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# Metabolic stress and high intensity exercise

**Julien S Baker*[1] and Duncan Buchan[1]**

[1]Institute of Clinical Exercise and Health Science, Applied Physiology Research Laboratory, School of Science and Sport, University of the West of Scotland, UK

Exercise-induced catecholamine responses are related to exercise intensity and are affected by the preceding training regimen. Although most investigations of catecholamine responses to training have utilized aerobic exercise models, it is likely that anaerobic training regimens will produce a considerably different physiological profile. Furthermore, depending on the acute resistance exercise variables (i.e., choice of exercise, order of exercise, rest intervals, load, volume of exercise), considerable physiological variation exists between different exercise protocols. To date, few studies have investigated catecholamine responses to an anaerobic overtraining models.

Total stress response to exercise is reflected by the adrenal medulla response. This may be measured by the accumulation of adrenaline (A) and noradrenaline (NA). This increase may be markedly influenced by the effect of physical exertion, cold and emotional factors. Plasma catecholamine levels also increase as a result of both increases in the duration of exercise and the severity of exercise [1]. The plasma concentrations of catecholamines have been shown to increase during different types of aerobic and anaerobic exercises [2]. However, it has been found that plasma adrenaline increase in heavy high intensity exercise may be higher than in aerobic exercise [3].

Different high intensity activities such as sprinting [4] and cycle ergometry [5,6] as well as heavy resistance exercise training [7] have all been found to elevate plasma catecholamine levels.

At identical submaximal exercise levels several authors observed [8] a lesser increase in free plasma adrenaline (A) and noradrenaline (NA) in endurance trained athletes than in sedentary subjects. Conversely, Kjaer *et al.* [9,10] found that endurance trained athletes compared to untrained ones exhibited not only similar plasma NA but also higher plasma A concentrations. Brief high intensity exhaustive exercise has been shown to increase plasma catecholamine levels to values higher than those observed during aerobic activity [8]. Therefore it may be possible that sprinters exhibit a higher adrenal response to sympathetic activity. It has often been emphasised that feedback mechanisms are important for the hormonal response to exercise [11]. A reduction in plasma glucose levels during exercise gives rise to an increase in the plasma concentration of adrenaline, and it has been postulated that increasing adrenaline secretion may function as a safety backup mechanism, preventing hypoglycaemia and lack of substrate in the working muscle [12]. It seems, however, that rapid changes in endocrine and metabolic responses found at the onset and the cessation of exercise, occur too quickly to be explained by feedback mechanisms only. This may be partly explained by the direct stimulation from the motor centres of the brain, the feed - forward or central command mechanisms which are also involved with respiratory and circulatory regulation [13]. These mechanisms will also be active in substrate mobilisation during exercise. Physical training may alter the hormonal response of several hormones to a given exercise work load. The release and rate of release of hormones are controlled in response to specific needs.

The rates of secretion should be related to their metabolic functions and needs of a particular circumstance ie whether high or low intensity [14].

Previous experiments have demonstrated the importance of anaerobic glycolysis in the resynthesis of adenosine triphosphate (ATP) during high intensity performance.

Exercise of this intensity although predominantly anaerobic, provides a severe challenge to the control of the cardiovascular system and elevates sympathetic activity.

The stimulation of glycogenolysis by adrenaline has been demonstrated, and significant correlations have been observed between ATP resynthesis and plasma adrenaline concentration [4]. These studies have also found that during maximal exercise large concentrations in circulating adrenaline and noradrenaline occur. Therefore, catecholamine consentration has a significant role to play in metabolic function during high intensitiy anaerobic metabolism.

## References

1. Åstrand PO, Rodahl K (1986) Textbook of Work Physiology. New York: McGraw Hill Book Company.
2. Galbo H (1986) Autonomic neuroendocrine responses to exercise. *Scandinavian Journal of Sports Science* 8: 3-17.
3. Kindermann, W, Schnabel A, Schmitt WM, Biro G, Cassens J, et al. (1982), Catecholamines, growth hormone, cortisol, insulin and sex hormones in anaerobic and aerobic exercise. *Eur J Appl Physiol Occup Physiol* 49: 389-399. [Crossref]
4. Cheetham M, Boobis L, Brooks S, Williams C (1986) Human muscle metabolism during sprint running. *J Appl Physiol (1985)* 61: 54-60.
5. Gaitanos GC, Williams C, Boobis LH, Brooks S (1993) Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol (1985)*75: 712-719.
6. Gratas-Delamarche A, Le Cam R, Delamarche P, Monnier M, Koubi H (1994) Lactate and catecholamine responses in male and female sprinters during a Wingate test. *Eur J Appl Physiol Occup Physiol* 68: 362-366. [Crossref]
7. Pullinen T, Nicol C, MacDonald E, KomiPV (1999) Plasma catecholamine responses to four resistance exercise tests in men and women. *Eur J Appl Physiol Occup Physiol* 80: 125-131. [Crossref]
8. Brooks S, Cheetham ME, Williams C (1985) Endurance training and the catecholamine response to maximal exercise. *J Physiol* 361: 81.
9. Kjaer M, Christensen NJ, Sonne B, Richter EA, Galbo H (1985) Effect of exercise on epinephrine turnover in trained and untrained male subjects. *J Appl Physiol (1985)* 59: 1061-1067. [Crossref]

***Correspondence to:*** Julien S Baker, Institute of Clinical Exercise and Health Science, Applied Physiology Research Laboratory, School of Science and Sport, University of the West of Scotland, Hamilton, Lanarkshire, Scotland, ML3 OJB, Tel: 01698 283100 ext: 8271; Fax: 01698 894404; E-mail: jsbaker@uws.ac.uk

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10. Kjaer M, Galbo H (1988) Effect of physical training on the capacity to secrete epinephrine. *J Appl Physiol (1985)* 64: 11-16. [Crossref]

11. Newsholme EA, Leech AR (1983) Biochemistry for the Medical Sciences. Chichester: John Wiley and Sons.

12. Kjaer M (1989) Epinephrine and some other hormonal responses to exercise in man: with special reference to physical training. *Int J Sports Med* 10: 2-15. [Crossref]

13. Rowell LB, Blackmon JR (1986) Lack of sympathetic vasoconstriction in hypoxemic humans at rest. *Am J Physiol* 251: H562-570. [Crossref]

14. Brooks S, Burrin J, Cheetham ME, Hall GM, Yeo T, et al. (1988) The responses of the catecholamines and beta-endorphin to brief maximal exercise in man. *Eur J ApplPhysiol Occup Physiol* 57: 230-234. [Crossref]