

# The Physiology of Marathon Running

*Just What Does Running a Marathon Do to Your Body?*

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Running a marathon has been viewed, and still is by many, as too extreme to be healthy. Certainly, the physical stress of running a marathon played some role in not holding a women's Olympic marathon race until 1984. On the flip side, casual runners think that if a pampered celebrity can run a marathon, it can't be all that strenuous. While marathon running is far from damaging, it should be respected for the physiological stress inflicted over its 26.2 miles.

For example, running a five-minute-per-mile marathon requires a 15-fold increase in energy production for over two hours. Even runners who finish in over four hours maintain a 10-fold increase in their metabolism. Such extended energy demands require the cardiorespiratory, endocrine, and neuromuscular systems to operate at an elevated level for an inordinate length of time. It is no wonder then that the story of Pheidippides and his marathon run to Athens easily grew into a tragic tale about how running a marathon killed the first person to do so. Fortunately, scientists have researched the physiological stresses of running a marathon. The findings from such studies can help potential marathon runners better appreciate what they will be up against and remind seasoned marathon runners just how amazing the human body is.

## SUDDEN DEATH

The physiology on marathon running starts with Pheidippides, who reputedly ran from the plains of Marathon to the city of Athens to report the victory of the Athenian army over the Persians. Upon his arrival, Pheidippides exclaimed, "Rejoice, we conquer" and dropped dead—or did he? The accuracy of this account has been questioned by modern scholars (Martin and Gynn 2000); however, the unfortunate outcome of Pheidippides is manifested in a few marathon runners every year. Just how stressful to the human body is running a marathon? This and other questions regarding marathon running were addressed at The Marathon:

Physiological, Medical, Epidemiological, and Psychological Studies conference in 1976. The boldest theory regarding marathon running was made by Dr. Tom Bassler (1977), who suggested that the stress of running a marathon built immunity to the development of fatty deposits within coronary arteries. In other words, running a marathon prevents coronary artery disease (CAD). Bassler compared marathon runners to the heart-disease-free Masai warriors and Tarahumara Indians in that they all maintain active lifestyles, eat healthy diets, and have enlarged and wide-bore coronary arteries.

After reviewing the cause of death in marathon runners from the previous 10 years, Bassler claimed that “there have been no reports of fatal, histologically proven, [CAD] deaths among 42K men.” While he noted that some runners have died while running marathons, he concluded that these deaths were due to other factors such as nonatherosclerotic heart diseases (such as myocarditis or coronary spasms), congenital abnormalities, hyperthermia, or undertraining. To his credit, Bassler also acknowledged that a low-fat diet and abstention from smoking play important roles in developing immunity to heart disease. Bassler concluded that whether running a marathon offered absolute protection from CAD would be proven within the following 10 years.

At the same conference, Bassler’s claim was refuted with four documented cases of marathon runners who had died from CAD (Noakes et al. 1977). Noakes (1987) bolstered his opposition with a follow-up report on a total of 36 documented cases of heart attacks or sudden death in marathon runners prior to 1984. Angiography, autopsy, or electrocardiographic results were available for 27 of the runners, 25 of whom had some degree of CAD. Sudden death occurred in 22 of the 36 runners, with 19 of those deaths occurring during, immediately after, or within 24 hours after running a marathon or a long training run. While this report clearly showed that marathon running alone does not guarantee a life free of CAD, it should be noted that the contributing factors of smoking and diet mentioned by Bassler were not addressed.

The most damning evidence against Bassler’s theory, however, came from one unfortunate case study. Jim Fixx was an overweight, overstressed smoker whose father suffered a heart attack at the age of 35 and died eight years later. Rehabilitation of a tennis injury motivated Fixx to start running to the point that he completed several marathons and wrote the bestseller *The Complete Book of Running*. Because of Fixx’s positive family history for heart disease and his passion for running, he understandably agreed with Bassler’s theory. His faith in Bassler’s theory may be why Fixx ignored chest pains while he ran, hoping they would eventually go away if he kept on running (Plymire 2002). Unfortunately, his passion for running came to an end along a Vermont road in 1984 when, in the middle of a run, Jim Fixx died of a heart attack. An autopsy found a complete blockage in one coronary artery, an 80 percent blockage in another, and signs of previous heart attacks.

The death of Jim Fixx convinced the world not only that running a marathon couldn't prevent CAD but that running could result in sudden death.

While true, the risk for sudden death is greater in marathon runners who, similar to Fixx, have a positive family history, elevated cholesterol, and warning signs such as angina, nausea, and epigastric discomfort (Noakes 1987). This was confirmed by Maron et al. (1996), who quantified the risk of marathon running with data from the 1976 to 1994 Marine Corp marathons and from the 1982 to 1994 Twin Cities marathons. Out of 215,413 runners, there were four deaths, three men and one woman. One of the men experienced chest pains at mile 20 and died 15 minutes after finishing, while the other three runners died on the course. All three men died of heart attacks, while the woman's death was attributed to an abnormal origin of the left main coronary artery on the aorta resulting in inadequate blood supply to the heart. Two of the men had significant blockage (greater than 50 percent) in three arteries, and the other had significant blockage in two arteries.

Roberts and Maron (2005) published an updated report with data through 2004 for the same two marathons. There was one additional death in nearly the same number of finishers as in their first study. Combining the data, there were five deaths and four successful resuscitations that occurred in 8 men and one woman. The updated risk of sudden death improved to 1 in 220,000 finishers. The authors point out that the decreased risk is likely due to the availability of external defibrillators due to three nonfatal heart attacks in their recent study compared to only one in their original study. Another study identified eight cases of sudden death in over 840,000 runners, or a nearly one in 100,000 ratio, during 19 years of the London and New York City marathons (Pedoe 2000).

