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EXPERT OPINION

Effects of various forms of food and fluid deprivation upon physical performance.

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Abstract

Background: Physical performance can be threatened by short periods of total fasting, by longer periods of severe dietary restriction and by dehydration. *Purpose*: This article reviews the extent of such effects as seen in athletes and others who are dieting for various reasons. *Methods*: A comprehensive review of the literature for papers examining physical performance in relation to fasting, dietary restriction and dehydration. *Results*: Functional losses with fasting are less than might be imagined, and (given the smaller effects that are seen in experimental animals) loss of motivation may well account for some of the observed negative effects (decreases in maximal oxygen intake, sub-maximal aerobic endurance and muscular strength) in tests requiring maximal effort from the subject. Two factors contribute to the limited effects of food deprivation: the decrease of body mass reduces the energy cost of performing tasks where the body mass is displaced, and the body has a surprising ability to make long-term adaptations to the metabolism of stored fat and tissue protein; heavy daily energy expenditures can be sustained on a fat/protein diet, despite ketosis. Fluid stores are smaller in relation to the demands of vigorous exercise, and many aspects of physical performance are impaired with dehydration equivalent to 2-5% of body mass. *Conclusions*: Although effects are relatively small, athletes who seek optimal performance should counter a deficiency of food or fluid as soon as possible. With careful management of lifestyle, performance can generally be maintained during the intermittent fasting of Ramadan, although this is probably not true for ultra-endurance events. **Health & Fitness Journal of Canada 2014;7(3):18-41.**

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Introduction

With the growing numbers of observant Muslims in North America, sports scientists, coaches, and trainers are increasingly confronted with the need to advise athletes who wish to observe Ramadan, the 29-day annual celebration when neither food nor fluid is ingested between sunrise and sunset (Bouhlel et al., 2013; Shephard, 2012a, b; 2013). However, the observance of Ramadan is but one of many circumstances where a person attempts to perform physical activity with a less than optimal supply of food and/or fluids. Other possible scenarios include becoming lost in the wilderness or marooned at sea, in addition to deliberate attempts by an athlete to reduce body mass (as when "making weight") or to alter body form (as in competitive gymnasts). Possible situations include short or longer periods of total fasting, chronic food deprivation, a lack of one or more key dietary constituents, and dehydration.

This article focuses upon body reserves of food and fluid, considering the limits of human adaptation to deficiencies of food and fluids, comparing and contrasting responses with those seen in laboratory animals. It examines the effects of prolonged starvation, briefer periods of fasting, severe food deficiencies and fluid deprivation upon

human performance, and concludes with a brief examination of the potential problem of potential adjustments to the metabolic ketosis that may arise from fasting.

Potential fasting scenarios

Total fasting may be imposed experimentally in the metabolic ward of a hospital in order to examine details of physiological responses. It may also occur by misadventure, if a party of mountaineers, arctic explorers or shipwrecked mariners find themselves in a situation where rigorous exercise is needed to escape from a dangerous situation, but supplies of food and/or water have been exhausted. Finally, a period of full or partial fasting may be required by the tenets of a specific religious belief (for example, the repeated abstinence from both foods and fluids expected during daylight hours throughout the month of Ramadan).

Severe food deprivation may occur in times of war and famine. It may also be imposed experimentally, in order to examine the physiological consequences of famine or of over-rigorous dieting in an attempt to control obesity. Some classes of athlete choose to impose upon themselves severe dietary and/or fluid restrictions in an attempt to qualify for a particular category in a weight-classified sport, or in the quest for a more attractive physical appearance in a subjectively rated competition such as gymnastics or figure skating. In the general population, an obese and sedentary middle-aged person may adopt a very low energy diet in order to reduce the body fat burden. The daily food intake may also be severely restricted by a variety of pathologies such as anorexia nervosa, bulimia and gastro-intestinal disorders. Finally, poverty or extreme vegetarianism may lead to a deficiency of one or more key nutrients, particularly essential amino acids and trace elements.

The primary nutritional deficiency may be a lack of water, rather than food. Dehydration can arise as a sequel to a shipwreck or becoming lost in the desert. It is also seen when prolonged exercise is undertaken under very hot conditions. However, perhaps most commonly it is induced deliberately by an athlete in an attempt to achieve a lower weight category than is appropriate to his or her body build.

Body reserves of food and fluid

The daily energy consumption of an adult ranges from about 8 MJ in a sedentary individual, to 16 MJ or more for many athletes (FAO/WHO/UNU, 2004). When the energy intake does not match this expenditure, the body draws upon its stores of carbohydrate, fat, and protein to cover the deficiency.

Carbohydrate

The carbohydrate reserves of the body are quite limited. In addition to small amounts of glucose in the plasma and tissues, glycogen is stored in the muscle (normally about 400 g) and in the liver (about 100 g), giving a total reserve equivalent to some 8 MJ of energy (Shephard, 1982). Carbohydrate reserves can be increased by at least 50% if the person concerned adopts a high carbohydrate diet (Hawley et al., 1997).

The rate of depletion of carbohydrate reserves depends not only on the daily energy expenditure, but also its nature. During moderate intensity rhythmic activity, much of the energy requirement is met from the metabolism of fat; moreover, a higher proportion of energy

is derived from fat in an endurance athlete than in a sedentary individual. At an intensity of effort demanding more than 70% of maximal oxygen intake (80-90% in an endurance athlete), a large and growing fraction of the total energy requirement is met from the metabolism of carbohydrates (Gmada et al., 2012). The energy required for vigorous resisted muscular contractions, also, is derived almost exclusively from carbohydrate (Shephard, 1982).

A physically active person is likely to deplete their carbohydrate stores within 24 hr of commencing a total fast. Any subsequent need for carbohydrate is met by the formation of glucose in the liver, the process of hepatic gluconeogenesis. Such glucose formation depends mainly upon the breakdown of tissue protein and the metabolism of circulating amino acids, although the process is supplemented by the use of available glycerol and lactate (Shephard, 1982).

Fat

Fat stores provide the body's main long-term reserve of energy. In sedentary North Americans, initial stores of fat are often quite large. An obese individual may store 20-30 kg of fat. If there is a major reduction in body fat stores during a period of fasting or dieting, there is a roughly corresponding decrease in body mass (Shephard, 1991); this reduces the energy cost of most tasks that require the displacement of body mass (Brown, 1966), although because of a loss of sub-cutaneous fat, a person also becomes more susceptible to heat loss in a very cold environment.

