Medical Hypotheses

Medical Hypotheses (1995) 44, 403–405

Pantothenic Acid as a Weight-Reducing Agent: Fasting Without Hunger, Weakness and Ketosis

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Abstract — With the conventional method of fasting or aggressive dieting to reduce excess body fat, hunger, weakness, ketogenesis and ketosis are the sequential events that follow. It is not fully understood why, under conditions of negative calorie balance where complete energy release from storage fat is critical, ketosis should arise with a concomitant wastage of energy. Here, I wish to propose a theory that relates the formation of ketone bodies under such conditions to a deficiency in dietary pantothenic acid. Supplementation of this vitamin would facilitate complete catabolism of fatty acids and thus the formation of ketone bodies could be circumvented. As a result, a sufficient amount of energy would be released from storage fat to relieve dieters of the sensation of hunger and weakness which otherwise would be difficult to endure. Hence, using this method for weight reduction together with a careful observation of calorie intake, I have great success in treating overweight-to-obese patients to lose weight.

Introduction

The battle against excessive body weight has been waged for decades, if not centuries. So far, it is a losing battle. The most widely used method for weight reduction is to achieve a negative calorie balance mainly through dieting. This sounds rather simple. However, in practice the failure rate is very high. With a reduced calorie intake, a dieter will invariably experience feelings of hunger and weakness. To relieve such discomfort, dieters would return to their old eating habits and regain the weight they have tried so hard to lose.

Biochemically, with a negative calorie balance, the limited glycogen store would be first mobilized to provide for energy deficit, and depot fat would be called upon for this function as well. However, the human body appears to have only a restricted capacity to fully metabolize its own storage fat, since total starvation for 2–3 days or prolonged dieting always leads to ketosis, which is the accumulation of ketone bodies in blood and their excretion in urine and expired breath. This represents, in the very least, a loss of the precious energy reserved for supposedly needy times, such as famine. However, it is interesting to note that the onset of ketosis during fasting is different among individuals under identical conditions; and the common laboratory animals develop very little ketosis, if any, during a fast of 2–5 days (1,2). This strongly suggests that if depot fat can be efficiently burnt off to provide for the energy deficit brought about by dieting, the onset and onslaught of ketosis could be postponed indefinitely or even prevented. It appears, then, that the solution to successful weight reduction might be resolved from a biochemical standpoint. To explain how this hypothesis is formulated, a brief recapitulation of fat catabolism is necessary (3,4).

Date received 14 September 1994
Date accepted 24 October 1994

Fat catabolism

It is well known that in fat catabolism, depot fat stored in the form of triglycerides is first broken down into long-chain fatty acids and glycerol. Then, through the process of β-oxidation, long-chain fatty acids are fragmented into units of acetyl-Co A which are finally metabolized via the citric acid cycle, releasing energy in the form of ATP with carbon dioxide and water as end products. For example, in the case of palmitic acid, a 16-carbon saturated fatty acid, it is first fragmented to 8 units of acetyl-Co A with an energy production of 35 ATP. It is of interest to note that this process of fragmentation requires the participation of 8 units of Coenzyme A. In turn, these 8 units of acetyl-Co A are completely metabolized via the citric acid cycle producing 96 ATP. However, under fasting conditions, ketone bodies are formed as a result of the condensation of 2 units of acetyl-Co A giving rise to acetoacetyl-Co A and a molecule of Coenzyme A, with the former being rapidly converted into acetoacetic acid (the parent compound of ketone bodies) and another molecule of Coenzyme A. The human body would try to offset the acidosis thus produced by eliminating a proportion of the ketone bodies so formed through expired air and urine. Hence, long-chain fatty acids are only partially metabolized under such conditions, resulting in a reduced liberation of the ATP initially stored in storage fat.

It is not well understood why ketone bodies should be formed under such circumstances when there is a shortage of energy supply. Here, I wish to suggest a theory for their formation under these conditions and a way to circumvent their synthesis. The solution arising thereof has the power of alleviating the sensation of hunger and weakness experienced by dieters during dieting.