Determining an exact risk of sudden death from marathon running would require accounting for the degree of preexisting heart disease, the quality of medical treatment at the marathon, and gathering much more data. However, it is noteworthy that these estimated sudden death risk ratios from marathon running are better than the estimated one death in 15,000 for jogging (Thompson et al. 1982) or one death in 18,000 for general exercise (Siscovick et al. 1984).

A few studies have looked for signs of heart damage immediately following and for hours after completing a marathon (Kratz et al. 2002; Siegel et al. 1997; Lucia et al. 1999). The levels of proteins typically used to diagnose cardiac damage were slightly elevated, indicating a mild stress to the heart, but none of the levels approach those seen following a heart attack. However, there are documented cases of individuals who experience cardiac arrest despite no evidence of CAD. Green et al. (1976) found extensive damage to the heart, but no CAD, in a 44-year-old marathon runner who collapsed after 24 miles of a marathon and later died. Ratliff et al. (2002) reported normal coronary arteries in a 22-year-old runner who collapsed from cardiac arrest at the finish line. The runner

survived, but because of acute kidney failure from dehydration and nonsteroidal anti-inflammatory drug (NSAID) use, he developed gangrene in his lower right leg, which had to be amputated.

The ability of the heart to effectively fill and pump blood has been researched in postmarathon runners. For example, Neilan et al. (2006) found mildly impaired heart function that persisted for one month. Therefore, any person considering running a marathon, particularly those over the age of 45, should check with a doctor before starting to train.

## HYPERTHERMIA

Besides supplying oxygen-rich blood to the body, the heart helps control body temperature by pumping warm blood to the skin where body heat is lost through the evaporation of sweat. During a marathon, heat loss and production can increase over 10-fold. High humidity and dehydration can make heat loss more difficult. High humidity levels reduce evaporation, while dehydration impairs the ability to transfer heat from the muscles to the skin. Either situation will increase body temperature and the risk for heat problems. Muscle weakness and disorientation can develop with body temperatures of 105-106 degrees Fahrenheit, and a loss of consciousness can occur with body temperatures near 107 degrees Fahrenheit. Without the ability to lose heat through evaporation, body temperature would rise fast enough to cause heat problems after only 15 to 20 minutes of running. Even with the ability to sweat, it is not uncommon for marathon runners to finish the 26.2 miles with body temperatures of 105 degrees Fahrenheit.

However, heat problems can occur in much milder conditions. For example, in the 2001 Chicago Marathon, a 22-year-old man collapsed within 300 yards of the finish line as he neared the three-hour mark. Despite quick medical attention, he later died with a body temperature of 107 degrees Fahrenheit despite temperatures in the 50s at the time of his collapse (Nevala 2001). According to Chevront and Haymes (2001), an elevated body temperature, or hyperthermia, during marathon running can be due to the climate, dehydration, a relatively high metabolic rate from running a faster-than-usual pace, or a combination of factors. Also, marathon runners may overdress or not remove layers or clothing as the air temperature rises over the course of the marathon. Even though runners can't control the climate, there are other things they can do to prevent hyperthermia.

Since dehydration reduces the amount of blood available for heat removal, one way to prevent hyperthermia would be to drink as much water as is lost through the sweat. The average sweat rate for runners is 1.2 liters per hour. However, most runners either can't tolerate drinking that much or choose not to drink that much liquid. Typically, runners drink as little as 200 milliliters per hour but rarely more than 1 liter per hour. Therefore, it is not uncommon for runners to lose 2

to 10 percent of their body weight through sweating. Studies have shown that dehydration of only 3 percent of body weight can decrease a runner's performance (Cheuvront and Haymes 2001). But dehydration is not the only factor that will increase body temperature.

There is evidence that a high energy production, or metabolic rate, while running may have a stronger influence. Thirty runners had their dehydration level measured and their metabolic rate estimated during the 1987 Cape Peninsula Marathon (Noakes et al. 1991). These values were then compared to their core body temperatures measured within two to five minutes after the marathon. The results showed it was not the degree of dehydration but the metabolic rate during the last four miles of the race that had the strongest correlation to body temperature. Toward the end of a marathon, when the speed and effort of running increase, the body becomes less efficient at using energy, which produces more excess heat, which in turn drives the body temperature even higher. Cheuvront and Haymes (2001) compiled data from 12 studies looking at dehydration in runners. Their results also showed a significant correlation between running speed and core temperature but not between dehydration and core temperature.

## HYPONATREMIA

Could it be that dehydration during marathon running has been overemphasized? Perhaps so, especially with the increase in cases of water intoxication, or hyponatremia, reported in recent marathons. The major cause of hyponatremia, or low blood-sodium levels, is drinking too much water, which dilutes sodium levels in the blood. Low sodium levels cause swelling or edema in the brain, which can be fatal. Davis et al. (2001) found 26 cases of hyponatremia in over 34,000 runners from the 1998 and 1999 San Diego Rock 'n' Roll Marathon. They found that hyponatremia was greater in women, slower runners (those who finish in over four hours), and in people who took over-the-counter nonsteroidal anti-inflammatory drugs (NSAIDs). Similarly, Hew et al. (2003) reported 21 cases of hyponatremia at the 2000 Houston Marathon. There was a similar number of cases in men and women, but hyponatremia was more common in slower runners and those who used NSAIDs.

Women may be at greater risk because less water can dilute sodium levels in smaller bodies; additionally, estrogen can further contribute to brain swelling once it starts. Slower runners are at a greater risk simply because they have more time during a marathon to drink too much water. For example, Hew et al. (2003) found that runners who developed hyponatremia drank about twice that of other runners and were on the course one to two hours longer. In addition, NSAIDs can increase the effect of antidiuretic hormones, which increases water retention. Hyponatremia can develop after completion of a marathon when hormonal

changes cause increases in absorption of water combined with sodium lost in the urine. Hyponatremia is rare, occurring in less than 0.3 percent of marathon runners, but the number of cases increased from 1993 to 2000 (Hew et al. 2003). This increase has been countered by a similar increase in education about the risk of overdrinking from the media and race directors.

