

# Lawrence Berkeley National Laboratory

## Recent Work

### Title

GROWTH HORMONE RESPONSE TO CONTINUOUS AND INTERMITTENT EXERCISE

### Permalink

<https://escholarship.org/uc/item/4s92c12t>

### Author

Karagiorgos, Athanase

### Publication Date

1977-07-01

0 0 7 3 4 8 3 3 0 0 1

11/2/78

Submitted to Journal of Applied  
Physiology

LBL-6499  
Preprint c. 1

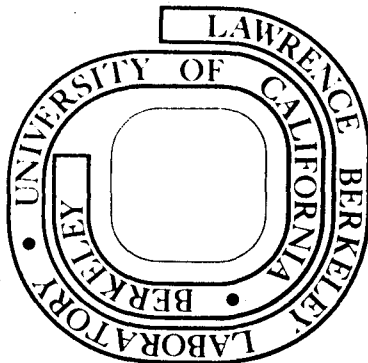
GROWTH HORMONE RESPONSE TO  
CONTINUOUS AND INTERMITTENT EXERCISE

Athanase Karagiorgos, Joseph F. Garcia, and  
George A. Brooks

July 1977

Prepared for the U. S. Energy Research and  
Development Administration under Contract W-7405-ENG-48

**For Reference**  
Not to be taken from this room



LBL-6499  
21

## **DISCLAIMER**

This document was prepared as an account of work sponsored by the United States Government. While this document is believed to contain correct information, neither the United States Government nor any agency thereof, nor the Regents of the University of California, nor any of their employees, makes any warranty, express or implied, or assumes any legal responsibility for the accuracy, completeness, or usefulness of any information, apparatus, product, or process disclosed, or represents that its use would not infringe privately owned rights. Reference herein to any specific commercial product, process, or service by its trade name, trademark, manufacturer, or otherwise, does not necessarily constitute or imply its endorsement, recommendation, or favoring by the United States Government or any agency thereof, or the Regents of the University of California. The views and opinions of authors expressed herein do not necessarily state or reflect those of the United States Government or any agency thereof or the Regents of the University of California.

0 0 0 0 4 8 0 3 0 0 2

GROWTH HORMONE RESPONSE TO  
CONTINUOUS AND INTERMITTENT EXERCISE

By: Athanase Karagiorgos,  
Joseph F. Garcia, and  
George A. Brooks

Exercise Physiology Laboratory,  
Department of Physical Education;  
Lawrence Berkeley Laboratory; and  
Laboratory of Chemical Biodynamics (LBL),  
University of California, Berkeley, CA 94720

Running Head: Growth Hormone In Exercise

Please send all correspondence to:

Dr. G.A. Brooks

Department of Physical Education,

103 Harmon Gym

University of California

Berkeley, CA 94720

## ABSTRACT: GROWTH HORMONE RESPONSE TO CONTINUOUS AND INTERMITTENT EXERCISE

To test a possible association between growth hormone (hGH) secretion during exercise and anaerobiosis, equivalent continuous and intermittent bouts of bicycle ergometer work were employed to observe the relationship between circulating levels of hGH, free fatty acids (FFA), blood glucose (GLUC), alanine (ALA), lactic acid (LA), and pyruvic acid (PA). Ten males (20 to 30 yrs.) were observed during 40 min. of continuous exercise (CE, 45% of the minimum load which elicited  $\dot{V}O_2$  max.), and during 20 bouts of intermittent exercise (IE, 1 min. on/off @ 2x CE work rate). Resting hGH was 1 to 2 ng/ml. After a 15 min. lag, hGH rose continuously, but during IE hGH tended to be higher ( $12.1 \pm 1.4$ ) than during CE ( $9.7 \pm 1.6$  ng/ml,  $\bar{x} \pm SEM$ ). In both exercise conditions FFA demonstrated an initial fall and then a continuous secondary rise with higher peak values during CE ( $0.516 \pm .058$ ) than during IE ( $0.388 \pm .048$  mEq/L.). PA and LA rose initially during CE, but then declined before reaching steady levels. During IE, LA and PA increased continuously reaching values 3x greater than during CE. ALA rose progressively during CE and IE, but was significantly higher during IE ( $442.2 \pm 29.3$  vs.  $367.9 \pm 30.9$   $\mu M$ ). GLUC also tended to be higher during IE ( $84.0 \pm 5.8$ ) than during CE ( $76.5 \pm 5$  mg%). Considering both CE and IE, no significant correlation was found between peak values of hGH and metabolite concentrations, rectal T., or  $O_2$  deficit. The results are interpreted to mean that hGH response to work is not directly related to "anaerobiosis".

