

# Is Potassium Needed in Sports Drinks for Fluid Replacement During Exercise?

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In a recently published Position Stand on Exercise and Fluid Replacement (1), the American College of Sports Medicine largely achieved its stated aim "to provide appropriate guidelines for fluid replacement that will help avoid or minimize the debilitating effects of water and electrolyte deficits on physiological function and exercise performance." However, with respect to potassium (K), a more explicit consideration of its importance as an electrolyte contained in the fluids that are ingested during and immediately following exercise may be warranted. The thesis explored here is that the repletion of body water losses with K-free fluids, whether or not they contain sodium or carbohydrate or both, is both suboptimal and counterintuitive during those competitive events for which "sports drinks" have been developed. The absence of comment regarding K in the ACSM position stand (1) implies the opposite.

Most commercially available sports drinks do contain K, usually at 2.5-5 mEq/L (12). A specific advantage of including K in fluid replacements is not generally recognized, and the results of a few studies with K-free fluids could suggest that no advantage exists (14, 15). A case for a potentially significant benefit can be made from a synthesis of dispersed evidence. Indeed, if there exists neither a physiological basis for nor an identifiable advantage to ingesting K during exercise, the ACSM position stand (1) might better have called for the removal of K-containing salts from sports drink formulations as unnecessary additives.

The position stand noted that K is lost in sweat and that its replenishment from normal diets is to be expected in the postexercise period. Research supports these assertions, for example, as shown by the work of Costill et al. (5), who studied daily K balances in conjunction with exercise at 50% of maximal aerobic capacity in the heat for 2 hr on consecutive days. The position stand also states that potassium is lost "to a lesser extent" than is sodium (Na) in sweat. That is somewhat misleading. It is true that K is lost in a *lesser amount* than is Na in sweat. But the concentration of Na in sweat is clearly much lower than that in plasma, so that sodium is conserved in relation to water loss. In contrast, the K concentration of sweat is at least equal to and often exceeds the plasma K concentration (5). Given that the plasma K concentration is itself elevated above baseline during exercise (2, 6, 14, 15), it is clear that K is lost from the extracellular fluid to a *greater extent* than is Na.

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## Potassium Metabolism During Exercise

Setting aside the extreme variants, a "voluntary dehydration" in the range of 2–4% of body weight (1–3 L) is a typical scenario in many competitive events (5, 9). The potential costs of this degree of dehydration are noted in the ACSM position stand (1) to include heat retention and diminished performance. Both field and animal evidence suggests that hypokalemia may be an important event in the pathophysiology of heatstroke (8). However, most individuals are expected to maintain adequate K status prior to and during an exercise bout. The potential linkage between performance and fluctuations in plasma K is the focus of the present discussion.

The net K deficit from an exercise bout is the sum of the K losses (sweat, gastrointestinal secretions, and urine) corrected for any K that is absorbed during the exercise. This is a small fraction of the total body K content, because the intracellular K concentration (150 mEq/L) far exceeds the extracellular K (4–5 mEq/L). There is no "storage pool" of potassium from which to offset extracellular losses, other than some hepatic K release coincident with glycogenolysis (4). Losses of K in sweat alone would likely range from 5 to 18 mEq at competitive activity levels. For example, losses of K through sweat during 2 hr of exercise in the heat are reported to be approximately 12 mEq at 50% maximal exertion (5) or 17.5 mEq at 65% maximal exertion (my calculations from the data in Reference 14). Cool temperature trials in the latter study produced sweat K losses approximating 11.5 mEq. There is consensus agreement that the total K deficit from competitive exercise events could be readily replaced during recovery or between sequential events from various K sources with little fear of pathology. Intake of a sports drink could offset sweat K losses in this range if the drink contained 5–10 mEq/L potassium and rehydration was 75–100% complete. Additional losses of K in the urine would add somewhat to the total K deficit, but these incremental losses are more variable, depend upon renal adjustments for Na homeostasis, and are rarely measured.

The focus for assessing the value of potassium as a component of a sports drink then shifts to the relationship between altered body K status and exercise performance. There is a substantial compartmental shift consisting of a rise in plasma K (2, 6, 14, 15, 17) that presumably results from excitation effluxes of K that exceed the Na–K pump capacity to restore ion gradients (3). An intracellular K depletion results. The magnitude of this hyperkalemic shift could impact performance during exercise at the levels targeted in the position stand as detailed below.

### **Hyperkalemia**

As noted, the literature consistently documents a rise in plasma K that occurs during exertion at levels comparable to competitive events. The most straightforward studies (14, 15) seem to argue against K as a necessary electrolyte in fluid replacement. A significant hyperkalemia (approximating a 15% rise) persisted in trained subjects throughout cycle ergometry to exhaustion at approximately 30–40 min at heavy intensity (85%  $\dot{V}O_{2max}$ , Reference 15) or during 30–90 min of treadmill running at a more moderate intensity (65%  $\dot{V}O_{2max}$ , Reference 14). In the

first report, no untoward effects of K-free fluid ingestion were noted. The second study (14) included trials at both 35 °C and 25 °C and featured various K-free fluid replacements, all of which were accompanied by gastric secretions of K that contributed to nonsweat K loss. The ability to sustain hyperkalemia during exercise beyond 90 min did differ significantly among the trials; however, the interpretation of this finding is unclear.