On the other hand, drinking water or sports drinks during a marathon should not be avoided. While studies have shown that increases in body temperature are related more to running speed than to dehydration, these studies did not look at the effect of dehydration on actual performance. Even though dehydration may not be related to increases in body temperature, drinking fluids during a marathon maintains adequate blood flow to the muscles to support the high-energy demands of running a marathon. In addition, sports drinks contain electrolytes that help prevent hyponatremia as well as sugars for additional fuel.

The best advice is to drink in moderation before and during a marathon. Common recommendations for marathon runners are to drink 20 ounces of fluid two to three hours before the race and another 8 ounces 30 minutes before. During the marathon, runners should drink 8 to 10 ounces of water or a sports drink every 10 to 20 minutes and afterward drink as much as they comfortably can.

## HYPOTHERMIA

Sometimes hypothermia, rather than hyperthermia, can be the main environmental concern for marathon runners. Between 1982 and 1987, the Glasgow Marathon was run in temperatures ranging from 39.5 to 59.3 degrees Fahrenheit (Ridley et al. 1990). The 1983 Bostonfest Marathon had 11.5 percent of runners request medical treatment, with hypothermia being the most common diagnosis (Jones et al. 1985). Obviously, the risk for hypothermia is greater in cold, windy, or wet weather; however, the American College of Sports Medicine (1996) cites other factors that may reduce body temperature. For example, if the second half of the marathon is run slower than the first half, not enough heat may be generated to maintain body temperature. Also, any sweat that builds up can saturate clothing, which will draw additional heat away from the body. Hypothermia can also occur after the race when heat radiates from a warm body to the cooler air temperature.

The best defense against hypothermia is to dress in layers with an outer layer that protects from wind and water. Layers should be removed as air temperature increases to avoid hyperthermia, and any wet layers should be replaced. While not as common as hyperthermia, the number of hypothermia cases could increase along with the popularity of trail and adventure marathons. Hypothermia can produce more than just cold limbs or noses. Irregular and life-threatening heart rhythms can develop, so it should not be treated any less seriously than hyperthermia.

## GLYCOGEN DEPLETION

Carbohydrate-loading dinners have become a staple of the modern marathon prerace events. And why not? When the facts are linked together, carbohydrate loading makes sense. For instance, carbohydrates provide energy to the muscles faster than fats and are required for optimal aerobic performances. Inside the body, carbohydrates are found as glycogen in the muscles and liver and as glucose in the blood. During a marathon, the muscle gains energy from the glycogen within its cells and from blood glucose. As the amount of glucose in the blood is used up, the liver converts its glycogen into glucose and releases it into the bloodstream to maintain a constant supply of glucose to the muscles. During prolonged exercise, glycogen levels become depleted, leaving the muscles with little of the high-performance fuel and forced to operate on slower-burning fats. This shift in fuel sources does not go unnoticed. Marathon runners describe it as like running into a wall or “bonking.” It is also known that a diet high in carbohydrates can increase the amount of glycogen stored inside the muscles and liver. Therefore, if glycogen depletion leads to a decrease in running speed and a high-carbohydrate diet can increase glycogen stores, then carbohydrate loading should prevent or delay hitting The Wall.

When carbohydrate-loading studies from all endurance sports were reviewed, Hawley et al. (1997) found that carbohydrate loading did improve performance in endurance sports lasting longer than 90 minutes. However, none of the studies used a full marathon as the performance distance. The closest are studies that looked at the effects of carbohydrate loading on 30K race times. Karlsson and Saltin (1971) found that carbohydrate loading improved 30K race times by an average of nearly eight minutes (143.0 to 135.3 minutes). The faster times were the result of not having to slow down rather than being able to run faster. Similarly, six men cut an average of 3.6 minutes (131.0 to 127.4 minutes) off their 30K time after increasing carbohydrate consumption by 200 grams the week before the race (Williams et al. 1992).

Other studies looked at whether carbohydrate loading could prolong the time to exhaustion. Overall, subjects were able to run 10 to 66 percent longer at 70 to 75 percent of maximal effort after having carbohydrate loaded (Brewer et al. 1988; Chryssanthopoulos et al. 2002; Galbo et al. 1967; Lamb et al. 1991).

On the other hand, a study by Sherman et al. (1981) found that eating a diet of 70 percent carbohydrate for three days elevated muscle glycogen levels but failed to improve 20.9K times compared with a diet of 50 percent carbohydrate. Also, in a second part of the study by Lamb et al. (1991), the run time to exhaustion was 16 minutes longer but not statistically better than the group that did not carbohydrate load.

While carbohydrate loading does appear to be effective for most runners, it does have its drawbacks. Consuming too many calories in the name of carbohydrate

loading can add extra body weight, which will increase the energy demands of running a marathon. Also, for every gram of glycogen stored, almost 3 grams of water are stored with it. This can leave a runner with a bloated or heavy feeling. Besides, it may be that hypoglycemia, not muscle glycogen depletion, is a greater concern. If a runner does not consume carbohydrates during a marathon, liver glycogen depletion can occur around two hours, leading to hypoglycemia. Even though muscle glycogen depletion may take longer to develop, hypoglycemia resulting from liver glycogen depletion can reduce running speed from inadequate neural stimulation (Noakes 2003). The problem is that the brain prefers glucose as its fuel, and hypoglycemia impairs brain functions, one of which is stimulation of the muscles. So despite glycogen remaining inside the muscles, hypoglycemia reduces the strength of stimulation from the brain to the muscles, resulting in weaker muscle contraction and slower running speeds (Nybo 2003).