Key Words: Exertion, Work, Growth Hormone, Blood, Metabolism, Lactic Acid, Anaerobiosis.

### INTRODUCTION

Since Yalow and Berson (31) developed radioimmunoassays for detection of hormones present in minute concentrations, the secretion of human growth hormone (hGH) has been studied under a variety of conditions. During fasting hGH plays a role in mobilizing free fatty acids in response to declining blood glucose. During exercise, a number of factors have been associated with the control of hGH secretion. Hartog, et al. (14) reported a rise of hGH during exercise which did not depend upon a prior mobilization of fatty acids and therefore, suggested that fat mobilization could inhibit the continued secretion of hGH. Sutton, et al. (28) have supplied data which suggest a relationship between blood hGH and lactate concentrations. However, Lassarre, et al. (17) have reported no correlation between hGH and lactate, but instead their data suggested a significant relationship between hGH and  $O_2$  deficit. A change of internal temperature during exercise has also been correlated with hGH response by Buchler (3).

In view of the possible role of lactate and other by-products of metabolism in determining the hGH response to exercise, we sought an approach which would maintain an equivalence of external work, but which would result in divergent patterns of blood lactate and other metabolites. For this reason we employed work equivalent bouts of continuous (CE) and intermittent (IE) exercise.

METHODS

Table 1 Ten healthy, fit, male volunteers, with no family history of diabetes or other endocrine diseases (Table 1), were used as subjects in this study. All subjects were familiar with bicycle ergometer exercise and other laboratory procedures prior to testing. Prior to experimentation, written informed consent was obtained and subjects were tested twice for the determination of  $\dot{V}O_2$  max. Experimental testing was carried out in the morning after an overnight fast. Upon reporting to the laboratory a catheter was placed in a forearm vein for sampling of blood. After a half hour rest period, subjects performed either 40 minutes of continuous exercise, or 20, one minute exercise bouts which were interspersed with one min. rest periods. For IE the work was 90% of the minimum which elicited  $\dot{V}O_2$  max. during CE. For CE the external work output was half that during IE. The order of continuous and intermittent exercise sessions was rotated and balanced across subjects. Blood was sampled intermittently during rest, exercise and recovery for determination of hGH, lactate (LA), glucose (GLUC), free fatty acids (FFA), pyruvate (PA), and alanine (ALA). Blood samples for LA, GLUC, PA, and ALA determinations were drawn with a minimum of stasis and immediately deproteinized in cold 6%  $HClO_4$  by Vortex mixing and centrifugation. Blood samples for FFA and hGH were collected in chilled, heparinized tubes and quickly cold centrifuged.

Lactate and pyruvate were determined enzymatically (enzyme kits, Sigma Chemical Co.). Glucose concentration was determined by the glucose oxidase method (Glucostat Reagent Kit, Worthington Biochem. Co.). Alanine was determined enzymatically as described by Pfleiderer (22). Free fatty acids

were determined colorimetrically by the method of Falhot, et al. (6), and hGH was determined by the radiomunoassay method of Yalow and Berson as modified by Garcia, et al. (8). Initial  $O_2$  deficit was computed as the difference between the steady-rate  $\dot{V}O_2$  and the actual  $\dot{V}O_2$  during the first five minutes of work for continuous exercise. Rectal temperature ( $T_R$ ) and electrocardiogram (ECG) were recorded on a Gilson recorder. Inspired ventilatory volume was recorded on the servo channel of the Gilson recorder from the potentiometer output of a Parkinson-Cowan gasometer. The entire expired air sample was collected in meteorological balloons during  $\dot{V}O_2$  determination periods and analyzed on Applied Electrochemistry S3A  $O_2$  and Beckman LB-2  $CO_2$  analyzers. The correction for  $H_2O$  vapor was made as outlined by Beaver, et al. (1).

