

Current Comments

To Fast or Not Too Much Fast. Part 2. A Controversial Question Gets Some Scientific Answers

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Last week I discussed some of the popular literature on fasting.¹ For anyone who has ever experienced the frustration of dieting without noticeable effect, fasting can seem an almost irresistible alternative. But many fasting enthusiasts also claim that fasts can help cure anything from multiple sclerosis and sterility to asthma and the common cold. Moreover, they credit fasting with lengthening the life span and restoring youth, health, and vitality.

Obviously, such claims only add to the significant credibility gap in this literature. To begin with, the popular fasting community's credibility is greatly weakened by general nonempiricism and wholesale disparagement of modern science and medicine. Many popular fasting publications completely lack conventional scientific documentation.

Fasting's supporters, on the other hand, point out a lack of scientific interest in their work. They note that the subjects in most scientific studies on fasting are allowed vitamin supplements and electrolyte replacement, and many are permitted fruit juices and tea. Such studies may not be relevant if you are trying to define the effects of a fasting diet limited to distilled water. Much legitimate scientific work on fasting has taken place, however.

About the only thing everyone agrees on is that fasting will make you lose weight. According to an article in *Scientific American* by Vernon R. Young and Nevin S. Scrimshaw, both of the Massachusetts Institute of Technology, it was

Walter Lyon Bloom, Piedmont Hospital, Atlanta, and Garfield G. Duncan, University of Pennsylvania School of Medicine, Philadelphia, among orthodox clinicians, who independently pioneered the "modern" study of fasting as a treatment for obesity,² though fasting had been used by medical authorities as a treatment for diabetes as early as the mid-1800s.³ (More on this later.)

In his 1959 study of nine obese patients who had fasted for periods ranging from four to nine days, Bloom found that upon the patients' resumption of normal eating, a 600-800 calorie per day diet produced satiety. Prior to fasting, this same amount had proven unsatisfactory. In addition, Bloom noted that in follow-up studies, patients had maintained a large part of their weight loss and, in most cases, had continued to lose weight.⁴

In the early-1960s, Duncan treated more than 1,300 obese patients at Pennsylvania Hospital, Philadelphia. Each patient fasted for periods ranging from ten days to two weeks, with patients returning for repeated fasts at varying intervals. In 1965, he reported his observations in *Postgraduate Medicine*. He concluded that intermittent fasting could be an effective temporary therapy for the obese patient whose condition was unresponsive to other forms of treatment. About half of the 890 patients in his series of studies continued to lose weight following their initial fast.⁵ Incidentally, the use of fasting as a

treatment for obesity as Duncan advanced it is still being carried on at Pennsylvania Hospital (on a much smaller scale) by his son, Theodore G. Duncan, who, like his father, is also associated with the University of Pennsylvania School of Medicine.

Favorable conclusions were reached by other investigators as well. A 1964 editorial in the *Journal of the American Medical Association (JAMA)* concluded that fasting seemed to help establish normal eating patterns in cases where nothing else worked.⁶ A 1962 article in *Medical World News* reported one investigator's remark that fasting helped break the "metabolic block" that encouraged people to overeat.⁷ And, in 1966, in an American Medical Association convention report in *JAMA*, US Air Force physician Robert M. Karns commented that in an Air Force study of 25 patients, fasting seemed to result in a "true loss of fatty tissue."⁸

Karns's study, however, was never published, and many further studies have failed to confirm that fasting is an effective therapy for the treatment of obesity. A relatively recent paper in the authoritative *Nutrition Reviews*, published by the Nutrition Foundation, reported that subjects did not change their eating habits following a fast, nor could they maintain their weight loss.⁹ (Incidentally, articles in *Nutrition Reviews* are normally written by a contributing editor, and are then reviewed by an associate editor as well as the editor.) An earlier study in 1969 had reported virtually the same results.¹⁰ So fasting without some accompanying behavior modification may be pointless.