Some experts feel that consuming carbohydrates during a marathon is just as important, if not more so, as carbohydrate loading (Ivy 1999). In fact, many studies have shown that carbohydrate ingestion during exercise helps prevent hypoglycemia and improves performance (Tsintzas and Williams 1998; Jacobs and Sherman 1999). For example, subjects ran two 30K races with either a high-carbohydrate meal before the race and water during the race or a no-carbohydrate solution before the race and a carbohydrate sports drink during the race. Race times were the same regardless of whether carbohydrates were emphasized before or during the race (Chryssanthopoulos et al. 1994). In a study by the same group of researchers, a prerun carbohydrate meal improved run times to exhaustion by about 10 percent, but combining the carbohydrate meal with a carbohydrate sports drink during the run increased performance an additional 10 percent (Chryssanthopoulos et al. 2002). In the marathon study, subjects ran three 42.2K treadmill time trials consuming only water, or a 5.5 percent carbohydrate sports drink, or a 6.9 percent carbohydrate sports drink. Race times were significantly faster (190 minutes) with the 5.5 percent solution compared with either water (193.9 minutes) or the 6.9 percent solution (192.4 minutes) (Tsintzas et al. 1995).

Collectively, these studies show the importance of carbohydrate as a fuel source during prolonged running, but the issue of when and how much carbohydrate should be consumed seems to be an individual matter. Ivy (1999) suggests that both carbohydrate loading before and carbohydrate consumption during an event are needed for those who run between 60 and 70 percent of their  $\dot{V}O_2$ max. But, for faster runners, carbohydrate consumption during exercise is not beneficial because glucose uptake into the muscles cannot occur fast enough to be useful. More research is needed to determine whether these conclusions pertain to marathon runners, but there is little doubt that increasing carbohydrate intake before and/or consuming carbohydrate during a marathon is critical for optimal performance.

The original version of carbohydrate loading starts with three days of a low-carbohydrate diet followed by intense exercise to deplete glycogen stores. This is followed by three days of a high-carbohydrate diet where glycogen levels are supercompensated from a normal value of 100 millimoles per kilogram to 220 millimoles per kilogram. Sherman et al. (1981) found that a gradual increase in dietary carbohydrate along with tapering training resulted in just slightly less glycogen (205 millimoles per kilogram) but with much less stress to the individual. Sherman et al.'s modified version of carbohydrate loading has since been the recommended technique of carbohydrate loading for most endurance athletes.

Consuming carbohydrates during a marathon should be done at a rate of 30 to 60 grams (120 to 180 calories) per hour and 30 to 40 minutes prior to fatigue. The amount and timing are based on the fact that glucose is absorbed into the bloodstream at a rate of 1.0 to 1.2 grams per minute (Ivy 1999). Various types of carbohydrate (glucose, a glucose polymer, or fructose) seem to be equally effective in maintaining blood glucose levels (Noakes 1988). Therefore, it is up to the individual to determine whether sport drinks, energy gels, bananas, flat cola drinks, or some other type of carbohydrate works best.

## INJURY

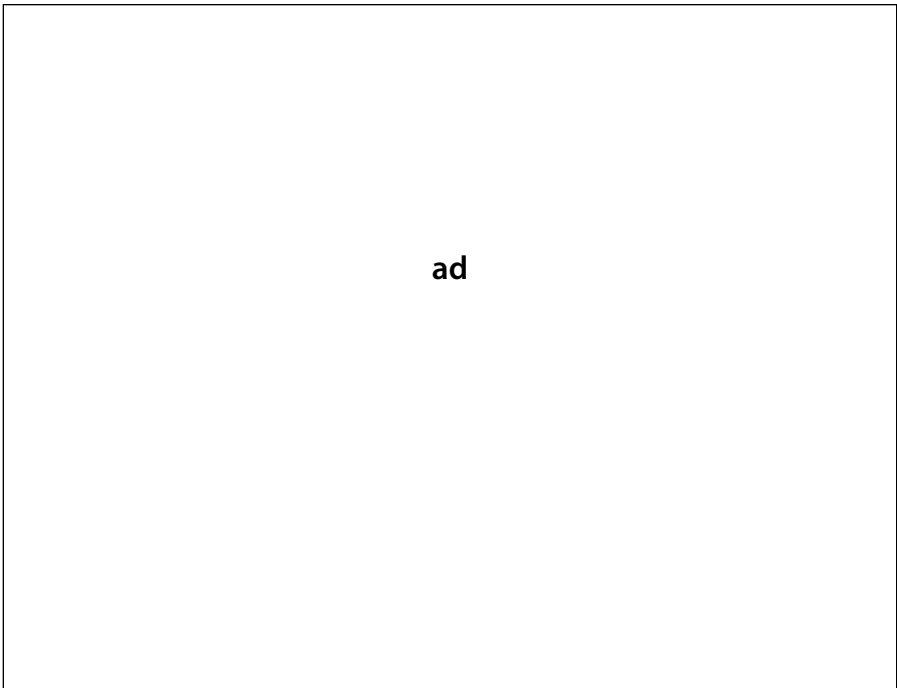
As if hitting The Wall wasn't worry enough, running a marathon can be a musculoskeletal nightmare as well. It takes between 30,000 and 50,000 steps to run a marathon. Every time the foot hits the ground, a stress three to four times body weight is absorbed by the ankles, knees, hips, and lower back. Also, with each stride, some muscles contract to propel the body forward while others control the degree of movement by being lengthened. The lengthening or eccentric contractions are notorious for damaging the muscle's infrastructure. As a result, muscle damage and inflammation can remain for seven days after having run a marathon (Hikida et al. 1983), while repair of muscle fibers can take three to 12 weeks (Warhol et al. 1985). It's not surprising then that postmarathon data have found "stiffness or pain" in 65 to 92 percent of marathon runners (Satterthwaite et al. 1996; Kretsch et al. 1984; Nicholl and Williams 1982).