For statistical purposes, means and variances were calculated and paired t-tests and correlation analyses were performed.

### RESULTS

Figure 1 Prior to exercise hGH was on the order of 1 to 2 ng/ml. (Figure 1). During both continuous and intermittent exercise, hGH remained essentially unchanged for 15 minutes and then began a continuous rise reaching maximum levels of  $9.7 \pm 1.6$  and  $12.1 \pm 1.4$  ng/ml. ( $\bar{X} \pm SEM$ ) for CE and IE, respectively. Although hGH concentrations tended to be higher during IE, the difference did not reach statistical significance (as determined by paired t-tests) until 20 minutes of recovery.

Figure 2 Blood lactate concentrations (Figure 2) at rest were found to be  $0.871 \pm .072$  and  $0.955 \pm .125$  mM prior to CE and IE, respectively. During CE LA increased initially from resting levels to  $2.080 \pm .282$  mM after 10 min. of exercise, but then declined before reaching steady levels of approximately 1.5 mM.



During IE LA increased from resting levels to  $4.513 \pm .709$  mM at the end of exercise reaching values of about five times greater than during rest.

Figure 3 Pyruvate (Figure 3) followed the same trend as LA. From resting levels of  $59.1 \pm 5.3$   $\mu$ M prior to CE, PA increased during CE to  $73 \pm 7.5$   $\mu$ M at 20 min., and then declined before reaching a steady level of approximately 70  $\mu$ M. During IE, PA increased from resting levels of  $69.2 \pm 6.5$ , to  $187 \pm 20$   $\mu$ M.

Figure 4 Blood glucose concentrations (Figure 4) were found to be  $74 \pm 4.8$  and  $75 \pm 5.3$  mg% prior to CE and IE, respectively. Although GLUC concentration during IE tended to be higher than during CE, the difference reached statistical significance only at 30 min. of exercise, where the mean values for CE and IE were  $75 \pm 3.8$  and  $87 \pm 3.4$  mg/100 ml, respectively ( $p < .05$ ).

Figure 5 Alanine concentration (Figure 5) rose progressively from resting levels of  $255 \pm 26$  and  $264 \pm 28$   $\mu$ M, to  $381 \pm 26$  and  $452 \pm 28$   $\mu$ M at 30 min. of CE and IE, respectively. Alanine concentration was significantly higher toward the end of IE than during the comparable period of CE ( $p < .05$ ).

Figure 6 Free fatty acids (Figure 6) at rest were found to be on the order of  $0.438 \pm .052$  and  $0.458 \pm .036$  mEq./liter prior to CE and IE, respectively. FFA demonstrated an initial fall in both experimental conditions, but then increased reaching levels of  $0.516 \pm .058$  and  $0.388 \pm .048$  mEq/liter for CE and IE at the end of exercise periods. During recovery, the secondary rise to  $0.751 \pm .088$  mEq/liter following CE was statistically greater than that following IE ( $0.480 \pm .040$  mEq/liter) ( $p < .05$ ).

Figure 7 Rectal temperature (Figure 7) rose from  $37.3 \pm 0.17$  and  $37.1 \pm 0.19^{\circ}$ C at rest, to  $38.1 \pm 0.14$  and  $38.3 \pm 0.13^{\circ}$ C at the end of CE and IE, respectively.

After 20 min. of recovery,  $T_R$  declined to 37.8 and 37.9°C following both CE and IE, respectively.

To test for a possible association between blood borne metabolites and other physiological variables with hGH levels, correlation analyses were performed. Parameters in the correlation matrices included: hGH, LA, PA, ALA, GLUC, FFA,  $T_R$ , %  $\dot{V}O_2$  max,  $\dot{V}O_2$  (L./min.), RQ, and  $O_2$  deficit. With pooled data the only significant correlations were between LA and PA, and between %  $\dot{V}O_2$  max and  $\dot{V}O_2$ . When correlations were run at specific time points within exercise treatments, the only significant correlations were between hGH and PA at rest, and between hGH and  $T_R$  at 30 and 40 min. of CE.  $O_2$  deficit during CE could also not be correlated with peak hGH. Although the ALA response to exercise (Figure 5) was similar to that for LA (Figure 2) and PA (Figure 3), ALA did not correlate significantly with either PA or LA.