If the customary pattern of physical activity were to be maintained during fasting, then energy needs would amount to perhaps 6 $MJ \cdot d^{-1}$ in a totally inactive person, but to at least 10-12 $MJ \cdot d^{-1}$ in most athletes. The energy yield of body fat is about 29 $kJ \cdot g^{-1}$, so if a person began with 15 kg of surplus fat, this could provide a total reserve of some 435 MJ of energy, sufficient to sustain the energy needs even of an active individual for a month or longer (Shephard, 1982). However, the tolerance of fasting would be much shorter in a typical e athlete, who would begin any period of food deprivation with much smaller reserves of body fat, and would face much higher daily energy demands.

Protein

The body has a labile reserve of some 300 g of protein (Swick and Benevenga, 1977), much of which is found in the liver. Over the first five days of starvation, this provides the main basis for the synthesis of glucose, at a rate of about 60 $g \cdot d^{-1}$. Thereafter, the main resource for continued hepatic gluconeogenesis is a progressive breakdown of muscle tissue. The American Heart Association has argued that unless dieters consume a minimum of 100 g of carbohydrate per day, a loss of tissue protein will be inevitable (St. Jeor et al. 2001). Unfortunately, about 1.6 g of protein is needed to provide the equivalent of 1g of glucose (Manninen, 2004), and the issue is further complicated in that not all amino acids can contribute to the synthesis of glucose.

The total muscle mass varies widely between a strength athlete, an endurance performer, and a sedentary adult. However, a typical muscle mass for a sedentary man would be about 35 kg (Shephard, 1991; Kim et al., 2004). A progressive breakdown of 100 g of lean tissue per day would provide a little over 1 MJ of energy per day, but would reduce

lean mass by almost a third over 100 d of food shortage. The lack of circulating amino acids would also limit any local response to strength training (Rasmussen et al., 2000; Tipton et al., 2001; Hawley et al., 2007). It seems likely that the loss of lean tissue would lead to a progressive decrease in muscular strength. Because of loss of plasma protein, tissue oedema might also develop, together with a natriuresis (excretion of sodium) and a secondary loss of potassium from the body (Phinney et al., 1980).

Onset of metabolic ketosis

The classic dictum of the biochemist was that "*fat burns in the flame of carbohydrate metabolism*." In the absence of sufficient carbohydrate metabolism, body fats are partially broken down to ketone bodies, and these cannot be metabolized in the liver because it lacks the necessary mitochondrial enzyme, succinyl CoA:3-ketoacid CoA transferase. Ketone bodies thus accumulate in the bloodstream, and by analogy with diabetic ketosis (where fat metabolism is increased because of a lack of insulin, and there is a massive and uncontrolled production of acidic ketone bodies), there was a fear that the end-result of an inadequate carbohydrate intake in an active individual would also be a fatal ketotic coma.

During the past 10 years, this view has been criticized (Manninen, 2004). It has been emphasized that an active person can maintain a normal blood pH during a period of carbohydrate restriction, and that ketone bodies can provide a useful substrate for cerebral metabolism, thus sparing any glucose in the blood stream. Indeed, both 3-hydroxybutyrate and amino-acetate yield somewhat more ATP per molecule than an equivalent amount of glucose. Moreover, the muscles can metabolize at least seven amino acids directly. It thus may be more correct to claim that "*fat and carbohydrate burn in the flame of protein metabolism*" (Manninen, 2004).

Assuming that the energy intake is 6 $MJ \cdot d^{-1}$ less than the energy requirement, this deficit could be covered by metabolizing a combination of 150 g of stored fat and 100 g of tissue protein. Ketosis will develop if fats are metabolized in the absence of a substantial quantity of carbohydrate. If this leads to an acidosis, it can impair athletic performance, but this is not invariably the case (Phinney, 2004). As discussed further below, with time, the body seems to adapt to exercising on a high fat/low carbohydrate diet.

Fluid

The body normally contains 45-50 L of water (Shephard, 1982). The maximal safe loss depends on circumstances, including the amount of exercise that must be performed, whether water has been liberated by the metabolism of intramuscular glycogen (a potential reserve of up to 2L), and environmental conditions (Shephard and Kavanagh, 1975). If a person is performing endurance exercise in a temperate climate, a loss of 5-6 L seems to be quite well tolerated (Shephard and Kavanagh, 1975).

Under resting conditions, normal fluid losses from the body amount to about 2.5 $L \cdot d^{-1}$ (a urine flow of 1.2-1.5 L, water loss at the skin surface of 0.5 $L \cdot d^{-1}$, respiratory losses of 0.4 $L \cdot d^{-1}$, and the water content of the feces of about 0.15-0.2 $L \cdot d^{-1}$)(Shephard, 1982). However, if exercise is performed in the heat an endurance athlete may lose an additional 2 $L \cdot h^{-1}$ or

more by sweating, and there is a rapid deterioration of function if body fluid reserves cannot be replenished (Shephard, 1982).

Total fasting

Studies of the impact of total fasting upon physical performance have been relatively few, although several papers

Table 1: The influence of short- and medium-term total starvation upon physical performance.

Author	Participants	Fast Length	Exercise intensity	Change with fasting
Short-term fasting (12-36 hr)				
Dohm et al. (1983)	Male Holtzman rats	24 hr	Treadmill, 28 m·min^{-1} 0% grade	80% increase in time to exhaustion with fast
Loy et al. (1986)	10 young competitive male cyclists	24 hr	Cycle ergometry at 86% or 79% of maximal oxygen intake	73.5% decrease at 86%, 25.7% decrease at 79%
Dohm et al. (1986)	9 healthy active young males	23 hr	Treadmill running at 70% of maximal oxygen intake	7.7% decrease when fasting; increased perception of exertion at 60 min
Gleeson et al. (1988)	6 healthy young males	24 hr	Cycle ergometry at 100% of maximal oxygen intake	13% decrease when fasting
Maughan & Gleeson (1988)	5 healthy males	36 hr	Cycle ergometry at 70% of maximal oxygen intake	35% decrease when fasting
Johnson & Leek (2010)	22 healthy young women	12 hr	Reach and balance	Deterioration when fasting
Medium-term fasting (2.5-10 d)				
Uyeno & Graham (1985)	Rats	3 d	Swimming time	Increased after fast
Young (1959)	Dogs	5 d	Treadmill running	Time to exhaustion increased with fasting
Gutiérez et al. (2001)	8 healthy young males	3 d	$PWC_{170,}$ Handgrip force and perception reaction time	Decrease of PWC170 (23-28%) but no change in other variables
Knapik et al. (1987)	8 healthy young men	3.5 d	Cycle ergometry at 45% of maximal aerobic power	No change
Knapik et al. (1987)	8 healthy young men	3.5 d	Isokinetic and isometric strength	No change of isometric strength or anaerobic capacity, but small decreases of isokinetic strength (11, 8.6% at 2 speeds of rotation)
Lastegola (1965)	6 healthy men	7 d	Treadmill maximal oxygen intake	31.6% decrease (less marked loss at 10 d of fasting)
Consolazio et al. (1967)	6 healthy men	9 d	Handgrip force	9.3% decrease