Fortunately, only a relatively few marathon runners have experienced injuries while running a marathon that caused them to seek medical attention. A survey of runners who completed the 1980 Melbourne Marathon found that 3 percent of runners reported serious injuries, with the most common being knee pain, hamstring problems, dehydration, blisters, and quadriceps pain or cramps (Kretsch et al. 1984). Similarly, the injury rate from 12 years of the Twin Cities Marathon was 2.1 percent of all runners (21.15 per 1,000 entrants), with the top five injuries being exercise-associated collapse (59.4 percent), blisters (19.9 percent), muscle strain (14.3 percent), muscle cramps (6.1 percent), and skin abrasions (1.9 percent) (Roberts 2000). The 1993 Auckland Marathon medical

staff reported a nearly three times higher injury rate of 6.2 percent, with cramps, exhaustion, hematomas, blisters, and lightheadedness being the most common problem (Satterthwaite et al. 1996.) One study that looked at muscle cramping in marathon runners determined that dehydration and electrolyte imbalances may not be responsible (Maughan 1986). It could be that fatigue from running farther or faster than accustomed and irregular stretching may play a stronger role in producing muscle cramps during a marathon (Schwellnus et al. 1997), while other experts feel dehydration and electrolyte imbalances may play a role, especially in hot conditions (Eichner 1998).

While muscle soreness is the major health issue for the average marathon runner, elite runners have additional concerns. Data from the 1986 Wonderful Copenhagen Marathon found the most common problem in elite runners was gastrointestinal (GI) distress (26 percent) followed by back or joint pain (20 percent), muscle cramps (16 percent), and blisters and other skin lesions (16 percent each) (Holmich et al. 1988). It has been speculated that elite runners who suffer from GI distress secrete higher levels of GI hormones (O’Conner et al. 1995) or consume higher amounts of NSAIDs (Smetanka et al. 1999).

Some of the factors that increase the risk for injury while running a marathon are running a first marathon, participation in other sports, illness during the two weeks prior, current use of medication, and training mileage (Kretsch et al. 1984;



Satterthwaite et al. 1999). Runners who train less than 60 kilometers per week were more likely to become injured while running a marathon (Kretsch et al. 1984). Higher levels of training have been shown to decrease the risk for knee injuries but increase the risk of injury to the quadriceps and hamstrings during a marathon (Satterthwaite et al. 1999). With the large number of training miles required to prepare for running a marathon, it is not surprising that 29 to 43 percent of runners develop injuries during training. In fact, the number of injuries from running a marathon is five to 10 times less than while training for a marathon (Chorly et al. 2002; Holmich et al. 1988; Holmich et al. 1989; Kretsch et al. 1984). The premarathon injury rate increases with the number of training miles run per week (Holmich et al. 1989), with most injuries occurring to the feet and knees, followed by the shins and hips (Chorly et al. 2002).

## IMMUNE SYSTEM

Microscopic damage to the muscles from running a marathon can cause more than soreness. As part of the repair process, cytokines are released from the injured area to promote the influx of white blood cells from the immune system. In particular, neutrophils, monocytes, and lymphocytes are elevated after prolonged endurance events such as a marathon (Nieman 2000; Pedersen and Hoffman-Goetz 2000). However, other markers of immune function are lower after running a marathon. For example, nasal and salivary immunoglobulin A (IgA) (Nieman et al. 2002a) is reduced for several hours after a marathon, while postmarathon levels of natural killer cells can be suppressed for at least one week (Berk et al. 1990). There is strong evidence that cortisol, a stress hormone typically released during prolonged exercise, is at least partially responsible for the decrease in natural killer cells. The muscle damage incurred from running a marathon can divert some immune cells for muscle repair and weaken others, leaving the immune system less able to protect against upper respiratory tract infections (Nehlsen-Cannarella et al. 1997; Nieman 1997).

While there is no direct evidence that those runners with the most weakened immune system are those who develop upper respiratory tract infections (URTI), there is evidence of a higher rate of URTI in marathon runners compared with nonrunners. Peters and Bateman (1983) found that 33 percent of the runners who completed the 56-kilometer Two Oceans Marathon developed URTI compared with 15.3 percent in people who did not participate. Also, there was a higher rate (47 percent) in runners who finished under four hours compared with 19 percent in runners finishing over 5.5 hours. Similarly, Nieman et al. (1990) found 12.9 percent of the finishers of the Los Angeles Marathon developed URTI in the following week compared with 2.2 percent in a similar group of nonparticipants. However, Ekblom et al. (2006) found runners reported a similar number of infectious episodes in the three weeks before the 2000 Stockholm Marathon as they did in the three weeks afterward.

The increased number of postmarathon URTI led to the development of the “open window” hypothesis, which says that running a marathon depresses the immune system for three to 72 hours and thus increases the susceptibility to URTI (Pedersen and Toft 2000). Just the possibility of such a relationship has led researchers to investigate whether nutritional supplementation can attenuate the negative effects of marathon running on the immune system. For example, consuming a 6 percent carbohydrate solution during actual and simulated marathons decreased the inflammatory response measured following the runs (Nehlsen-Cannarella et al. 1997; Nieman et al. 2001; Nieman et al. 2003). The glucose solutions helped to maintain blood glucose levels, reducing the release of cortisol, which is thought to weaken the immune system. Glucose consumption during a marathon did not, however, prevent a decrease in salivary immunoglobulin A (IgA), which is one of the first lines of defense against URTI-causing microorganisms (Nieman et al. 2002a).

Free radicals, byproducts of aerobic metabolism, also appear to play a role in promoting the muscle-damage-induced inflammatory response. There is evidence that antioxidants like vitamin C combat free radicals and may help prevent a postmarathon weakening of the immune system. One study found fewer posttrace URTI in ultramarathon runners who consumed 600 milligrams per day of vitamin C for three weeks prior to the ultramarathon (Peters et al. 1993). However, glucose

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intake was not controlled for, and as noted previously, consuming glucose can reduce the amount of stress placed on the immune system following a marathon. In studies where glucose was controlled for, consuming high doses of vitamin C (500 to 1,500 milligrams per day) for seven to 14 days before a marathon or ultramarathon provided no additional benefit to the immune system or in preventing URTI (Nieman et al. 2002b).

