# Changes in serum potassium concentration in asthmatic and normal subjects during exercise

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ABSTRACT Serum potassium concentrations were measured before, during, and after exercise in 32 patients with asthma (12 with exercise induced asthma), and in seven normal subjects. The changes in serum potassium in response to exercise did not differ significantly in the two groups or between those with and without exercise induced asthma. The potassium response to exercise is unlikely to be relevant to the mechanism underlying exercise induced bronchoconstriction.

It has been suggested that serum potassium may influence airway smooth muscle responsiveness and may play a part in the pathogenesis of exercise induced asthma.<sup>12</sup> In normal subjects serum potassium concentrations are known to increase linearly with increasing work intensity.<sup>3</sup> Recently Haas *et al*<sup>2</sup> found higher serum potassium concentrations after exercise in asthmatic patients than in normal subjects. As serum potassium is known to be under adrenergic control,<sup>4</sup> they suggested that these findings were in keeping with an impairment of adrenergic function in asthmatic patients.<sup>2</sup>

The aim of this study was to investigate changes in serum potassium in response to exercise in asthmatic patients with and without exercise induced asthma and in normal subjects.

## **Patients and methods**

Seven normal non-atopic subjects (six of them men; mean (SEM) age 28 (1-7) years) and 32 patients (23 of them men; mean age 26 (1-4) years) with asthma<sup>5</sup> were studied. Twenty five patients had extrinsic atopic asthma, three predominantly intrinsic asthma. The normal subjects and 12 of the asthmatic patients were having no medication. Twenty patients were taking inhaled salbutamol, adjusting the dose as required (0-600  $\mu$ g daily); six used sodium cromoglycate and one oral theophylline occasionally. Drugs were withheld for at least 12 hours before the study. All patients were symptom free and had no evidence of airways obstruction at the beginning of the study. Informed consent was obtained from all subjects and thestudy was approved by the local ethical committee.

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Accepted 28 February 1989

Physical examination and the chest radiograph showed nothing abnormal. Mean (SEM) values for baseline FEV<sub>1</sub>/VC (forced expiratory volume/vital capacity) and FEV<sub>1</sub> (% predicted) were 80.3 (2.2) and 101.4 (2.7) in the normal subjects and 78.4 (1.6) and 102.5 (2.2) in the patients. All subjects performed treadmill exercise for six minutes at a slope of 10%, the treadmill speed being adjusted to achieve a heart rate of around 180 beats/min for the last minute. If the FEV<sub>1</sub> fell by more than 10% at 0, 10, or 20 minutes after exercise the subject was considered to have exercise induced asthma.

Blood was sampled from an indwelling Cournand needle (inserted into a brachial artery before exercise) during the last 30 seconds of exercise and 10 and 20 minutes after exercise, the arterial blood being allowed to flow into the syringe with no tourniquet or suction. Potassium concentrations were determined by flame photometry within one hour of sampling.

Analysis of variance was used for comparisons and a Tukey-HSD post hoc test when appropriate. Values are given as means with standard errors in parentheses.

# Results

Exercise did not result in bronchoconstriction in the normal subjects or in 20 of the asthmatic patients. In the other 12 asthmatic subjects the mean (SEM) percentage falls in FEV, 0, 10, and 20 minutes after exercise were 17.5 (3.7), 17.9 (4.5), and 17.8(5.5). The mean maximum percentage fall in FEV, in the subjects with exercise induced asthma was 24 (5.3), compared with 4.2 (0.9) in those without exercise induced asthma. The previous treatment for those with exercise induced asthma was similar to that of those without exercise induced asthma (5 v 7 patients having no treatment, 7 v 13 inhaled salbutamol only, 4 v 2 inhaled sodium cromoglycate). There was no significant difference between the normal subjects and those with and without exercise induced asthma for treadmill speed (9.0 (0.5), 7.8 (0.3) and 8.3 (0.3) km/h), or for maximum heart rate (187 (0.6), 183 (3), and 186 (4) beats/ min). The decrease in pH at peak exercise was significantly greater (p < 0.05) in the normal subjects (7.17 (0.03)) than in those with (7.26 (0.03)) or without exercise induced asthma (7.26(0.03)).

There were no significant differences in serum potassium concentration between the three groups at any time (table).

### Discussion

The main finding of this study is that asthmatic subjects had similar increases in arterial serum potassium concentration in

Mean (SEM) changes in serum potassium concentrations (mmol/l) before, during, and after exercise in normal subjects and asthmatic patients with and without exercise induced asthma (EIA)

	Before exercise	Peak exercise	After exercise	
			10 min	20 min
Normal		2		
subjects	3.7 (0.09)	5·6 (0·18)	3·4 (0·08)	3.6 (0.08)
Patients with EIA	3.7 (0.04)	5.3 (0.14)	3.7 (0.07)	3.8 (0.06)
Patients without EIA	3.7 (0.05)	5-3 (0-14)	3.7 (0.05)	3.7 (0.05)

response to exercise whether or not they developed exercise induced asthma. There was no significant difference in serum potassium concentration between those with and without exercise induced asthma at any time. The serum potassium concentration had returned to pre-exercise values in all three groups 20 minutes after exercise, though bronchoconstriction persisted in the patients with exercise induced asthma. These findings suggest that differences in serum potassium are not relevant to the mechanism underlying exercise induced asthma—by potentiating cholinergic activity in the airways, for example.<sup>1</sup>

There was no significant difference in serum potassium concentrations at peak exercise between the three groups. The work loads between the two asthmatic groups were well matched. As none of the normal subjects or patients took regular exercise the larger fall in arterial pH at peak exercise is likely to be the result of a slightly heavier work load in the normal subjects. Nevertheless, the potassium response to exercise was of the same order of magnitude in all three groups. This result is contrary to the results of Haas *et al*,<sup>2</sup> who found raised venous serum potassium concentration in asthmatic subjects "at peak exercise" and 10 minutes after exercise. In their study, however, the "peak exercise," blood samples were taken during the first minute after exercise, a time at which serum potassium is known to fall rapidly.<sup>46</sup> Ten minutes after exercise serum potassium concentration was

slightly higher in the asthmatic than in the normal subjects in the present study, though the difference was not significant (p = 0.06). Some of the discrepancies between the two studies t publ might be due to variations in timing of blood samples and differences between arterial and venous serum potassium during periods of rapid change.6 With arterial blood potaslishe sium concentration is independent of the site of puncture and of peripheral influences such as local muscle activity and haemolysis. Different exercise protocols were applied in the two studies and most patients in the study of Haas et al<sup>2</sup> had some pre-exercise airways obstruction, unlike the patients in \_ our study. The mean maximum post-exercise percentage fall in FEV, was 24% in the current study compared with 16% in the study of Haas et al.<sup>2</sup>

In the current study the potassium response to exercise was similar in asthmatic patients with and without exercise induced asthma and is thus not likely to be relevant to the rechanism of exercise induced asthma.

I thank Mr K Kyd for his expert technical assistance and Mr A Caine for his help with editing.

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