

## Increased Na<sup>+</sup>K<sup>+</sup>ATPase content is associated with improved potassium regulation during maximal exercise after sprint training in non-diabetics, but not in type 1 diabetes mellitus

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Sprint training attenuates the rise in plasma [K<sup>+</sup>] ( $\Delta$ [K<sup>+</sup>]) during intense exercise (Harmer *et al.*, 2000) and increases Na<sup>+</sup>K<sup>+</sup>adenosine triphosphatase (Na<sup>+</sup>K<sup>+</sup>ATPase) content (McKenna *et al.*, 1993) in non-diabetics. In type 1 diabetes mellitus (T1D), Na<sup>+</sup>K<sup>+</sup>ATPase content has been reported to be higher than normal (Schmidt *et al.*, 1994). However, plasma [K<sup>+</sup>] may also be higher if subjects with T1D are hyperglycaemic (Shalwitz *et al.*, 1991). The effects of intense exercise and training on plasma [K<sup>+</sup>] regulation and Na<sup>+</sup>K<sup>+</sup>ATPase content in T1D have never been examined.

Eight subjects with T1D and seven non-diabetics (CON) undertook 7 weeks of sprint cycling training. Before training, subjects cycled to exhaustion at 130%  $\dot{V}O_{2\text{peak}}$ . After training subjects cycled at the same workrate for the same duration. Subjects with T1D delayed insulin administration until after testing, which was conducted in the fasted state. Vastus lateralis biopsies obtained at rest were assayed for Na<sup>+</sup>K<sup>+</sup>ATPase content (<sup>3</sup>H]ouabain binding). Arterialised venous blood drawn during rest, exercise and recovery was analysed for plasma glucose, [K<sup>+</sup>], [Na<sup>+</sup>], catecholamines, insulin (IRI), and glucagon (IRG).

Na<sup>+</sup>K<sup>+</sup>ATPase content (T1D, 328±24; CON, 313±29 pmol•(g ww)<sup>-1</sup>) and  $\Delta$ [K<sup>+</sup>] with a single bout of maximal exercise did not differ between groups (T1D 1.3±0.1; CON, 1.6±0.3 mmol•l<sup>-1</sup>). Noradrenaline and the rise in plasma glucose were higher in T1D during exercise ( $P<0.05$ ). In late recovery in T1D, plasma glucose ( $P<0.001$ ), [K<sup>+</sup>], and IRG/IRI were higher, and plasma [Na<sup>+</sup>] lower than in CON ( $P<0.05$ ). Training increased Na<sup>+</sup>K<sup>+</sup>ATPase content by 8.2±2.2% and reduced  $\Delta$ [K<sup>+</sup>] by 21±7% ( $P<0.05$ ), with no difference between groups. These variables were correlated in CON ( $r = -0.65$ ,  $P<0.05$ ), but not T1D.

These findings demonstrate that acute regulation of plasma [K<sup>+</sup>] during a single bout of maximal exercise is similar in subjects with T1D who are relatively hypoinsulinaemic versus non-diabetics, however in late recovery, hyperglycaemia-induced hyperkalaemia may be anticipated. Sprint training enhanced plasma [K<sup>+</sup>] regulation, associated with increased Na<sup>+</sup>K<sup>+</sup>ATPase content in CON. Although K<sup>+</sup> regulation was also improved in T1D, the lack of correlation with Na<sup>+</sup>K<sup>+</sup>ATPase content suggests that other factors, e.g. altered hormonal conditions (higher noradrenaline), may play a significant role during intense exercise.

Harmer, A. R., McKenna, M. J., Sutton, J. R., Snow, R. J., Ruell, P. A., Booth, J., Thompson, M. W., Mackay, N. A., Stathis, C. G., Cramer, R. M., Carey, M. F. & Eager, D. M. (2000) Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. *Journal of Applied Physiology*, 89: 1793-1803.

McKenna, M. J., Schmidt, T. A., Hargreaves, M., Cameron, L., Skinner, S. L. & Kjeldsen, K. (1993) Sprint training increases human skeletal muscle Na<sup>+</sup>-K<sup>+</sup>-ATPase concentration and improves K<sup>+</sup> regulation. *Journal of Applied Physiology*, 75: 173-180.

Schmidt, T. A., Hasselbalch, S., Farrell, P. A., Vestergaard, H. & Kjeldsen, K. (1994) Human and rodent muscle Na<sup>+</sup>-K<sup>+</sup>-ATPase in diabetes related to insulin, starvation and training. *Journal of Applied Physiology*, 76: 2140-2146.

Shalwitz, R. A., Gingerich, R. L., McGill, J. B. & McDonald, J. M. (1991) Effect of hyperglycemia on plasma sodium and potassium concentration revisited. *Clinical Chemistry*, 37: 293-294.

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