have reported responses to short-term (12-36 hr) and longer (3-10 d) periods of total food deprivation. Some investigators have suggested that fasting might actually improve endurance performance, in part because the resting plasma fatty acid concentration is increased (Conlee et al., 1976), thus sparing the remaining glycogen reserves (Costill et al., 1977; Hickson et al., 1977); in part also because a decrease of body fat reduces body mass and thus the energy cost of displacing both the limbs and the entire body (Brown, 1966). If there is any such benefit, it seems more apparent in laboratory animals than in human subjects.

Short-term fasting (12-36 hr)

Over the first 12-36 hr of fasting, much of the energy needed by a person who is resting is found from a depletion of glycogen reserves, and there is a relatively limited usage of protein and fat stores. The early decrease of body mass is also relatively small, reflecting the metabolism of <1 kg of stored food, and the loss of up to 2 kg of body water associated with stored molecules of glycogen (Shephard, 1982). At this stage, a decrease of blood glucose may impair cerebral function and have a negative effect upon motivation. Moreover, the depletion of glycogen reserves is likely to have an adverse effect upon sustained or repeated muscular contractions and the endurance of sustained sub-maximal aerobic activity.

Empirical observations during short-term fasting have looked mainly at changes in the endurance of sub-maximal aerobic exercise (Table 1). There is less likelihood of a decrease in motivation due to a decrease in blood glucose levels in animals than in humans. Dohm et al. (1983) observed a dramatic improvement in the treadmill endurance of rats after they had undergone a 24-hour fast. An 18% decrease in body mass in the fasted animals was associated with an increase of the time to exhaustion when running on a treadmill; 284 min, as compared with 158 min for the control animals. The augmentation of endurance performance was judged too large to attribute simply to the decrease in body mass. Dohm et al. (1983) suggested that glycogen sparing from an increased level of circulating fatty acids might also have been a contributing factor. The earlier data of Uyeno and Graham (1965) support these findings; they showed an increased swimming time in fasted rats; gains were seen within 6 hr of food deprivation, and surprisingly were still present after 3 d without food.

In contrast with these animal experiments, human data have consistently shown a negative effect of fasting upon endurance performance, possible contributing factors including a loss of motivation and a decrease in the plasma alkaline reserve. Loy et al. (1986) tested competitive cyclists at 79% or 86% of their maximal oxygen intake. Initial muscle glycogen stores were unchanged by 24 h of fasting, but plasma free fatty acid levels were increased, thus potentially sparing carbohydrate usage during the test exercise. Nevertheless, the time over which the required pedal cadence could be maintained was drastically shortened with fasting, from 115 to 42 min at 86% of maximal aerobic power, and from 191 to 142 min at 79% of maximal aerobic power.

Dohm et al. (1986) tested 9 well-conditioned young men after 23 hr of total fasting. They noted that this period of food deprivation was associated with a small decrease (7.7%) in the duration of

treadmill running at 70% of maximal oxygen intake, with an increased rating of perceived exertion after 60 min of running, and increased plasma levels of free fatty acids and beta-hydroxybutyrate. Under fasting conditions, the respiratory gas exchange ratio indicated a substantial increase of fat usage during the first half of the sub-maximal exercise period.

Gleeson et al. (1988) compared times to exhaustion in 6 young men pedalling a cycle ergometer at 100% of their personal maximal oxygen intake; times were significantly shorter after a 24-hour fast (212 s.) than when they were eating a normal diet (243 s). During fasting, the pre-exercise bicarbonate and base excess were also reduced, and blood levels of glycerol, free fatty acids and beta-hydroxybutyrate were increased. The deterioration of performance was thus thought due mainly to a worsening of the pre-exercise acid/base status during fasting (Greenhaff et al., 1987a, b), although there may also have been some effects from a decrease of motivation.

A 36-hour fast (Maughan and Gleeson, 1988) reduced the cycle ergometer endurance time at 70% of maximal oxygen intake from 120 to 78 min, despite increased plasma levels of free fatty acids. None of the group became hypoglycaemic, despite the likely depletion of hepatic glycogen stores, and none reached high blood lactate levels. In this study, one controversial factor that was suggested as contributing to the impaired performance during fasting was a fall of branch-chained plasma amino acids, with resulting alterations in brain tryptophan levels and central fatigue.

There has been only limited examination of the impact of fasting upon the various components of cerebral function. However, Johnson and Leck (2010) found that 12 hr of fasting was sufficient to cause a decline in functional reach and the ability to balance on a single limb (whether the eyes were opened or closed). They linked these changes to other typical manifestations of hypoglycaemia (loss of coordination, a staggering gait, fatigue, disorientation, dizziness and vertigo), although they did not make any specific measurements of blood glucose concentrations.

Medium-term fasting (3-10 d)

As in short-term fasting, periods of 3-10 days without food have very different effects in animals and in humans. Loy et al. (1986) found that the swimming time to exhaustion was increased in rats after 3 d of food deprivation. This confirmed earlier observations on rats that had been forced to exercise to exhaustion in activity cages during periods of starvation (Samuels, Gilmore and Reinecke, 1948). Likewise, Young (1959) found that in dogs, the time to exhaustion during treadmill running was increased even after 5 d without food. One factor contributing to the increased performance of the experimental animals during these several studies of sustained fasting was a substantial decrease of body mass.