Glutamine is an amino acid that provides energy to the cells of the immune system. During a marathon, glutamine levels drop, which could contribute to a weakening of the immune system. One study found that supplementation with glutamine after a marathon resulted in fewer postrace URTI (Castell and News-holme 1997). However, more research is needed to confirm whether glutamine supplementation has a direct effect on strengthening the immune system (Pedersen and Hoffman-Goetz 2000).

Running a marathon temporarily suppresses the immune system, but is the suppression great enough to increase the risk for developing URTI? While there are findings that suggest such a connection, it has yet to be proven that other factors are not responsible. For example, it is possible that high and turbulent airflow rates, cooling and drying of airways, exposure to unfamiliar microorganisms at unique marathon locations, changes in nutrition, muscle microtrauma, travel influences such as sleep deprivation and time-zone shifts, and psychological stress could also lead to an increase in postmarathon URTI (Shephard and Shek 1999). Regardless, it is not unreasonable for marathon runners to follow the guidelines presented by Nieman (2000):

- Keep other life stresses to a minimum.
- Eat a well-balanced diet.
- Obtain adequate sleep.
- Avoid putting hands to eyes and nose.
- Avoid sick people and large crowds.
- Avoid overtraining and rapid weight loss.
- Use carbohydrate beverages before, during, and after marathon races and long training runs.

## OTHER PHYSIOLOGICAL BENEFITS

It could very well be that no other sport is so popular yet as potentially harmful as marathon running. Studies on marathon runners indicate that the physiological stresses of running a marathon far outweigh the physiological benefits. At best, a successful marathon runner will have a few thousand fewer calories to carry around and, once the recovery process is complete, stronger bones, heart, and

muscles. The other benefits either come from the miles of premarathon training or are more psychological or emotional in nature. Despite the fact that running a marathon is hard on the body, even deadly, from an exercise physiologist's standpoint, every runner who crosses the finish has personally validated the miracle that is the human body.

## REFERENCES

- American College of Sports Medicine. 1996. Heat and cold illnesses during distance running. *Medicine and Science in Sports and Exercise* 28(12):i-x.
- Bassler, T.J. 1977. Marathon running and immunity to atherosclerosis. *Annals of the New York Academy of Sciences* 301:579-592.
- Berk, L.S., D.C. Nieman, W.Y. Youngberg, K. Arabatzis, J. Simpson-Westerberg, J.W. Lee, S.A. Tan, and W.C. Eby. 1990. The effect of long endurance running on natural killer cells in marathoners. *Medicine and Science in Sports and Exercise* 22(2):207-212.
- Brewer, J., C. Williams, and A. Patton. 1988. The influence of high carbohydrate diets on endurance running performance. *European Journal of Applied Physiology and Occupational Physiology* 57:698-706.
- Castell, L.M., and E. Newsholme. 1997. The effect of oral glutamine supplementation on athletes after prolonged exhaustive exercise. *Nutrition* 13:738-742.
- Cheuvront, S.N., and E.M. Haymes. 2001. Thermoregulation and marathon running: Biological and environmental influences. *Sports Medicine* 31(10):743-762.
- Chorley, J.N., J.C. Cianca, J.G. Divine, and T.D. Hew. 2002. Baseline injury risk factors for runners starting a marathon training program. *Clinical Journal of Sports Medicine* 12:18-23.
- Chryssanthopoulos, C., C. Williams, W. Wilson, L. Asher, and L. Hearne. 1994. Comparison between carbohydrate feedings before and during exercise on running performance during a 30-km treadmill time trial. *International Journal of Sport Nutrition* 4:374-386.
- Chryssanthopoulos, C., C. Williams, A. Nowitz, C. Kotsiopolou, and V. Vleck. 2002. The effect of a high carbohydrate meal on endurance running capacity. *International Journal of Sport Nutrition and Exercise Metabolism* 12:157-171.
- Davis, D.P., J.S. Videen, A. Mario, G.M. Vilke, J.V. Dunford, S.P. Van Camp, and L.G. Mararam. 2001. Exercise-associated hyponatremia in marathon runners: A two-year experience. *The Journal of Emergency Medicine* 21(1):47-57.
- Eichner, R. 1998. Treatment of suspected heat illness. *International Journal of Sports Medicine* 19(2):S150-S153.
- Eklblom, B., O. Eklblom, and C. Malm. 2006. Infectious episodes before and after a marathon race. *Scandinavian Journal of Medicine and Science in Sports* 16(4): 287-293.
- Galbo, H., J. Holst, and N.J. Christensen. 1967. The effect of different diets and of insulin on the hormonal response to prolonged exercise. *Acta Physiologica Scandinavica* 107:19-32.
- Green, L.H., S.I. Cohen, and G. Kurland. 1976. Fatal myocardial infarction in marathon racing. *Annals of Internal Medicine* 84(6):704-706.
- Hawley, J.A., E.J. Schabort, T.D. Noakes, and S.C. Dennis. 1997. Carbohydrate-loading and exercise performance: An update. *Sports Medicine* 24(2):73-81.
- Hew, T.D., J.N. Chorley, J.C. Cianca, and J.G. Divine. 2003. The incidence, risk factors, and clinical manifestations of hyponatremia in marathon runners. *Clinical Journal of Sport Medicine* 13:41-47.

