closest, while a second "force" pulls it toward its neighbors on the path.

"Each city becomes associated with a particular section of the path," the researchers say. "We call this algorithm the elastic net method because of the way the initial path is gradually deformed to produce the final tour."

The elastic net algorithm lends itself to implementation in computers that can perform many operations in parallel at the same time. The method comes up with paths that are within 1 percent of the shortest known tours connecting 100 randomly distributed cities. The algorithm also provides a general method for matching a set of arbitrarily connected points to a second set of points located in a geometrical space of any dimension.

The elastic net algorithm originated in a mathematical model that was first used to suggest how nerve cells in two different locations can be linked so that neighbors in one set of cells are also neighbors in the corresponding set of cells elsewhere. Now Durbin and Willshaw are looking into how their new algorithm sheds further light on neural connections.

— *I. Peterson*

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