In contrast to the response seen in animals, fasting humans typically show a reduced absolute maximal oxygen intake or peak aerobic power (measured in $L \cdot min^{-1}$), a shortening of the endurance time during sustained submaximal aerobic effort, and in some instances decreased isokinetic muscle strength. Factors differentiating these responses from those of animals may include a greater loss of motivation in humans, and a lesser compensation of the fluid and

mineral losses associated with a metabolic ketosis (Young, 1959).

Gutiérez et al. (2001) fasted 8 young men for 3 d. This led to a decrease of physical working capacity at a heart rate of 170 bpm (the loss of working capacity was similar whether the subjects rested or exercised during their fast); however, they observed no changes in handgrip force or perception-reaction time. The body mass decreased by 3.5 kg over the 3-d study, but muscle mass decreased by only 0.5%, and body fat by 0.4%, so much of the 3.5 kg loss must have reflected a decrease in the fluid content of the body.

Knapik et al. (1987) studied the effects of a 3.5-d total fast in 8 young men. There were no changes in the endurance of cycle ergometry at a relatively low intensity of effort (45% of maximal oxygen intake). The anaerobic power and isometric strength of the torso and handgrip also remained unchanged, but a small decrement of isokinetic elbow flexion strength was seen at two speeds of rotation (11% at 0.52 $rad \cdot s^{-1}$, and 8.6% at 3.14 $rad \cdot s^{-1}$).

In a study where 12 participants engaged in substantial physical work during 5 d of fasting (walking at 5.6 $km \cdot h^{-1}$, 10% grade for 3 h 15 $min \cdot d^{-1}$), Henschel, Taylor and Keys (1954) noted a 7.7% decrease of absolute maximal aerobic power ($L \cdot min^{-1}$) over 5 d of fasting. Scores for the Harvard fitness index also showed a substantial decrease by Day 4 of the fast, although the relative maximal aerobic power ($mL \cdot kg^{-1} \cdot min^{-1}$) remained unchanged throughout the study. The authors concluded that an acidosis was responsible for much of the deterioration in performance during fasting; however, the 5.5 kg decrease of body mass would seem a more plausible explanation of the unchanged maximal aerobic power.

Lategola (1965) observed a 31.6% decrease of the absolute maximal oxygen intake ($L \cdot min^{-1}$) over a 7-d fast, although this effect was slightly attenuated by the 10th day of fasting. However, as in the study of Henschel, Taylor and Keys (1954), the relative aerobic power ($mL \cdot kg^{-1} \cdot min^{-1}$) and treadmill endurance times showed no significant change during the fast. In the same study, there was a small decrease of handgrip strength, statistically significant at 9.3% by the 9th day of fasting (Consolazio et al., 1967).

As in shorter periods of starvation, effects upon cerebral function have received only limited attention, although Taylor et al. (1945) noted a deterioration of motor speed and coordination in subjects who were required to exercise for 3 h 15 $min \cdot d^{-1}$ during a fast that lasted for 2.5 d.

There are numerous hormonal effects of total fasting that may influence the availability of metabolites, the rate of tissue breakdown and the individual's tolerance of ketosis. In particular, the normal secretory bursts of growth hormone are augmented in both number and amplitude after 2 d of fasting (Hartman et al., 1992). Following a 4-d fast non-obese individuals also show a decrease in secretions of insulin and insulin-like growth factor 1 (Bang et al., 1994), thus encouraging the release of amino acids from muscle for gluconeogenesis (Pozefsky et al., 1976) and decreasing the anabolic response to any sort of muscle training.

Table 2: Main changes of physical performance seen in Minnesota starvation experiment (24% loss of body mass over 24 wk).

- Decline in oxygen cost of walking at 5.6 $km \cdot h^{-1}$, proportional to decrease in body mass.
- Harvard Fitness Test Scores: control 64; 12 wk 33; 24 wk 18
- Duration of run: control 242 sec; 12 wk 106 sec; 24 week 50 sec
- Decrease of heart volume
- 22% decrease of red cell count
- Decrease of maximal oxygen intake and grip strength when 12-16% loss of body mass.

Severe food deprivation

The Minnesota starvation study

Ancel Keys and his colleagues (1950) made a very thorough evaluation of the physiological effects of prolonged semi-starvation (24 wk) in experiments upon a panel of 36 moderately active and healthy conscientious objectors during and immediately following World War II.

The initial body fat stores of their subjects averaged 9.6 kg. At least one member of the group failed to comply with the dietary restrictions that were imposed, and information on this individual was excluded from the final data analyses. During the 24-week trial, the subjects were given a very limited diet of the type that might be anticipated under famine conditions (mainly potatoes, rutabagas, turnips, bread, and macaroni). The total intake of food energy was reduced from a normal figure of 14.5 to 6.5 $MJ \cdot d^{-1}$. In addition to any required laboratory exercise tests, the subjects continued to undertake about 15 h of light housekeeping per week, as their physical condition allowed. Subjective complaints included feelings of hunger, and in a number of individuals there was also an enhanced sensitivity to cold (probably related to a loss of sub-cutaneous fat). Clinical examination showed ankle oedema and an accumulation of fluid in the knee joints, suggestive of a decrease in plasma proteins.

The course of weight loss was carefully documented (Table 2). On average, the men lost 16.8 kg (24% of their body mass) over the 24 wk. Specific gravity measurements suggested that 6.5 kg of the total loss was fat; some of the remainder was likely water, but 9-10 kg, or about a third of the subjects' initial lean tissue mass was also metabolized. Decreases in limb circumference were consistent with such losses. However, the plasma volume showed only a small decrease. There was also a 56% increase in extracellular fluid volume, and since the abdominal circumference showed relatively little change, an intra-abdominal accumulation of ascitic fluid may be presumed, again probably a reflection of low plasma protein concentrations.

The changes of performance developing during the severe restriction of food intake were much as would be predicted from the studies of total fasting (Table 2). As the fast continued, the oxygen cost of sub-maximal treadmill walking at a speed of 5.6 $km \cdot hr^{-1}$ declined in almost direct proportion to the decrease of body mass. One measure of the decline in aerobic performance was the score obtained on a treadmill version of the Harvard Fitness Test. For this test, subjects were required to run at 11.3 $km \cdot hr^{-1}$, 8.6% grade, and an arbitrary score of 50 T/SP was calculated, where T was the duration of the run (in seconds), and SP was the sum of 3 recovery heart rates (counted between 1-1.5, 2-2.5, and 4-4.5 min post-exercise). The tolerated length of run decreased from an initial

242 sec to 106 sec at 12 wk, and 50 sec at 24 wk of food restriction, while the respective Harvard fitness test scores were 64, 33 and 18 (Buskirk, 1957). The maximal aerobic power (measured in $L \cdot min^{-1}$) also showed a substantial deterioration by the time that the body mass had decreased by 12-16% (Taylor et al., 1957). Furthermore, the heart size as seen in PA radiographs decreased over the experiment, and there was also a 22% decrease in the red cell count.

