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Effect of Acute Submaximal Exercise on Plasma Renin and Aldosterone Levels in Sedentary People

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This study aimed to examine how acute submaximal exercise affected plasma renin and aldosterone levels in sedentary people. The study registered 7 healthy male sedentary subjects whose mean age was $23.00 \pm$ 1.29 years and mean body weight was 74.00 ± 10.19 kg. Before starting the trial, the subjects were put on an Astrand Bicycle Ergometer test to identify Max VO₂ and above the threshold heart rates. The subjects with above the threshold heart rates were exercised on ergometric bicycles for 0.5 h at 70 % of Max VO₂. Blood samples were collected from the subjects before beginning the experiment. Second round of blood samples were collected immediately after the exercise ended. Third and fourth rounds of blood samples were collected after 2 and 24 h of exercise, respectively. Plasma renin and aldosterone levels were measured in the collected samples (using RIA method). Renin and aldosterone levels measured immediately after exercise were significantly higher than those before exercise, as well as those 2 and 24 h after exercise (p < 0.05). Renin and aldosterone levels 2 h after exercise did not differ from preexercise levels. The lowest renin and aldosterone levels were found in the measurements performed 24 h after exercise (p < 0.05). Results of the study demonstrate that acute submaximal exercise brings about significant changes in renin and aldosterone levels of sedentary people.

Key Words: Submaximal exercise, Renin, Aldosterone.

INTRODUCTION

Renin-angiotensin-aldosterone (RAA) system, as well as hormones like ANP (Atrial Natriuretic Peptide) and ADH (Antidiuretic Hormone), plays an important role in the regulation of physiological responses to exercise, such as adaptation of cardiovascular activity and fluid-electrolyte balance. Renin-angiotensin-aldosterone system has a central part in the regulation and physiological maintenance of fluid-electrolyte balance, blood volume and arterial blood pressure¹⁻⁴. Long-term exercise leads to loss of fluids and salt from the body and changes fluid-electrolyte balance. This in turn increases hyperosmotic hypovolemia and circulating concentrations of fluid-regulating hormones⁵. Physical activity activates β -adrenoreceptors on juxtaglomerular

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cells, thereby increasing the renal sympathetic tonus, which in turn results in elevated renin secretion. Increase in renal sympathetic tonus leads to vasoconstriction of the afferent arteriole, which causes renal hypoperfusion and renin secretion^{1,4-6}.

The role of aldosterone which is known as a sodium-retaining hormone that is controlled by angiotensin II and that prevents loss of sodium due to perspiration during long-term exercise was first emphasized by Costill *et al.*⁷. Buono and Yeager⁸ reported that renin and aldosterone levels increased during and after exercise and were restored to pre-exercise levels within 6 to 12 h resting period, while sodium and water excretion remained suppressed for up to 48 h. In a study carried out to determine plasma concentrations that maintain the fluid-electrolyte balance in athletes, Pastene *et al.*⁹ observed significant increases in plasma renin and aldosterone concentrations before and after submaximal exercise.

In the present study, it is aimed to examine how plasma renin and aldosterone levels in sedentary people were affected by acute submaximal exercise.

EXPERIMENTAL

The study included 7 healthy sedentary males who were students in different departments of Selcuk University and whose mean age was 23 ± 1.29 years and mean body weight was 74 ± 10.19 kg.

Exercise protocol: The subjects were put to an Astrand Bicycle Ergometer test before exercise to determine Max VO₂ and above the threshold heart rate. When the subjects had above the threshold heart rates, they were exercised on Monark 814-E and 818 model ergometric bicycles for 0.5 h at 70 % of Max VO₂. Blood samples were collected from the subjects before they started exercise. Second round of blood samples were collected immediately after the exercise test ended. Third and fourth rounds of blood samples were collected 2 and 24 h after exercise, respectively. Plasma renin and aldosterone levels were measured in the collected blood samples.

Plasma renin and aldosterone measurements: Blood samples duly collected from elbow vena were transferred into tubes containing ethylenediamine tetraacetic acid (EDTA) and centrifuged at 3500 rpm for 15 min at 4 °C to obtain plasma. From the plasma samples plasma renin level was determined using Irma make DSL-25100 Active Renin Irma kit and aldosterone level was determined using Irma make DSL-8600 Active Aldosterone kit by radioimmunoassay method in the Gamma Counter located in the Central Biochemistry Laboratory of Selcuk University Meram Medical School.

Statistical analyses: SPSS package software was utilized in the statistical analyses of the data obtained. Arithmetic means of the measured parameters were calculated. "Paired t" test was employed in identifying the differences between measurement times.