In contrast, data from two other studies (2, 6) suggest that added K may be important during fluid replacement. In one study (2), plasma K rose 10% during the first hour of cycling at 60% of maximal  $\dot{V}O_2$  with ad libitum water ingestion. Thereafter, for the remaining 2 hr of cycling plasma K declined toward baseline, and during recovery it further decreased to a low but normokalemic level. Subjects who were given ad libitum access to a sports drink containing K at 5 mM had a greater rise in K (15%) during the first hour, and they maintained normokalemia better during recovery. It was proposed that a significant improvement in perceived exertion reported by those given the sports drink was due to carbohydrate inclusion; however, it could just as readily reflect a benefit from K replacement.

In another study (6), significant hyperkalemia was reported during a 70% maximal exercise, as expected. During a second exercise bout that followed a 4-hr recovery period, hyperkalemia remained significant for subjects who were given free access to a sports drink containing K (14 mg per 100 ml). However, the rise in plasma K was not significant for subjects given a K-free fluid replacement ad libitum. These data suggest that the failure to respond with hyperkalemia was an adjustment for a K deficit during the second bout, whereas those subjects ingesting the sports drink had re-equilibrated their K and could respond normally to the exercise. The authors attributed an improved endurance capacity in the latter group to the presence of carbohydrate in the sports drink. Again, it could be equally well argued that restoration of K balance improved their performance. Direct studies would be necessary to determine which factor, if either, predominates in performance enhancement.

A kinetic analysis by Sejersted (16) suggested that the Na-K pump activity is sensitive to plasma K. A plasma K level in the lower region of normokalemia during exercise should then, perhaps, be viewed from the perspective of a relative hypokalemia, given that the metabolic circumstances require substantially increased pump activity. Clinical hypokalemia may not be necessary to impart a negative performance outcome. In addition, Nye (13) presented compelling arguments that elevated arterial K is the regulator of the drive to breathe during exercise. Briefly, arterial K concentration rises in proportion to work demand and could regulate ventilatory drive. This hypothesis is supported by investigative data reviewed by Nye. If this mechanism is operative, then fluid ingestion to offset losses would seem optimal when intakes do not disrupt the hyperkalemic response, that is, when the fluid absorbed maintains plasma K at approximately 4.7–5 mEq/L during intense exercise (2, 6, 14).

### ***Total K Deficit and Sports Drinks***

Three mechanisms account for a K deficit during exercise. Sweat and gut losses of K are straightforward. In addition, renal excretion of K is coupled to sodium

retention via aldosterone (7). It is possible that the magnitude of hyperkalemia that accompanies a significant exertion might be dampened secondary to urine K loss from aldosterone release that is compensating for a drop in plasma Na. Otherwise, a more pronounced efflux of K would be required to maintain hyperkalemia. These possibilities have not been examined experimentally. Low-Na sports drinks that do not compensate for the likely effect on K balance via the aldosterone response would seem to be suboptimally formulated.

There also appears to be no benefit from ingesting any fluids that are relatively hypokalemic during the exercise bout. This action would either dilute the plasma K concentration or stimulate K efflux from intracellular pools.

### ***Pathophysiology Versus Diminished Performance***

Cells, primarily muscle, contain an aggregate of 3,000–4,200 mEq of K for mobilization. While membrane potentials and intracellular K-requiring processes may be disrupted by K efflux, clinical deficiency symptoms are not expected until approximately 2% (50–80 mEq) of this reservoir is lost. This is consistent with the report of an extreme exercise setting where heat stroke and rhabdomyolysis might be linked to intracellular K depletion (10, 11) as sweat losses approached 10 L. Thus, a serious concern regarding K provision during exercise may only exist during ultramarathon-type events.

The degree of risk associated with K depletion during exercises of the category reflected in the ACSM position stand (1) is quite low. K loss in sweat alone will not produce a worrisome deficit. A high urine K loss consequent to large volume diuresis would be required. Only a small minority of individuals consume the liters of low-Na, low-K fluids and overhydrate during exercise as would be required to trigger this depletion.

## **Conclusion**

The ACSM position stand notes the importance of sodium replacement for water balance as well as to enhance palatability and prevent the (remote) possibility of hyponatremia consequent to extreme exercise. Enhanced palatability would certainly be desirable in overcoming voluntary dehydration. Sports drinks generally include sodium at or below the recommended level of 20–30 mEq/L. Many of these drinks also incorporate carbohydrate in accord with the guidelines (1).

I suggest that potassium and sodium should be considered in tandem as electrolytes that should be present in the fluids ingested during and immediately after any strenuous exercise. During an activity that induces substantial sweat losses, it may be metabolically advantageous to provide exogenous K. Despite a clear and sustained hyperkalemia during competitive exercise, evidence of performance benefits are indirect and mixed. The hyperkalemic response during exercise is sustained by compartmental shifts that might be compromised during repeated bouts of competitive exertion.

Future studies might evaluate the relationship, if any, between plasma K and performance variables. Any benefit to be derived from preserving intracellular K concentrations could be examined as well.

K losses in sweat, expected to be 5–18 mEq, pose little or no immediate danger. A fluid replacement that includes K when sodium content is within the recommended range (1) will diminish the additional K loss triggered by renal Na retention. Sports drinks are convenient and popular, and they do include K to replace some of the K deficit from exercise. Manufacturers should continue including K in their formulations.

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### **Editor's Note**

The views contained in this commentary are the author's. Other points of view are welcome.

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