In terms of muscular performance, there were large decrements of handgrip force and a marked deterioration in coordination as fasting continued (Brožek, 1955; Brožek et al., 1946). Scores on a back-lift dynamometer had decreased by 16% at 12 wk and 31% at 24 wk; decreases of handgrip at 12 and 24 wk were 22% and 40%, respectively. Clinical observations also suggested deterioration in the patellar reflex (Keys et al., 1950), and pattern tracing ability was impaired, although tapping speeds showed little change. Finally, as the experiment progressed, there were marked personality changes, particularly an increase of apathy and introversion; such changes could certainly have a negative effect upon scores in tests requiring maximal subject cooperation.

Other studies of severe food deprivation

Further studies of severe food deprivation have been conducted on military volunteers. In 1953, subjects were restricted to a diet of 2.41 $MJ \cdot d^{-1}$ for 12 d, and in 1955 to 4.2 $MJ \cdot d^{-1}$ for 24 d (Taylor et al., 1957). In contrast to the earlier experiments on the conscientious objectors, the observers in the military trial considered that apprehension and loss of motivation contributed to the poor and variable performance of the soldiers during food deprivation, and many of the tests showed no consistent difference between controls and those undergoing semi-starvation.

Data on maximal oxygen intake were obtained on a few subjects. The reductions in maximal oxygen intake in absolute units ($L \cdot min^{-1}$) were for acute starvation (4.5 d) 7.5%, for severe food restriction (2.41 $MJ \cdot d^{-1}$ for 12 d) 4.1%, and for prolonged but more moderate food restriction (4.2 $MJ \cdot d^{-1}$ for 180 d) 37.3% (Buskirk, 1957). However, as in the other studies noted above, the maximal aerobic power per unit of body mass remained unchanged throughout. Some acidosis and dehydration developed with prolonged food restriction, but the heart rate when walking at 5.6 $km \cdot hr^{-1}$ was actually less than in the control period (Buskirk, 1957).

Finally, studies in patients with chronic malnutrition due to gastro-intestinal disorders or deliberate dieting have shown an increased response of the adductor pollicis to electrical stimulation of the ulnar nerve, with a slowing of muscular relaxation, and a decrease of muscular endurance (Lopes et al., 1982).

Inadequate fluid intake

Fluid requirements depend largely upon the individual's rate of sweating, as determined by the volume of activity that is being performed, and environmental conditions of temperature, humidity and wind speed. The impact of a given sweat loss upon athletic performance depends also on the extent to which intramuscular glycogen is depleted; the release of bound water from glycogen can in some circumstances contribute as much as 2% to the overall pool of body fluids (Shephard, 1982).

Table 3: Effects of dehydration on muscular strength and power.

Author	Extent of dehydration (% body mass)	Variable	Reported change	Dehydration Method
Bigard et al. (2001)	2.95%	Knee extension strength and endurance	No change of strength, trend to faster fatigue	Sauna
Bijlani & Sharma (1980)	2-3%	Forearm extension	Reduced endurance at 3%	Heat
Bosco et al. (1968)	2.5%	Maximal isometric strength	Strength/mass ratio decreased, 11% decrease in strength	Water deprivation
Caterisano et al. (1988)	3.0%	Cybex muscular endurance	Decrease in inactive and anaerobically trained, but not in aerobically trained	Sauna
Evetovich et al. (2002)	2.9%	Maximal & sub-maximal isometric, isokinetic biceps strength	No significant change	Water deprivation
Fogelholm et al. (1993)	5-6%	Sprint and 1 min anaerobic test	Unchanged performance	Combination of techniques
Greenleaf et al. (1967)	3.3%	Maximal isometric strength (female)	No significant change elbow flexion or knee extension	Exercise/heat
Greiwe et al. (1998)	3.8%	Isometric strength of knee extensors and elbow flexors	No significant change	Sauna
Gutiérrez et al. (2003)	1.8% (men) 1.4% (women)	Rowing & handgrip strength, squat & counter-movement jumps	No effect in men; decrease of squat jump in women after rehydration	Sauna
Hayes & Morse (2010)	1.0, 2.6%	Isometric, isotonic leg torque, vertical jump	Isometric force reduced at 1%, isokinetic torque reduced at 2.6%, no change of jump	Exercise/heat
Hickner et al. (1991)	4.0%	Isometric arm cranking	4.5% decreased power	Combination of techniques
Hoffman et al. (1995)	1.9%	Squat & counter-movement jumps, 30 sec anaerobic test	No significant changes	Exercise/water deprivation
Houston et al. (1981)	4.5%	Isokinetic knee extension	10.5-11.5% decrease at speeds of 30-300 degrees·s^{-1}	Combination of techniques
Judelson et al. (2008)	5.0%	Squat performance	No change	Heat/water deprivation
Kraemer et al. (2001)	6.0%	Isometric & isokinetic strength	11.4% decrease of handgrip force, decrease of isokinetic force in extension and flexion of knee & elbow at various speeds	Combination of techniques
Montain et al. (1998)	4.2%	Knee extension	Strength unchanged, time to exhaustion -15%	Exercise/heat
Périard et al. (2012)	3.2%	Knee extensors	No change of isometric strength or fatigability	Exercise/heat
Saltin (55)	3.8%	Isometric strength elbow flexors, knee extensors	No significant change	Exercise/heat
Saltin (1964)	3.8%	Isometric strength elbow flexors, knee extensors	No significant change	Sauna
Schoffstall et al. (2001)	1.5%	IRM bench press	5.6% decrease	Sauna
Serfass et al. (1984)	5.0%	Isometric and isokinetic tests on biceps	No change in performance	Combination of techniques
Viitsalo et al. (1987)	3.4%; 3.8%	Vertical jump	Improved performance	Sauna, diuretic
Watson et al. (2005)	2.0%	100, 200, 400 m sprints, vertical jump	No changes	Furosemide-induced
Webster et al. (1990)	4.9%	Peak torque, average work per repetition	Decreased performance in upper limb, no change lower limb	Exercise in sweat suit

The minimum fluid need has been estimated at about 250 mL per kJ of energy expended. Most experimental studies of fluid deprivation have been of relatively short duration; possibly, the body may make some adaptations to longer periods of water deprivation. Empirical data show a consistent deterioration of aerobic performance as fluid is restricted, and muscle force is also reduced with more extensive fluid loss.