Still another study, conducted by Jules Hirsch, professor and senior physician, Rockefeller University, suggested that fasting did not diminish the number of fat cells—they all simply shrank in size.¹¹ This is in keeping with the suggestion, presented in a 1978 review in the *American Journal of Public*

Health, that obesity's intractability to therapy is, perhaps, a biological inevitability.¹² This is because, as the hypothesis goes, obesity may occur in two predominant forms: hypertrophic obesity, in which there is a normal amount of overfilled fat cells in the body, and hyperplastic obesity, in which there is an excess of normal-sized fat cells. It is the latter condition that is thought to be partially genetic in origin. Since it appears certain that fat cells, once generated, are not destroyed—except in cases of severe starvation—loss of weight can only occur through depleting the fat cells of their reserves of fat. But this would drive the organism to eat more, in order to fill the depleted fat cells. Thus, for the hyperplastically obese, obesity may be the body's "normal" state. Lending credence to this hypothesis is a 1966 US Army study by M. Mager and P.F. Iampietro, which found that following a fast, the body compensates for its depleted fat reserves with an acceleration in the conversion of calories to fat.¹³

Can fasting be harmful? One 1965 study links fasting with the formation of gastric ulcers in mice.¹⁴ More ominous, however, were two case reports in *Lancet* in 1968 concerning obese patients who had died while undergoing fasting therapy.^{15,16} In another case, an article in *Lancet* discussed an autopsy's findings that a significant portion of the heart muscle of an otherwise healthy obese patient had been consumed during the fast.¹⁷ Some fasting enthusiasts claim that during a fast, the body consumes old, worn-out tissue before vital, healthy cells. But it is very doubtful whether the body is that selective about where it gets the protein to metabolize during a fast.¹⁸⁻²³ However, Joy Gross, director of the Pawling Health Manor, Hyde Park, New York, claims that it would be totally irresponsible to allow a prolonged fast without proper monitoring.²⁴

The subject of sudden death occurring during a fast calls to mind the recent liquid protein diet fad, which was responsible for 58 deaths as of June 1978.²⁵ The liquid protein used in this diet is seriously deficient in such important nutrients as potassium, phosphorus, magnesium, and essential amino acids.²⁶ To subsist on liquid protein alone is dangerous indeed.^{27,28} Incidentally, the liquid protein diet should not be confused with the protein-sparing modified fast (PSMF) propounded by George L. Blackburn and Bruce R. Bistrian, Harvard Medical School.²⁰ The PSMF is designed to maximize fat loss while providing essential nutrients. It consists of eating minimal amounts of such protein-rich foods as meat and fish, and of taking daily supplements of vitamins, potassium, calcium, and other minerals. But patients on PSMF must follow the prescribed regimen. One case report describes a PSMF patient who neglected to take the mineral supplements and suffered a nonfatal heart attack.²⁹

However, it should be noted here that even fasting's staunchest champions do not recommend the practice for everyone. They emphasize that anyone with diabetes, heart disease, epilepsy, or ulcerative colitis, or anyone taking medication for any reason, should fast only after consulting their doctor, and even then, only under the strictest supervision.³⁰ In fasting, ketosis is always a potential danger. Ketone bodies are by-products of the metabolism of fatty acids by the liver.²³ They are highly acidic. During a fast, increased amounts of fatty acids are broken down by the body to compensate for the lack of food.²¹⁻²³ Thus, ketone bodies accumulate in the blood. The resulting acidosis can produce coma or even death.³¹ In addition, fasting may lead to hyperuricemia, or increased levels of uric acid in the blood.³² In some individuals, prolonged deprivation may lead to an at-

tack of gout, a condition in which uric acid levels become so high that the acid crystallizes out of the blood, especially in the areas surrounding the joints.³² However, Gross and others claim that careful monitoring would prevent anyone from abusing the fasting regimen in this way.