## DISCUSSION

Observations of blood lactate concentration suggested a combination of elevated lactate production and diminished removal associated with circulatory lag during intermittent exercise. Despite three-fold greater LA levels during IE than CE (Figure 2), we were unable to establish a clear difference between hGH responses in the two exercises (Figure 1). Therefore, we must conclude that lactate concentration is not a determining factor in controlling the circulating level of hGH during exercise. In this regard our data are similar to those of Hartley, et al. (12,13). Our data further show that concentrations of PA and ALA (Figures 3 and 5), other consequent products of rapid glycolysis, are also apparently not related to hGH levels.

Our inability to correlate  $O_2$  deficit with hGH response is further evidence that hGH secretion is unrelated to anaerobiosis during exercise. We, therefore, interpret our results to indicate that hGH response is not correlated with the accumulation of an "anaerobic metabolite" or to systemic hypoxia. Indeed it seems difficult to see how the  $O_2$  deficit could constitute a viable stimulus for the secretion of hGH.

Previously, Schwarz, et al. (27) obtained results similar to ours and could not correlate hGH responses with metabolite concentrations during exercise. They, therefore, attributed the hGH response to physical stress rather than to an increase in energy expenditure. In our experiments, subjects perceived IE to be more stressful than CE, but hGH responses were similar in both treatments. Our results are, therefore, not consistent with the hypothesis that stress is a major determinant of hGH response during exercise.

During CE and IE an initial fall of plasma FFA concentration (Figure 6) was followed by a gradual increase probably as a result of enhanced lipolysis.

This was particularly evident during the CE, where there occurred a large FFA overshoot during the post-exercise period. Similar results have been shown by Carlson (4) and Gollnick (9). Although hGH was slightly higher during IE than CE, during IE the FFA concentrations were found to be significantly lower than during CE, and the post-exercise overshoot was less profound. This could be attributed to the three-fold higher concentration of lactate during IE, which may have inhibited lipolysis (15,16).

Lipman, et al. (18) have shown that infusion of lipids during sleep, which resulted in an elevation of FFA, could induce inhibition of sleep-related hGH release. This negative feedback effect of FFA on hGH was not apparent in the present study. Support for this conclusion is to be found in the data of Hansen (11) who has shown that hGH hyper-response to exercise is unaffected by intravenous infusion of lipids and heparin.

Our finding of higher blood glucose during IE than during CE (Figure 4) may reflect a greater participation of catecholamines during IE. On the basis of our respiratory exchange data and the glycogen depletion studies of others, we can assume that the elevated blood glucose during IE was due to greater mobilization of carbohydrates rather than to lesser utilization. Although an increase in glucose uptake and utilization by exercising muscle has been reported (26), we cannot support the possibility that this increase in glucose uptake results from an increase of insulin secretion during exercise. If anything, circulating insulin levels are decreased during exercise (12,13, 23). Dieterle, et al. (5) have suggested that the increased GLUC and FFA uptake by exercising muscle could be stimulated by high concentration of LA. This is not confirmed yet for glucose, but Issekutz, et al. (15,16) have shown the concentration of FFA in blood can be decreased by LA infusion in exercising dogs. In addition to the possible effects of catecholamines on

blood GLUC concentration during exercise, increased glucagon secretion could have enhanced hepatic glycogenolysis and gluconeogenesis during IE (7,10).

Conflicting reports concerning the role of catecholamines on growth hormone release may be attributable to species differences. Whereas Meyer and Knobil (19) and Muller, et al. (20) have shown a stimulatory effect of elevated catecholamines on growth hormone in monkeys and rats, several studies on humans indicate an inhibitory response of elevated catecholamine concentration on hGH release (2,24,25). In humans an elevated insulin concentration results in elevated plasma concentration of hGH (21). However, using infusions of insulin combined with epinephrine, Muller-Hess, et al. (21) have shown that exogenous epinephrine inhibits the effects of insulin on hGH release at rest. Muller-Hess, et al., therefore, suggested that elevated epinephrine levels inhibit the insulin induced hGH release. If catecholamines were elevated during IE in our study, and if their effects were to diminish the hGH response, our failure to observe a greater hGH response during IE may be explained.