- Hikida, R.S., R.S. Staron, F.C. Hagerman, W.M. Sherman, and D.M. Costill. 1983. Muscle fiber necrosis associated with human marathon runners. *Journal of the Neurological Sciences* 59:185-203.
- Holmich, P., S.W. Christensen, E. Darre, F. Jahnsen, and T. Hartvig. 1989. Non-elite marathon runners: Health, training and injuries. *British Journal of Sports Medicine* 23:177-178.
- Holmich, P., E. Darre, F. Jahnsen, and T. Hartvig-Jensen. 1988. The elite marathon runner: Problems during and after competition. *British Journal of Sports Medicine* 22(1):19-21.
- Ivy, J.L. 1999. Role of carbohydrate in physical activity. *Clinics in Sports Medicine* 18(3):469-484.
- Jacobs, K.A., and W.M. Sherman. 1999. The efficacy of carbohydrate supplementation and chronic high-carbohydrate diets for improving endurance performance. *International Journal of Sport Nutrition and Exercise Metabolism* 9:92-115.
- Jones, B.H., P.B. Rock, L.S. Smith, M.A. Teves, J.K. Casey, K. Eddings, L.H. Malkin, and W.T. Matthews. 1985. Medical complaints after a marathon run in cool weather. *The Physician and Sportsmedicine* 13(10):103-110.
- Karlsson, J., and B. Saltin. 1971. Diet, muscle glycogen, and endurance performance. *Journal of Applied Physiology* 31(2):203-206.
- Kratz, A., K.B. Lewandrowski, A.J. Siegel, K.Y. Chun, J.G. Flood, E.M. Van Cott, and E. Lee-Lewandrowski. 2002. Effect of marathon running on hematologic and biochemical laboratory parameters, including cardiac markers. *American Journal of Clinical Pathology* 118:856-863.
- Kretsch, A., R. Grogan, P. Duras, F. Allen, J. Sumner, and I. Gillam. 1984. 1980 Melbourne Marathon study. *The Medical Journal of Australia* 22:809-814.
- Lamb, D.R., A.C. Snyder, and T.S. Baur. 1991. Muscle glycogen loading with a liquid carbohydrate supplement. *International Journal of Sport Nutrition and Exercise Metabolism* 1:52-60.
- Lucia, A., L. Serratos, A. Saborido, J. Pardo, A. Boraita, M. Moran, F. Banders, A. Megias, and J.L. Chicharro. 1999. Short-term effects of marathon running: No evidence of cardiac dysfunction. *Medicine and Science in Sports and Exercise* 31(10):1414-1421.
- Maron, B.J., L.C. Poliac, and W.O. Roberts. 1996. Risk for sudden death associated with marathon running. *Journal of the American College of Cardiology* 28(2): 428-431.
- Martin, D.E., and R.W.H. Gynn. 2000. *The Olympic Marathon: The History and Drama of Sport's Most Challenging Event*. Champaign, Illinois: Human Kinetics.
- Maughan, R.J. 1986. Exercise-induced muscle cramp: A prospective biochemical study in marathon runners. *Journal of Sports Sciences* 4(1):31-34.
- Nehlsen-Cannarella, S.L., O.R. Fagoaga, D.C. Nieman, D.A. Henson, D.E. Butterworth, R.L. Schmitt, E.M. Bailey, B.J. Warren, A. Utter, and J.M. Davis. 1997. Carbohydrate and the cytokine response to 2.5 h of running. *Journal of Applied Physiology* 82(5):1662-1667.
- Neilan, T., D. Yoerger, P. Douglas, J. Marshall, E. Halpern, D. Lawlor, M. Picard, and M. Wood. 2006. Persistent and reversible cardiac dysfunction among amateur marathon runners. *European Heart Journal* 27(9):1079-1084.
- Nevala, A.E. 2001. Runner who died had been very fit. *Chicago Tribune*. October 9.
- Nicholl, J.P., and B.T. Williams. 1982. Medical problems before and after a popular marathon. *British Medical Journal* 285:1465-1466.
- Nieman, D.C. 1997. Immune response to heavy exertion. *Journal of Applied Physiology* 82(5):1385-1394.

