MEDICINE

Body's Emergency Fuel

Recent research shows that unesterified fatty acid, stored in the adipose tissue and transferred when needed to the blood plasma, gives emergency sustenance to the body.

➤ THE MAJOR energy source for powering the heart when the human is not eating, together with its supply depots and transport system, has been found.

The fuel that keeps the human power plant going during fasting between meals, snacks and coffee breaks is unesterified fatty acid (UFA). Without it, a human might die between dinner and breakfast. The alternative would be to eat continuously.

UFA is the body's starvation energy. Although it has been known to exist in blood plasma for many years, it is only within the past few years that its life-sustaining role has been unraveled.

Heart muscle cells can never stop working. They, like other cells, must have food to convert into energy, much the same as an automobile needs gasoline and a steam power plant needs coal. Without eating, even for as short a period as between dinner and breakfast, food must be available to these cells. An emergency supply or some other source of fuel is needed. This has been found to be unesterified fatty acid. UFA literally gives the heart the "oomph" needed to sustain life in the absence of the foods we normally eat.

This fact was uncovered only recently by scientists at the National Heart Institute and in independent studies based on different techniques by sicentists at the Rockefeller Institute in New York.

The Heart Institute researchers have been able to draw this picture of UFA and its transport system so far: it is the body's starvation energy, held in storage depots in the adipose tissue. When the human is not eating, UFA is transferred from its adipose tissue depots to the blood and expressed to the cells where it is needed.

This was determined largely from two lines of investigation. The first resulted from studies of arteriovenous blood samples taken from human volunteers who were subjected to starvation periods of 15 or more hours and then feeding periods.

These studies showed that blood traveling from fatty tissue to the heart during and after starvation is rich in UFA. On its return, the blood in the arteries is conspicuously low in UFA. Thus, the concentration of unesterified fatty acid in blood plasma clearly showed a transport of UFA from adipose tissue to myocardium, skeletal muscle and viscera.

When glucose and insulin are administered to fasting humans, the amount of UFA decreases. The same is true when proteins are given but to a lesser degree. This suggests that the adipose tissue responds to starvation needs of the body through some mechanism sensitive to the bodily balance of non-fat calories.

The second line of investigation, involving a radioactive tracer, led to the timing of the UFA express train from the moment it leaves its adipose tissue depot until its waste is exhaled as carbon dioxide from the lungs. This study showed that in about two minutes the energy from UFA begins to be used by the hungry cells.

The implications of these findings are many. Several national health problems, like obesity and diabetes, are related directly or indirectly to fat metabolism and adipose tissue.

They might be used as a measuring device to find out which drugs affect adipose tissue metabolism and why—thereby leading to some of the answers about how persons become and stay fat.

They promise to be instrumental in the study of diabetes, which is rising in intensity as a degenerative disease, where carbohydrate metabolism is impaired and fat metabolism accelerated. Insulin, for example, is known to affect adipose tissue.

They may be a foot in the door for research on hardening of the arteries.

As one scientist remarked in regard to arteriosclerosis, diabetes and obesity in their relation to unesterified fatty acid and its role during fasting, "fat eaters live off of fat, whether on a diet or not."

(This is the second in a series on research at the National Institutes of Health.)

Science News Letter, August 23, 1958

ZOOLOGY

"Social Pressures" at Zoo Affect Animals' Lives

➤ "SOCIAL PRESSURES" at the zoo have been blamed for the development of arteriosclerosis among some of the animals.

After a 40-year study of birds and mammals that died in the Philadelphia Zoo, two scientists have reported that they found a high frequency of narrowed blood vessels among the caged animals.

Neither aging nor diet seem to bring on the disease as much as "social pressure" resulting from an increased animal population in the zoo.

This "social pressure" or "group relationship" of growing numbers of animals caged as groups in the Zoo caused an imbalance in the secretion of hormones by the adrenal glands, they suggested. Adrenal glands adjust the body to stresses.

The opposite, inactivity due to being caged alone, was suggested as the second factor.

A report on autopsies of more than 11,000 birds and mammals revealed that among the mammals, the greatest incidence of arteriosclerosis was found in the family that includes the South American rodent, the agouti, relative of the guinea pig.

Second highest was the family that includes the kangaroo. The lowest incidence was found to be among the family that includes squirrels.

Among the birds, a higher frequency of arteriosclerosis was found among the larger birds, pheasants, ducks, geese, swans, cockatoos, parrots, macaws, storks, herons, hawks and eagles.

The study was made by Dr. Herbert L. Ratcliffe, director of the Penrose Research Laboratory of the Philadelphia Zoological Society and professor of comparative pathology at the University of Pennsylvania, and Dr. Michael T. I. Cronin, chief of the pathology division of Schering Corporation, and reported to *Circulation* (July).

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