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RESULTS AND DISCUSSION

In the study, the highest renin levels were obtained immediately after exercise (p < 0.05). Renin levels measured 2 h after exercise were not different than preexercise levels and levels measured 24 h after exercise. Renin levels found 24 h after exercise were significantly lower than pre-exercise levels (p < 0.05), though not different than the levels 2 h after exercise (Table-1 and Fig. 1).

The highest aldosterone levels in the study were obtained immediately after exercise (p < 0.05). Aldosterone levels after 2 h of exercise were not different than pre-exercise levels, but significantly higher than those measured 24 h after exercise (p < 0.05). Aldosterone levels found 24 h after exercise were significantly lower than both pre-exercise levels and levels measured 2 h after exercise (Table-1 and Fig. 1) (p < 0.05).

| TABLE-1 |
|---|
| PLASMA RENIN AND ALDOSTERONE LEVELS OF SUBJECTS (n = 7) |

| Parameters | Before exercise | Immediate after exercise | 2 h after exercise | 24 h after exercise |
|---------------------|------------------|--------------------------|-----------------------|---------------------|
| Renin (pg/mL) | 12.12 ± 1.68 b | 34.19 ± 5.97 a | 11.94 ± 1.89 bc | 10.28 ± 0.82 c |
| Aldosterone (pg/mL) | 146.53 ± 14.66 b | 217.82 ± 17.26 a | 145.21 ± 19.89 b | 129.94 ± 5.38 c |
| Aldosterone (pg/mL) | | | | 129.94 ± 5.38 C |

abc = Different letters in same line are significant for parameters (p < 0.05).

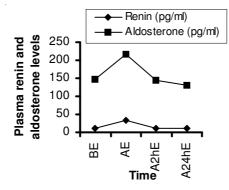


Fig. 1. Means of renin and aldosterone levels (pg/mL). BE = Before exercise, AE = Immediately after exercise, A2hE = 2h after exercise, A24hE = 24h after exercise

In the study significant increases were observed in plasma renin levels immediately after exercise. The fact that Convertino *et al.*¹⁰, Kosunen *et al.*¹¹ and Kotchen *et al.*¹² all reported significant increases in plasma renin levels with submaximal and maximal exercise (70-100 % Max VO₂) in studies investigating effects of exercise on sedentary subjects lends support to the increased renin levels obtained after exercise in present study. The increased renin levels found just after exercise in present study may have resulted from fluid-electrolyte loss that occurred due to

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exercise in present subjects. Likewise, several researchers observed significant elevations in plasma renin activities immediately after exercise and argued that this significant increase in plasma renin activities resulted from fluid-electrolyte loss caused by exercise¹³⁻¹⁶. The significant decline obtained in renin levels 2 h following exercise in present study may be explained by post-exercise resting and replacement of lost fluid-electrolyte amount by water intake. Decreased renin activities obtained 2 h after exercise in present study were reported by several other researchers as well^{4,17-20}. The fact that plasma renin levels measured 24 h after exercise were found significantly lower than pre-exercise levels may be attributed to restoration of fluid-electrolyte balance to its pre-exercise state and the resulting decrease of renin level under normal limits, as reported by Staessen *et al.*²¹.

The significant increase in aldosterone levels immediately after the exercise test, relative to pre-exercise levels in present study may be explained by an increase in plasma aldosterone level parallel to the exercise-associated increase in plasma renin level. Likewise, it was emphasized in several studies that plasma aldosterone levels increased with exercise in trained people^{15,16,20-22}. The increase found in aldosterone levels following exercise may be explained by the adaptation mechanism that arises in response to metabolic changes caused by exercise²²⁻²⁶. Similarly restoration of aldosterone levels measured 2 h after exercise to pre-exercise levels is parallel to the results of studies on this topic^{19,21,27,28}. Decreased aldosterone levels found in the measurements 24 h after exercise in present study can be seen as a result of the restoration of fluid-electrolyte balance in extracelular fluid, as well as the restoration of osmotic pressure, as reported by researchers working on this topic^{16,18,20}.

Conclusion

It can be said that acute submaximal exercise used in this study has significant effects on plasma renin and aldosterone levels of sedentary people.