Some of fasting's advocates claim that fasting has rejuvenating effects. Aging research is currently split between a number of theories on the etiology of aging. But none of those theories under serious consideration today are concerned with the accumulation of cellular waste products or toxins as a cause of aging.^{33,34} Alexis Carrel's conclusion that cells are virtually immortal if kept free of their own wastes, hailed by fasting enthusiasts as proof of their claims, has been discredited by the classic work of L. Hayflick and P.S. Moorhead.³⁵ They found that cultured human cells undergo a finite number of population doublings, then die. In a more recent paper, presented at the 62nd Annual Meeting of the Federation of American Societies for Experimental Biology, Hayflick reported that these observations have been confirmed time and again in hundreds of laboratories throughout the world.³⁶

Popular authors like Gross and Herbert M. Shelton, who ran his own fasting institution for over 50 years, claim that fasting and the elimination of "stressful foods" (like meat and most dairy products) from the diet will increase human life spans. Such claims lean heavily on the work of Clive M. McCay, then of Cornell University. He did a series of experiments in 1939 which restricted carbohydrate intake by rats.³⁷ Popular fasting advocates like Gross and Shelton claim that McCay's studies prove that a rich diet will kill you prematurely, and that a diet restricted in the type of calories consumed, combined with periodic fasting, will help prolong your life.^{38,39}

In none of McCay's studies, however, did he find any evidence that the life spans of normal animals were shortened due to richness of diet, nor did he apply his findings to human beings. In fact, a 1978 article in *Geriatrics* specifically warns against applying the results of studies using laboratory animals directly to humans.⁴⁰ A 1977 article in *Nutrition Reviews* by Morris H. Ross, senior member, Institute for Cancer Research, Philadelphia, cautions that despite the increase in longevity that is evident in animals maintained on severely limited food rations, it is not safe to assume that these benefits would necessarily result outside the protective confines of the laboratory.⁴¹ Ross points out that under natural conditions, a prolonged period of drastic undernutrition (such as is used in these types of studies) could produce impairment of physical development, and may lead to a greater risk of cancer.

Finally, in a 1952 review of the effects of diet on aging, McCay himself stated: "The type of calories [consumed] seemed to matter little in regard to span of life.... The degree of body fatness... is more important than such variables as protein or exercise."⁴² (p. 147, 154) Thus, the scientific literature rejects the claim that fasting prevents aging. What the popular fasting community may be trying to say, however, is that fasting may prevent *premature* aging. Even if human beings have a genetically programmed maximum life span, bad living could certainly reduce it. A healthful life-style may let you live to your full potential.

And indeed, there is some recent evidence that certain dietary restrictions *do* positively affect longevity. G. Fernandes and E.J. Yunis, University of Minnesota, and R.A. Good, Memorial Sloan-Kettering Cancer Center, demonstrated that the life span of one breed of mouse was lengthened by simple caloric restrictions, while the longevity of

another breed was extended by a reduction in protein intake.⁴³ Both of these dietary manipulations apparently delayed the advance of autoimmune disease, in which the body's natural defenses against invading foreign matter turn against the body itself.

One area of research that does seem to hold promise for humans concerns fasting and the treatment of diabetes. In fact, the only research front in our *ISI/BIOMED*™ system that deals with fasting concerns fasting and diabetes. That research front is entitled *Effects of Fasting on Insulin Action in Adipocytes*.

Briefly, diabetes mellitus is a disorder in the synthesis, secretion, or function of the hormone insulin, which regulates the metabolism of carbohydrates in the body.³¹ In one variety of diabetes, called "maturity-onset," or "non-insulin dependent," a person is genetically predisposed toward abnormalities in the way insulin interacts with the receptors on target cells. In people thus predisposed, obesity can trigger the latent condition into an overt disease. Overeating causes the pancreas to produce constantly high levels of insulin. This in turn makes the target receptors resistant to insulin. The result is that cells cannot assimilate carbohydrates properly, despite an abundance of insulin.