- Nieman, D.C. 2000. Exercise effects on systemic immunity. *Immunology and Cell Biology* 78:496-501.
- Nieman, D.C., J.M. Davis, D.A. Henson, J. Walberg-Ranking, M. Shute, C.L. Dumke, A.C. Utter, D.M. Vinci, J.A. Carson, A. Brown, W.J. Lee, S.R. McAnulty, and L.S. McAnulty. 2003. Carbohydrate ingestion influences skeletal muscle cytokine mRNA and plasma cytokine levels after a 3-h run. *Journal of Applied Physiology* 94:1917-1925.
- Nieman, D.C., D.A. Henson, O.R. Fagoaga, A.C. Utter, D.M. Vinci, J.M. Davis, and S.L. Nehlsen-Cannarella. 2002a. Change in salivary IgA following a competitive marathon race. *International Journal of Sports Medicine* 23:69-75.
- Nieman, D.C., D.A. Henson, S.R. McAnulty, L. McAnulty, N.S. Swick, A.C. Utter, D.M. Vinci, S.J. Opiela, and J.S. Morrow. 2002b. Influence of vitamin C supplementation on oxidative and immune changes after an ultramarathon. *Journal of Applied Physiology* 92:1970-1977.
- Nieman, D.C., D.A. Henson, L.L. Smith, A.C. Utter, D.M. Vinci, J.M. Davis, D.E. Kamin-sky, and M. Shute. 2001. Cytokine changes after a marathon race. *Journal of Applied Physiology* 91:109-114.
- Nieman, D.C., L.M. Johanssen, and J.W. Lee. 1990. Infectious episodes in runners before and after a road race. *The Journal of Sports Medicine and Physical Fitness* 29(3):289-296.
- Noakes, T., B. Adams, K. Myburgh, C. Greeff, T. Lotz, and M. Nathan. 1988. The danger of an inadequate water intake during prolonged exercise. *European Journal of Applied Physiology* 52:210-219.
- Noakes, T.D. 2003. *The Lore of Running*. Champaign, Illinois: Human Kinetics.
- Noakes, T.D. 1987. Heart disease in marathon runners: A review. *Medicine and Science in Sports and Exercise* 19(3):187-194.
- Noakes, T.D., K.H. Myburgh, J. Du Plessis, L. Lang, M. Lambert, C. Van Der Riet, and R. Schall. 1991. Metabolic rate, not percent dehydration, predicts rectal temperature in marathon runners. *Medicine and Science in Sports and Exercise* 23(4):443-449.
- Noakes, T., L. Opie, W. Beck, J. McKechnie, A. Benchimol, and K. Desser. 1977. Coronary heart disease in marathon runners. *Annals of the New York Academy of Sciences* 301:593-619.
- Nybo, L. 2003. CNS fatigue and prolonged exercise: Effect of glucose supplementation. *Medicine and Science in Sports and Exercise* 35(4):589-594.
- O'Conner, A.M., D.F. Johnston, K.D. Buchanan, C. Boreham, C. Trinick, and C.J. Ridoch. 1995. Circulating gastrointestinal hormone changes in marathon running. *International Journal of Sports Medicine* 16(5):283-287.
- Pedersen, B.K., and L. Hoffman-Goetz. 2000. Exercise and the immune system: Regulation, integration, and adaptation. *Physiological Reviews* 80(3):1055-1081.
- Pedersen, B.K., and Toft, A.D. 2000. Effects of exercise on lymphocytes and cytokines. *British Journal of Sports Medicine* 34:246-251.
- Pedoe, D.T. 2000. Sudden cardiac death in sport—Spectre or preventable risk? *British Journal of Sports Medicine* 34:137-140.
- Peters, E.M., and E.D. Bateman. 1983. Ultramarathon running and upper respiratory tract infections: An epidemiological survey. *South African Medical Journal* 64(15):582-584.
- Peters, E.M., J.M. Goetzche, B. Grobbelaar, and T.D. Noakes. 1993. Vitamin C supplementation reduces the incidence of postrace symptoms of upper-respiratory-tract infection in ultramarathoners. *American Journal of Clinical Nutrition* 57(2):170-174.

- Plymire, D.C. 2002. Running, heart disease, and the ironic death of Jim Fixx. *Research Quarterly for Exercise and Sport* 73(1):38-46.
- Ratliff, N.B., K.M. Harris, S.A. Smith, M. Tankh-Johnson, C.C. Gornick, and B.J. Maron. 2002. Cardiac arrest in a young marathon runner. *Lancet* 360(9332):542.
- Ridley, S.A., P.N. Rogers, and I.H. Wright. 1990. Glasgow marathons 1982-1987: A review of medical problems. *The Scottish Medical Journal* 35:11-14.
- Roberts, W., and B. Maron. 2005. Evidence for decreasing occurrence of sudden cardiac death associated with the marathon. *Journal of the American College of Cardiology*. 46(7):1373-1374.
- Roberts, W. O. 2000. A 12-yr profile of medical injury and illness for the Twin Cities Marathon. *Medicine and Science in Sports and Exercise* 32(9):1549-1555.
- Satterthwaite, P., P. Larmer, J. Gardiner, and R. Norton. 1996. Incidence of injuries and other health problems in the Auckland Citibank marathon, 1993. *British Journal of Sports Medicine* 30:324-326.
- Satterthwaite, P., R. Norton, P. Larmer, and E. Robinson. 1999. Risk factors for injuries and other health problems sustained in a marathon. *British Journal of Sports Medicine* 33:22-26.
- Schwellnus, M., E. Derman, and T. Noakes. 1997. Aetiology and skeletal muscle "cramps" during exercise: A novel hypothesis. *Journal of Sports Sciences* 15(3): 277-285.
- Shephard, R.J., and P.N. Shek. 1999. Exercise, immunity and susceptibility to infection. *The Physician and Sportsmedicine* 27(6):47-71.
- Sherman, W.M., D.L. Costill, and J.M. Miller. 1981. Effect of exercise-diet manipulation on muscle glycogen and its subsequent utilization during performance. *International Journal of Sports Medicine* 2:114-118.
- Siegel, A.J., M. Sholar, J. Yang, E. Dhanak, and K.B. Lewandrowski. 1997. Elevated serum cardiac markers in asymptomatic marathon runners after competition: Is the myocardium stunned? *Cardiology* 88:487-491.
- Siscovick, D., N. Weiss, R. Fletcher, and T. Lasky. 1984. The incidence of primary cardiac arrest during vigorous exercise. *The New England Journal of Medicine* 311(14):874-877.
- Smetanka, R.D., G.P. Lambert, R. Murray, D. Eddy, M. Horn, and C.V. Gisolfi. 1999. Intestinal permeability in runners in the 1996 Chicago Marathon. *International Journal of Sports Medicine* 9(4):426-433.
- Thompson, P.D., E.J. Funk, R.A. Carleton, and W.Q. Sturner. 1982. Incidence of death during jogging in Rhode Island from 1975 through 1980. *Journal of the American Medical Association* 247(18):2535-2538.
- Tsintzas, K., and C. Williams. 1998. Human glycogen metabolism during exercise: Effect of carbohydrate supplementation. *Sports Medicine* 25(1):7-23.
- Tsintzas, K., C. Williams, R. Singh, W. Wilson, and J. Burrin. 1995. Influence of carbohydrate-electrolyte drinks on marathon running performance. *European Journal of Applied Physiology and Occupational Physiology* 70:154-160.
- Warhol, M.J., A.J. Siegal, W.J. Evans, and L.M. Silverman. 1985. Skeletal muscle injury and repair in marathon runners after competition. *American Journal of Pathology* 118(2):331-339.
- Williams, D., J. Brewer, and M. Walker. 1992. The effect of a high carbohydrate diet on running performance during a 30-km treadmill time trial. *European Journal of Applied Physiology and Occupational Physiology* 65:18-24.