REFERENCES

- 1. F. Fallo, J.S. Med. Phys. Fit., 33, 306 (1993).
- R. Mogulkoc, O. Dogru, A.K. Baltaci, B. Yilmaz and H. Kelestimur, *Neuro. Endocrinol. Lett.*, 21, 35 (2000).
- 3. R. Mogulkoc and A.K. Baltaci, Life Sci., 79, 817 (2006).
- 4. B. Melin, C. Jimenez, G. Savourey, J. Bittel, J.M. Cottet-Emard, A.M. Pequignot, A.M. Allevard and C. Gharib, *Eur. J. Appl. Phys.*, **76**, 320 (1997).
- O.M. Podhorska, P. Dziegel, A. Gomulkiewez, K.B. Dolinska, C.E. Murawska, Z. Jethon and M. Zabel, *Rocz. Akad. Medy. Bialy.*, 49, 8 (2004).
- 6. A. Ekmekci, A. Canberk and K. Berkman, Renin-Angiotensin Sistemi, Angiotensinler, Angiotensin Reseptörleri ve Angiotensin Antogonistleri, Park Matbaacilik, Istanbul (2000).
- 7. D.L. Costill, G. Branam, W. Fink and R. Nelson, *Med. Sci. S.*, **8**, 209 (1976).
- 8. M.J. Buono and J.E. Yeager, J.S. Med. Phys. Fit., 31, 48 (1991).
- 9. J. Pastene, M. Germain, A.M. Allevard, C. Gharib and J.R. Lacour, *Eur. J. Appl Phys. Occup. Phys.*, **73**, 49 (1996).
- 10. V.A. Convertino, L.C. Keil and E.M. Bernauer, J. Appl. Phys., 50, 123 (1981).

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- 11. K.J. Kosunen and A.J. Pakarinen, J. Appl. Phys., 41, 26 (1976).
- 12. T.A. Kotchen, L.H. Hartley, T.W. Rice, E.H. Mougey, L.G. Jones and J.W. Mason, *J. Appl. Phys.*, **31**, 178 (1971).
- B. Melin, J.P. Eclache, G. Gelen, G. Annat, A.M. Allevard, E. Jarsaillon, A. Zebidi, J.J. Legros and C.I. Gharib, *Eur. J. Appl. Phys.*, 44, 141 (1980).
- 14. H.U. Attenkirch, R. Gerzer, K.A. Kirsch, J. Weil, B. Heyduck, I. Schultes and L. Röcker, *Eur. J. Appl. Phys.*, **61**, 209 (1990).
- 15. O. Bocqueraz, N. Koulmann, B. Guigas, C. Jimenez and B. Melin, *Med. Sci. S. Exer.*, **36**, 1730 (2004).
- S.A. Kavouras, L.E. Armstrong, C.M. Maresh, D.J. Casa, J.A. Herrera-Soto, T.P. Scheett, J. Stoppani, G.W. Mack and W.J. Kraemer, *J. Appl. Phys.*, 100, 442 (2006).
- 17. M. Aurell and P. Vikgren, Eur. J. Appl. Phys., 31, 839 (1971).
- S. Kozlowski, Z. Brzezinska, K. Nazar, W. Kowalski and M. Franczyk, *Clin. Sci. Mol. Med.*, 15, 723 (1973).
- 19. C.M. Maresh, B.C. Wang and K.L. Goetz, Eur. J. Appl. Phys., 54, 398 (1985).
- 20. R.M. Morgan, M.J. Patterson and A. Nimmo, The A. Coll. S. Med., 182, 37 (2004).
- 21. J. Staessen, R. Fagard, P. Hepsel, P. Lijnen, L. Vanhees and A. Amery, J. Appl. Phys., 63, 188 (1987).
- 22. B.J. Freund, E.M. Shizuru, G.M. Hashiro and J.R. Claybaugh, J. Appl. Phys., 70, 900 (1991).
- 23. M.D. Guyton and J.E. Hall, Textbook of Medical Physiologh, Tibbi Fizyoloji, (Trans: H. Çavusoglu), Yüce Yayinlari, Alemdar Ofset, Istanbul, edn. 9 (1996).
- 24. B. Yilmaz, Hormonlar ve Üreme Fizyolojisi, Feryal Matbaa, 1.Basim, Ankara, pp. 247-371 (1999).
- 25. M. Gunay and I. Cicioglu, Spor Fizyolojisi, Gazi Kitabevi, Baran ofset, 1.baski, Ankara (2001).
- 26. A. Noyan, Yasamda ve HekimLikte Fizyoloji", 12. Baski, Meteksan An. Sir, Ankara (2000).
- E.S. Williams, M.P. Ward, J.S. Milledge, W.R. Withey, M.W.J. Older and M.L. Forsling, *Clin. Sci.*, **56**, 305 (1979).
- J.S. Milledge, E.I. Bryson, D.M. Catley, R. Hesp, N. Luff, B.D. Minty, M.W. Older, N.N. Payne, M.P. Ward and W.R. Withey, *Clin. Sci.*, 62, 595 (1982).

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