It has been common practice since the 1950s to treat all forms of diabetes with insulin and oral antidiabetic drugs.³ But for the non-insulin dependent diabetic, such treatment may only serve to make target cells even more insulin-resistant. So in 1971, John K. Davidson, Emory University School of Medicine's Grady Memorial Hospital, stopped prescribing insulin and antidiabetic drugs for obese maturity-onset diabetics, instituting instead a program of week-long fasts and aggressive diet therapy.^{44,45} He found that in 60 percent of the patients, symptoms were satisfactorily controlled, while the rest re-

quired only much-reduced doses of insulin.⁴⁶ Since that initial study, Davidson and his colleagues have treated over 5,000 individuals using a combination of fasting and diet.⁴⁷

Although science has failed to validate any of the claims of popular fasting enthusiasts and natural hygienists, I myself feel better when I fast, and I think fasting does me good. This is, of course, subjective. But there are numerous anecdotal accounts in the scholarly fasting literature of individuals who seemed to spontaneously recover from an illness during a fast—even though medical science had reached its limits in treating them. Scientists may argue that these recoveries are due to remissions that would have happened anyway, or that they show the power of the mind over the body—the so-called placebo effect. They may be right on both counts. And in fact, Norman Cousins, editor emeritus of *Saturday Review* and now adjunct professor, department of psychiatry and biobehavioral science, UCLA School of Medicine, in his book *Anatomy of an Illness as Perceived by the Patient*,⁴⁸ tells how he conquered a supposedly fatal illness through good humor and positive thinking, proving—in anecdotal fashion at least—that the interaction of mind and body can baffle even the best of physicians.

But neither position is tenable without large-scale evidence. Unfortunately, as in so many other areas of medicine, we have many more questions than answers. If the National Institutes of Health or other US research agencies are not motivated to study fasting in the sense that fasting enthusiasts use the term, we might argue that it would be in the interest of the World Health Organization to do so. If Americans and other over-consuming peoples could be convinced that fasting (or eating less) increased the life span and the quality of life, then we presumably could better

help feed other, less fortunate countries. But this may be a simplistic view of the world food problem. The nature of this problem has been described in detail in a book recently published by the International Institute for Applied Systems Analysis.⁴⁹

For a proper discussion of fasting, a distinction between short-term and long-term fasting is needed. I consider myself an advocate of short-term fasting. Oddly enough, though, some of the thinnest people I know, easily classified as light eaters, find it difficult to go more than three or four hours without some nourishment. Thus, while I can easily go through an entire day without eating (especially if my secretary does not remind me about lunch), many of my colleagues cannot. Some need morning coffee with plenty of cream and sugar, others need a light snack, and still others a complete meal. Clearly, we are all different. The reasons for that may be cultural, environmental, genetic, or psychological.

My own experiment with fasting proved uneventful (if you could say that losing ten pounds in five days is uneventful). At least there were no apparent side effects, other than occasional hunger pangs. I did not have the slightest problem reading or writing. I was encouraged to eat an orange or drink some juice, but did not feel the need. My son Stefan, who is significantly overweight, spent most of his time sleeping or watching TV. He refused to go to the cinema with me, lest he be tempted to stop at a hamburger stand or eat popcorn. He had experienced many days of "fasting" in Vietnam, so it was no surprise that the effects of this fast on him were minimal. It would seem, however, for certain people, that fasting is the only effective way to reduce or keep trim, simply because there is no easy way to control the amount you eat once you start. Of course, Gross and other fasting enthusiasts hope that your

fasting experiment, combined with an introduction to vegetarianism, will start you on a new path—a new set of eating habits. Whether or not such behavior modification can be accomplished by most people in this way is yet to be determined.

Like so many other subjects I have covered, the ultimate answer is moderation. You probably don't need fasting if you are one of those lucky people who is able to combine moderate exercise with moderate food intake. Presumably, it would be better if your diet contained a significant amount of high-fiber and low-fat food, and a minimum of animal protein.⁵⁰ Indeed, a recent article in

JAMA showed that the addition of only nine ounces of lean beef to the diets of vegetarians produced a rapid and significant increase in blood cholesterol levels.⁵¹ A smaller but still significant rise in blood pressure was also measured. But if I am unable to resist a big Thanksgiving Day dinner, or an occasional trip to the local ice cream parlor, an occasional fast and/or some vigorous exercise does me good.

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