In conclusion, our results suggest that hGH response to exercise is not correlated with the accumulation of an "anaerobic" metabolite. This interpretation is in agreement with the most recent findings of Sutton, et al. (29). Our results do not agree with those of Lassare, et al. (17) who related the rise of hGH during exercise with  $O_2$  deficit. It appears that further research into the control of hGH response in exercise is required.

**Acknowledgements:**

Research supported in part by a DHEW Biomedical Sciences Support Grant (RRO7006-10), in part by DHEW Arthritis, Metabolism and Digestive Diseases Support Grant (1 R01 AM195 77-01), in part by the Division of Biomedical and Environmental Research of the United States Energy Research and Development Administration, and in part by a University of California Chancellor's Patent Fund Grant. The generous support of James A. Bassham and Mina J. Bissell of the Laboratory of Chemical Biodynamics, LBL, U.C., Berkeley is acknowledged.

**Present address of Athanase Karagiorgos:**

Physical Therapy and Rehabilitation Clinic  
665 Beacon Street, Suite 304  
Boston, MA 02215.

REFERENCES

1. BEAVER, W.L. Water vapor corrections in oxygen consumption calculations. J. Appl. Physiol. 35:928-931, 1973.
2. BLACKARD, W.G., AND G.J. HUBELL. Stimulatory effect of exogenous catecholamines on plasma hGH concentrations in presence of beta-adrenergic blockade. Metabolism 19:547-552, 1970.
3. BUCKLER, J.M.H. Exercise as a screening test for GH release. Acta Endocrinol. 69:219-229, 1972.
4. CARLSON, A.L. Lipid metabolism and muscular work. Fed. Proc. 26:1755-1759, 1967.
5. DIETERLE, P., P. BANHOLZER, R. DIETERLE, J. HENNER, AND K. SCHWARZ. The influence of lactate on muscle metabolism: lactate as a possible regulating factor for the increased energy supply during muscular work. Horm. Metab. Res. 3:340-344. 1971.
6. FALHOLT K., B. LUND AND W. FALHOT. An easy colorimetric micromethod for routine determination of free fatty acids in plasma. Clin. Chim. Acta 46:105-111, 1973.
7. FELIG, P., J. WAHREN, R. HENDLER AND G. AHLBORG. Plasma glucagon levels in exercising man. N. Engl. J. Med. 287:184-185, 1972.
8. GARCIA, J.F., J.A. LINFOOT, E. MANOUGIAN, J.L. BORN AND J.H. LAWRENCE. Plasma growth hormone studies in normal individuals and acromegalic patients. J. Clin. Endocrinol. Metab. 27:139-1402, 1967.

9. GOLLNICK, P.D. Exercise, adrenergic blockage, and free fatty acid mobilization. Am. J. Physiol. 213:734-738, 1967.
10. HAGGENDAL, J., L.H. HARTLEY, AND B. SALTIN. Arterial noradrenaline concentration during exercise in relation to the relative work levels. Scand. J. Clin. Lab. Invest. 26:337-342, 1970.
11. HANSEN, P. AA. The effect of intravenous infusion of lipids on the exercise-induced serum growth hormone rise in normal and juvenile diabetics. Scand. J. Clin. Lab. Invest. 28:207-212, 1971.
12. HARTLEY, L.H., J.W. MASON, R.P. HOGAN, L.G. JONES, T.A. KOTCHEN, E.H. MOUGEY, F.E. WHERRY, L.L. PENNINGTON, AND P.T. RICKETTS. Multiple hormonal responses to graded exercise in relation to physical training. J. Appl. Physiol. 33:602-606, 1972, (a).
13. HARTLEY, L.H., J.W. MASON, R.P. HOGAN, L.G. JONES, T.A. KOTCHEN, E.H. MOUGEY, F.E. WHERRY, L.L. PENNINGTON, AND P.T. RICKETTS. Multiple hormonal responses to prolonged exercise in relation to physical training. J. Appl. Physiol. 33:607-610, 1972, (b).
14. HARTOG, M., R.J. HAVEL, G. COPINSCHI, J.M. BARLL, AND B.C. RITCHIE. The relationship between changes in serum levels of growth hormone and mobilization of fat during exercise in man. Q. J. Exp. Physiol. 52:86-96, 1967.
15. ISSEKUTZ, B.Jr., W.A.S. SHAW, AND A.C. ISSEKUTZ. Lactate metabolism in resting and exercising dogs. J. Appl. Physiol. 40:312-319, 1976, (a).
16. ISSEKUTZ, B.Jr., W.A.S. SHAW, AND T.B. ISSEKUTZ. Effect of lactate on FFA and glycerol turnover in resting and exercising dogs. J. Appl. Physiol. 39:349-353, 1975.