Aerobic performance

The endurance of sustained, sub-maximal aerobic effort commonly deteriorates if any type of fluid deprivation or loss causes the body mass to decrease by >2%; both running speeds and times to exhaustion are decreased (Buskirk et al., 1958; Craig and Cummings, 1966; Kozlowski, 1966; Webster et al., 1990; Walsh et al., 1994; Fritzsche et al., 2000; Cheuvront et al., 2003, 2007; Casa et al, 2005).

A 1.6-2.1% diuretic-induced dehydration was sufficient to increase the times for performance of 5,000 and 10,000 m runs by 8 and 6% respectively (Armstrong et al., 1985); an increased cardiovascular strain, an impaired thermoregulation and metabolic disturbances may all have contributed to a decrease of performance over such distances. Nadel and colleagues (1980) noted that with 2.2% diuretic-induced dehydration, the cardiac stroke volume during exhausting exercise was decreased by an average of 17 $mL \cdot beat^{-1}$ (about 15%).

Yoshida et al. (2002) tested 4 differing levels of dehydration, induced by exercise in the heat; in their study, scores on a Harvard fitness test were decreased with 2.5% dehydration.

Caldwell and associates (1984) tested a group of boxers, wrestlers and judoku. After a diuretic-induced 3.1% loss of body mass, they observed a 2.9% decrease of maximal oxygen intake and the work-rate during maximal exercise was also decreased; a weight loss achieved in 24 hr or less by use of a sauna or diuretics was more detrimental to performance than a loss that developed over 48 hr. Webster et al. (1990) also found a 6.7% decrease of peak oxygen intake with a 4.9% weight loss.

Saltin (1964a) found no change of maximal aerobic power when 5% dehydration was induced by a period of sauna exposure, but subjects did show a substantial decrease of endurance during submaximal effort. During submaximal effort, heart rates were also higher with dehydration (Saltin, 1964b).

Two studies found no adverse effects upon physical performance from 3.3% dehydration. Penkman et al. (2008) found no change in the outcome of a 2000 m rowing event, and Greenleaf, Prang and Averkin (1967) observed no change in the scores on a Harvard Fitness test following dehydration.

Muscular performance

There is general agreement that substantial dehydration can have adverse effects upon muscular performance (Fogelholm, 1994; Judelson et al. (2007), although there remains a need to clarify the critical amount of dehydration needed for a significant functional limitation (Table 3). One review concluded that hypohydration could decrease strength by 2%, muscular power by 3%, and high intensity endurance by as much as 10% (Judelson et al., 2007). The consensus of one recent review (Kraft et al., 2012) was that maximal isometric strength and peak

torque were unchanged up to 4% dehydration, but that the torque during slower contractions was impaired with >2% dehydration. However, individual responses differ, depending on the method of dehydration, other possible associated stressors such as prolonged exercise, heat exposure or food restriction, and alleviating factors such as high initial fluid reserves (Table 3).

Individual studies confirm the impression that losses of performance are larger for power and endurance than for isometric strength. Serfass and associates (1984) found no deterioration of handgrip force or ability to make 180 repeated contractions in wrestlers after dehydration equal to 5% of body mass. Judelson et al. (2008) also found no deterioration of back-mounted squat performance, even with 5% dehydration. However, there were substantial hormonal changes, including increased levels of cortisol and noradrenaline, and a reduced testosterone response to exercise.

Evetovich et al. (2002) found no deterioration in maximal or submaximal isometric or isokinetic performance of the biceps with 2.9% dehydration. Even 4.2% dehydration had little effect on knee strength; in contrast, muscular endurance was reduced by 15%, although a part of this loss could reflect a loss of motivation and central drive (Montain et al., 1998).

Webster and co-workers (1990) observed a decrease in upper limb but not lower limb torque with 4.9% dehydration. In one of the few studies of female subjects, Greenleaf and colleagues (1967) tested young women following a 3.2% dehydration induced by a combination of exercise and heat; there were no changes in maximal isometric force of knee extension or elbow flexion, nor were there changes in scores for sit-ups, push-ups, and 100 yard and 400 yard runs.

Furosemide-induced dehydration (a 2.2% decrease of body mass) did not alter the performance of sprinters in 50, 200 and 400 m races, nor did it change their vertical jump heights (Watson et al., 2005), although in all of these and other jumping events a smaller body mass was being propelled after dehydration. Others, also, have found no change of jump height with a rapid 1.8-2% dehydration (Hoffman et al., 1995; Gutiérrez et al., 2003), and jump performance has even been improved with a 3-6% decrease in body mass (Viitasalo et al., 1987).

A few studies have found changes in isometric performance. Thus, Hickner et al. (1991) observed a decreased power output on an isometric arm-cranking test with 4.5% dehydration. Hayes and Morse (2010) produced dehydration by jogging in the heat; no deterioration was seen in vertical jump height or isokinetic leg extension at a rotation speed of 120 degrees·s^{-1} s, but isokinetic extension torque at 30 degrees·s^{-1} was impaired by >2.6% dehydration, and isometric leg extension was impaired with >1% dehydration. Bosco et al., (1968) also saw a decline of maximal isometric strength with dehydration, probably related to electrolytic disturbances within the muscle cytoplasm. Further, the time to muscular exhaustion and the tolerance of repeated muscular efforts was also impaired with >2% dehydration (Kraft et al., 2012).

Anaerobic power and capacity

Some authors have found no effect of fluid deprivation upon anaerobic power and capacity. Others have seen some impact, but nevertheless, the fluid deficit needed to influence anaerobic function

has been larger than that needed for a decrease of aerobic power, probably in the range 3-4% (Kraft et al., 2012).