The role of pantothenic acid

The formation of a ketone body with the liberation of two molecules of Coenzyme A is likely to be a mechanism to conserve Coenzyme A during conditions when there is an increased demand for this compound to participate in the continual fragmentation of long-chain fatty acids to release stored energy. If there were a plentiful supply of pantothenic acid in the body, there would not be a shortage of Coenzyme A and the condensation of acetyl-Co A units for its production would no longer be necessary. As a result, ketone bodies would not be formed and acetyl-Co A molecules would enter the citric acid cycle directly and be completely metabolized. Thereby, sufficient energy would be generated to compensate for the energy deficit brought about by a low calorie intake. Consequently, the dieters' feeling of hunger and weakness would be assuaged.

With this hypothesis in mind, my method of increasing energy yield and circumventing ketogenesis during periods of low calorie intake is to supplement dieters with a massive dose of pantothenic acid daily. This treatment is coupled with a carefully-designed diet, containing all the essential nutrients, and with an intake of about 1000 calories a day. Dieters were found to achieve a steady and gradual weight loss of about 1 kg per week as described in the following study.

A clinical study was carried out in 100 individuals of Chinese descent, 40 males and 60 females. The age range was 15–55, with even distribution. Dieters were given orally 2.5 g of pantothenic acid 4 times a day. These dieters' goals varied, which range from losing less than 5 kg to more than 30 kg. Approximately half of them aimed at shedding about 10–20 kg. The average weight loss was found to be 1.2 kg per week. Ketone bodies in urine were monitored regularly and were found to be absent in most circumstances. In a minority of cases, the presence of ketone bodies were detected in trace to small amounts. The dieters did not complain of hunger nor weakness and all, without exception, could carry on their daily routines normally. In fact, many even reported an improved sense of well-being. The explanation for this probably lies in the fact that Coenzyme A, straddling the crossroads of the pathways of carbohydrate, fat and protein metabolisms, plays a key role in the biosynthesis of many lipids and steroids and performs a myriad of functions. When the body's demand for pantothenic acid is met, it may then function in an optimal state. As a result, the individual concerned may feel better.

There are several points worthy of note with the present method of weight reduction:

1. No side-effects have been observed in patients administered pantothenic acid at the present dose level (10 g/day), because the compound is a water-soluble vitamin (5);
2. Restriction of fluid intake is not necessary, and urinary output from dieters actually increased – part of which may represent the end product of fatty acid metabolism, aside from carbon dioxide;
3. Exercise, which is stressed so much in conventional weight reduction methods, is not required here, though exercise would certainly help to burn off storage fat faster;
4. Pantothenic acid is not an appetite suppressant. On the other hand, it may even be called a hunger suppressant when taken at a large dose during

periods of low calorie intake. This may be accounted for by the fact that it renders the mobilization and release of energy from fat depot more efficiently.

It seems that the availability of pantothenic acid is of overriding importance in determining an individual's readiness to put on weight. When an individual has a deficiency in this compound, his or her capacity to utilize fat is curtailed but the ability to store fat is retained, hence such individuals are more inclined to put on weight.

It appears that pantothenic acid is the key to unlocking and metabolizing depot fat. If an ample supply of pantothenic acid can be maintained in the body, conversion of fat into energy will always be efficient. Hence, the present method also solves the difficult problem of maintaining one's desired body weight once adequate weight reduction has been achieved. Depending on the biochemical variability of an individual, I found that a maintenance dose of 1–3 g, together with a careful diet, is all that is needed to maintain one's body weight.

References

1. Folin O, Denis W. On starvation and obesity, with special reference to acidosis. J Biol Chem 1915; 21: 183–192.
2. Fery F, Balasse E O. Ketone body turnover during and after exercise in overnight-fasted and starved humans. Am J Physiol 1983; 245: E318–325.
3. Greville G D, Tubbs P K. The catabolism of long-chain fatty acids in mammalian tissues. Essays Biochem 1968; 4: 155–212.
4. Stumpf P K. Metabolism of fatty acids. Ann Rev Biochem 1969; 38: 159–212.
5. Ralli E P, Dumm M E. Relation of pantothenic acid to adrenal cortical function. Vitam Horm 1953; 11: 133–158.