The effects of an oral glucose load on plasma K^+ and electrolyte homeostasis at rest, during high intensity intermittent exercise and recovery and on skeletal muscle Na^+,K^+-ATPase isoform abundance

C.H. Steward,[1] R. Smith,[1,2] N.K. Stepto,[1] M. Brown,[3] I. Ng[4] and M.J. McKenna,[1] [1]Institute of Sport, Exercise and Active Living (ISEAL), Victoria University, Melbourne, VIC 8001, Australia, [2]Western Hospital, Footscray, VIC 3011, Australia, [3]Department of Pharmacology, University of Melbourne, Parkville, VIC 3052, Australia and [4]Royal Melbourne Hospital, Melbourne, VIC 3000, Australia.

The regulation of K^+ and Na^+,K^+-ATPase (NKA) are of major physiologic importance, including during exercise where muscle K^+ disturbances have been linked to fatigue (McKenna *et al*., 2008). The effects of acute oral glucose supplementation on carbohydrate metabolism are well established, however the effects on muscle NKA and K^+ homeostasis are not well known. Insulin infusion reduced plasma [K^+], with K^+ uptake in the splanchnic region and skeletal muscle due to NKA stimulation (DeFronzo *et al*., 1980). This study therefore investigated the effects of glucose supplementation on endogenous insulin, arterial plasma electrolyte and acid-base homeostasis before, during and after high-intensity intermittent cycling exercise; in addition the effects on skeletal muscle NKA isoform protein abundance were examined. Participants performed two trials in a randomised cross over design, ingesting either 75 g glucose (CHO) or a placebo (CON); sixty min later participants commenced exercise, which comprised three cycling exercise bouts (EB) for 45 s at 130% $\dot{V}O_{2peak}$, followed by a fourth bout at 130% $\dot{V}O_{2peak}$, continued until fatigue. Radial arterial (a) and antecubital venous (v) blood samples taken simultaneously throughout the rest, exercise and recovery phases were analysed for plasma K^+, Na^+, H^+, glucose and Lac^- concentrations ([ion]), and their arterio-venous [ion] differences calculated. A *vastus lateralis* muscle biopsy was taken prior to glucose/placebo ingestion, immediately prior to exercise and at fatigue, and analysed for muscle NKA $\alpha_{1\text{-}3}$ and $\beta_{1\text{-}3}$ isoform protein abundance (western blotting).

The $[glucose]_a$ was greater during CHO than CON (main effect; $P<0.001$). The $[glucose]_a$ during CHO was greater than CON from 10 min after ingestion through until EB3 ($P<0.001$); a similar temporal pattern was observed for $[glucose]_v$ ($P<0.001$). Arterial plasma [insulin] was increased at each time point measured ($P<0.001$) and was greater during CHO than CON ($P<0.001$). The $[K^+]_a$ increased during exercise for both conditions, however $[K^+]_v$ only increased during CON whereas a decrease compared to rest was found for CHO. During CHO, both $[K^+]_a$ and $[K^+]_v$ were lower after glucose ingestion compared to CON, with the effect most prominent during exercise and early recovery ($P<0.05$). The $[K^+]_{a\text{-}v}$ across the forearm increased during exercise and was more positive in CHO ($p<0.05$), indicating a greater net uptake of K^+ into the relatively inactive forearm muscles during exercise. During exercise the change in $[K^+]_v$ from rest was positive for CON and remained negative following CHO, indicating an increase in *in vivo* NKA activity. Arterial [Na^+] was higher in CHO ($P<0.05$) increasing throughout the exercise period, with $[Na^+]_{a\text{-}v}$ more positive in CHO than CON ($P<0.05$) during exercise and early recovery. There was no difference in time to fatigue during the final bout between trials. There were no significant main effects for time, treatment or time–by-treatment interactions for any of the NKA α, β_1 or β_2 isoforms following glucose ingestion or for exercise. The muscle NKA β_3 protein abundance was however increased following exercise ($P<0.05$) during CON only. Thus glucose ingestion attenuated the exercise-induced rise in plasma [K^+] during exercise, likely consequent to increased [insulin]. These systemic K^+-lowering effects probably indicate increased NKA activity consistent with the greater $[K^+]_{a\text{-}v}$. This increased activity was not due to increased NKA isoform protein abundance and therefore would reflect greater *in vivo* NKA activation.

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McKenna MJ, Bangsbo J, Renaud JM. (2008) *Journal of Applied Physiology*, **104**: 288-95.