In one trial, a 2.7% decrease of body mass had no effect on anaerobic power, anaerobic capacity or the fatigue index as measured by the Wingate test (Cheuvront et al., 2006). Other authors found that scores on a 30 s Wingate test of anaerobic power and capacity were unchanged by thermal dehydration of as much as 5% (Jacobs, 1980; Hoffman et al., 1995). However, Webster et al., (1990) noted a 21% decrease of anaerobic power and a 10% decrease of anaerobic capacity when wrestlers reduced their body mass by 5% over 36 hr; in their study, the anaerobic test continued for 40 rather than 30 s. Likewise, Jones et al. (2008) found that 3.1% dehydration reduced the peak anaerobic power by 14.5% in the upper body and 19.2% in the lower body. Yoshida et al. (2002) tested 4 differing levels of dehydration, induced by exercise in the heat with partial fluid deprivation; they concluded that anaerobic power was decreased with 3.9%, but not with 2.5% dehydration. Times for both 5 and 10 m dashes were also increased with 3% dehydration (Magal et al., 2003).

Thermoregulation

Quite small decreases in body fluids have adverse effects upon the body's capacity for thermoregulation (Claremont et al., 1975; Kovacs, 2008), probably because of a 27% reduction in cardiac stroke volume and a 20% reduction in skin blood flow; there was no evidence of any attenuation of sweating when subjects cycled for 2 hr in the heat without fluid replacement (Montain and Coyle, 1992).

Cerebral function

Cognitive performance (particularly motivation to maximal effort) begins to deteriorate with a cumulative fluid loss decreasing body mass by >2% (Murray, 2007). Degoutte et al. (2006) examined the cerebral effects of combined weight loss. Judoku were asked to restrict the intake of food and fluid in order to reduce their body mass by 5% in the week preceding competition. Compared to competitors who maintained their body mass, food intake was reduced by some 4 $MJ \cdot d^{-1}$, and the Profile of Mood States questionnaire showed a loss of vigor, with increases of fatigue and tension and a 5% decrease in hand-grip force. Greater dehydration impairs cerebral blood flow, with impairment of coordination and eventual loss of consciousness (Shephard, 1982).

Other adverse effects

Other adverse effects of exercising while dehydrated include an increase of oxidant stress that could potentially increase muscular soreness, seen with 3% depletion of water (Paik et al., 2009), and a deterioration in some aspects of immune function, particularly a slowing of lymphocyte proliferation rates (Penkman et al., 2008) that could increase susceptibility to infection.

Time needed for rehydration

If a competitor has been "making weight," performance is commonly restored over about 5 hr of rapid rehydration, but there may be a persistent 2% deficit of body mass, and this can lead to a continuing impairment of physical working capacity (Herbert and Ribisl, 1972). A full recovery from dehydration can take as long as 24-36 hr, thus potentially leading to poor

performance on the part of athletes who attempt a rapid rehydration immediately after completing their "weigh-in" for a weight-classified sport.

The issue of Ketosis

The classical view of ketosis

Many nutritional scientists have argued that an adequate metabolism of carbohydrate is important to avoiding ketosis and thus optimizing physical performance. Patients following low carbohydrate diets commonly complain of weakness and early fatigue. The classic studies of Christensen and Hansen (1939) demonstrated that relative to a high carbohydrate diet, a low carbohydrate intake cut the time to exhaustion on a cycle ergometer from 206 to 81 min.

The example of the Inuit

The lifestyle of the traditional Inuit provides an interesting exception to this classical viewpoint, since they have typically engaged in a very high level of daily physical activity (Shephard and Rode, 1996) despite having access to little or no dietary carbohydrate during their hunting expeditions (Phinney, 2004). Possibly, their adaptation to a protein/fat diet has been helped by an increased intake of salt from the drinking of brackish water and the ingestion of caribou blood (which itself has a high salt content)(Phinney, 2004).

Adaptation of Europeans to a ketotic diet

Europeans, also, can develop a tolerance to a fat and protein diet over the course of several weeks. The U.S. Schwatka expedition, organized between 1878 and 1880 CE to seek evidence of the ill-fated Franklin expedition provides one good example of this phenomenon. Lieutenant Frederick Schwatka and a party from the U.S. Army spent nearly a year searching the arctic tundra. They travelled by foot and by dog sled, covering a distance of 4360 km in just over 11 months, and wintering on King William Island. They had no sources of food other than the Inuit diet of fish and game after the first month of their journey. Schwatka commented (Stackpole, 1965): *"When first thrown wholly upon a diet of reindeer meat, it seems inadequate to properly nourish the system, and there is an apparent weakness and inability to perform severe exertive fatiguing journeys. But this soon passes away in the course of two or three weeks."* At the end of the year, Schwatka and an Inuit companion remained in sufficiently good condition to cover the final 105 km of their trek in less than 2 d.

Laboratory trials of a ketotic diet

In 1929, the Schwatka observations were replicated in the laboratory, with a subject eating a diet of meat and fat for an entire year. He remained in apparently good health throughout this period (McLellan and Dubois, 1930; McLellan, Rupp, and Toscani, 1930). A recent and more sophisticated study of a ketogenic diet was conducted in a metabolic ward. The participants received 1.2 g protein per kg of body mass each day, and essentially lived off their reserves of fat for a 6-week period (Phinney, 2004). During repeated treadmill testing, a back-pack was adjusted to maintain a constant total weight of body mass plus pack. No decline of maximal oxygen intake was seen over the 6-week study.

A further 4-week experiment (Phinney et al., 1983a, b) provided well-trained cyclists with less than 20 g of carbohydrate per day. Their carbohydrate oxidation fell by 30%, but the subjects

maintained a normal blood glucose concentration throughout the study. Moreover, they showed no change in either their maximal oxygen intakes or their endurance times when cycling at 62-64% of maximal oxygen intake. Tests in elite gymnasts have also shown no deterioration of performance in any of a series of gymnastic performance tests (hanging straight leg raise, ground push up, parallel bar dips, pull up, squat jump, countermovement jump, 30 sec continuous jumps) after 30 d of consuming a high protein ketogenic diet (Paoli et al., 2012).

Other reports have shown varied effects of a high ketone diet, ranging from enhanced through unchanged to impaired physical performance. Brinkworth et al. (2009) assigned 60 middle-aged obese individuals to a 30% energy deficit, an isoenergetic, or a hyper-energetic diet. The energy deficit diet had no effect on maximal or sub-maximal aerobic activity or muscle strength over 30 d of dieting. Walberg et al. (1988) exposed obese women to an iso-nitrogenous but low calorie and very low carbohydrate diet for 4 wk; study participants showed a decrease in body mass of about 2 $kg \cdot wk^{-1}$, and a final 12% decrease of the absolute maximal aerobic power, measured in $L \cdot min^{-1}$, but no change of relative aerobic power, measured in $mL \cdot kg^{-1} \cdot min^{-1}$. Russell et al. (1983) assessed muscle function by electrical stimulation of the adductor pollicis muscle. After 2 wk of partial and 2 wk of complete fasting, there was an increase of muscle force, a slowing of relaxation, and greater fatigue. White and associates (2007) found that after 2 wk of a low carbohydrate, low calorie, ketogenic diet, the perceived fatigue at any given intensity of exercise was increased, and they argued that this could have a negative impact upon the habitual physical activity of an ordinary individual who was attempting to decrease his or her body mass. Bogardus et al. (1981) examined the effects of a very low carbohydrate diet (<1%) when exercising at 75% of maximal oxygen intake; in this study, there was a 50% decrease of endurance time on a cycle ergometer, but this could reflect either a physiological change such as the reduction in glycogen stores or a decrease in motivation of the subjects when their diet was restricted.

Two important caveats

There seem two important caveats with respect to the apparent tolerance of a prolonged ketotic diet. Firstly, the individual's protein intake must be increased sufficiently (possibly by as much as 30-50 $g \cdot d^{-1}$) to permit adequate gluconeogenesis without a progressive breakdown of muscle protein, and secondly, because intramuscular glycogen reserves are depleted, one must accept that the ability to perform sustained muscular efforts is likely to be compromised.

As the body adapts to a ketotic diet, the ketones may possibly replace some of the glucose that normally provides the main source of metabolites for the brain (Bistrian, 1978). However, if the total intake of protein is deficient, the development of ketosis is likely to increase the rate of protein loss relative to dieting with a non-ketotic diet (Vasquez and Adibi, 1992). If protein intake does not match the demands of augmented gluconeogenesis, there will be a progressive loss of lean tissue, and this will have adverse effects upon both aerobic and muscular activity, as seen in the starvation experiments of Ancel Keys and his colleagues (1950). This point is

well illustrated in a recent experiment; moderately obese women with a daily food intake of 2.1 MJ, a carbohydrate intake <10 $g \cdot d^{-1}$ and a protein intake of 1.5 g per kg of body mass showed no significant decrease of maximal oxygen intake when their body mass had decreased by 17 kg; however, a parallel group who were given what would have been regarded as an adequate protein intake if they were not dieting (1.1 $g \cdot kg^{-1}$) showed a 15% decline of maximal aerobic power (Davis and Phinney, 1990). The group with the lower protein intake also showed a 13% decrease of peak isokinetic force, although there were no inter-group differences in the time to muscular fatigue (Davis and Phinney, 1990).

Practical implications

Although it is difficult to separate reduced motivation from the phyiological effects of fasting and dehydration, if there is a substantial reduction in the intake of food or fluid, there are generally negative implications for athletic performance. Where possible, athletes should thus maintain energy and fluid balance, and correct any deficit as rapidly as is practical. Although the decrease of body mass associated with fasting can increase the tolerance of running to exhaustion in laboratory animals, in humans, even brief periods of total fasting (24-36 hr) generally decrease times to exhaustion and impair sustained muscular contractions, often with a deterioration in motor coordination and balance. Longer periods of fasting (2.5-10 d) reduce the human PWC_{170} and the maximal oxygen intake (as measured in $L \cdot min^{-1}$, but not in $mL \cdot kg^{-1} \cdot min^{-1}$). Some studies also show decreases in isokinetic and isometric strength. Further, the Minnesota starvation experiments demonstrate that over 24 wk of grossly inadequate food intake, maximal oxygen intake, the Harvard Fitness test score, the duration of treadmill running and handgrip force are all reduced, with a decrease in red cell count and radiographic estimates of heart volume. The only positive effect of a limited food intake is a decrease in the oxygen cost of walking, seemingly due to a decrease in body mass.

Athletic performance is also vulnerable to relatively small disturbances of fluid balance. A dehydration of 2% is often sufficient to impair aerobic function, thermoregulation and cerebral function, although larger fluid losses (4-5%) are usually needed to reduce anaerobic power, anaerobic capacity and muscular performance. Athletes seeking to "make weight" following deliberate dehydration may require 24 hr to achieve full rehydration and thus a recovery of performance.

Ketosis is associated with a lack of dietary carbohydrate, but this seems less detrimental to physical performance than was once believed. The traditional Inuit population can sustain high daily energy expenditures very well on a diet that is mainly protein and fat, and Europeans also seem capable of adapting to a very small dietary intake of carbohydrate. As adaptation develops, the brain can metabolize ketotic products thus sparing body glucose reserves.

Although athletic performance is likely to be less than optimal, studies of athletes observing the intermittent fasting required by Ramadan have not to date demonstrated any dangers to health, and changes in physical performance have been small (Shephard, 2012 a, b). Potential issues include a reduced period for sleeping, disturbances of circadian rhythm, a decrease of plasma glucose and

fluid reserves in the afternoon and evening, and a decrease in the levels of plasma amino acids needed for an optimal training response. However, most of these issues can be circumvented within the requirements of Islam, given a very careful regulation of food and fluid intake during the hours of darkness. The one caveat is that most of those studied have been engaged in team sports, and further information is needed on the possible risks of dehydration and glycogen depletion in longer events such as marathon and ultra-marathon runs.

Conclusions

Athletic performance can be threatened by either short periods of total fasting or longer periods of severe dietary restriction. However, the functional losses are less than might be imagined, and (given the smaller effects that are seen in experimental animals) loss of motivation may well account for some of the observed negative effects in tests that require maximal effort from the subject. Two factors contribute to the limited effects of food deprivation: the decrease of body mass reduces the energy cost of performing tasks where the body mass is displaced, and the body has a surprising ability to adapt to the metabolism of stored fat and tissue protein. Fluid stores are smaller in relation to the demands of vigorous exercise, and many aspects of physical performance are impaired with dehydration equivalent to 2-5% of body mass.

Author's Qualifications

The author's qualifications are Roy J. Shephard, C.M., M.B.B.S., M.D.[Lond.], Ph.D., D.P.E.,LL.D.,D.Sc.

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