17. LASSARE, C., F. GIRARD, J. DURAND, AND J. RAYNAUD. Kinetics of human growth hormone during submaximal exercise. J. Appl. Physiol. 37:826-830, 1974.
18. LIHMAN, R.L., A.L. TAYLER, F.A. SCHANK et al. Inhibition of sleep related GH release by elevated FFA. J. Clin. Endocrinol. Metab. 35:592-594, 1972.
19. MEYER, V., AND E. KNOBIL. Growth hormone secretion in the unanesthetized rhesus monkey in response to noxious stimuli. Endocrinology 80:163-171, 1967.
20. MULLER, E.E., T. SAITO, A. ARIMURA, AND A.V. SCHALLY. Hypoglycemia, stress and growth hormone release: blockage of growth hormone release by drugs acting on the central nervous system. Endocrinology 80:109-117, 1976.
21. MULLER-HESS, R., C.A. GESER, E. JEQUIER, J.P. FELBER AND V. VANNOTI. Effects of adrenaline on insulin-induced release of growth hormone and cortisol in man. Acta Endocrinol. 75:260-273, 1974.
22. PFLEIDERER, G. L-Alanine determination with glutamate-pyruvate transaminase and lactic dehydrogenase. In, Methods Of Enzymatic Analysis, H. Bergmeyer, ed., Academic Press, New York, 1965, pp. 253-259.
23. PRUETT, E.D.R. Plasma insulin concentration during prolonged work at near maximal oxygen uptake. J. Appl. Physiol. 29:155-158, 1970.
24. QUABBE, H-J., H-J. BRATZKE, U. SIEGERS, AND K. ELBAN. Studies on the relationship between plasma FFA and growth hormone secretion in man. J. Clin. Invest. 51:2388-2397, 1972.
25. RABINOWITZ, D., T.J. MERIMEE, J.A. BURGESS AND L. RIGGS. Growth hormone and insulin release after arginine: indifference to hyperglycemia and

- and epinephrine. J. Clin. Endocrinol. Metab. 26:1170-1179, 1966.
26. SANDERS, C.A., G.E. LEVINSON, W.H. ABLEMAN, AND N. FREINKEL.  
Effect of exercise on the peripheral utilization of glucose in man.  
N. Engl. J. Med. 271:220-225, 1964.
27. SCHWARZ, F., D.J. TER HAAR, H.G. VAN RIET AND J.H.H. THIJSSEN.  
Response of growth hormone, FFA, blood sugar and insulin to exercise in  
obese patients and normal subjects. Metabolism. 19:1013-1020, 1969.
28. SUTTON, J.R., J.D. YOUNG, L. LAZARUS, J.B. HICKIE, AND J. MAKSVYTIS.  
The hormonal response to physical exercise. Australas. Ann. Med.  
18:84-90, 1969 (a).
29. SUTTON, J.R., N.L. JONES, AND C.J. TOEWS. Growth hormone secretion in  
acid-base alteration at rest and during exercise. Clin. Sci. Mol. Med.  
50:241-247, 1976, (b).
30. WINTER, J.S.D. The metabolic response to exercise and exhaustion in  
normal and growth-hormone-deficient children. Can. J. Physiol. Pharmacol.  
52:575-582, 1974.
31. YALOW, R.S., AND S. BERSON. Immunoassay of endogenous plasma insulin in  
man. J. Clin. Invest. 39:1157-1159, 1960.

Table 1. Descriptive anthropometric and metabolic data on subjects.

Subject	Age (years)	Weight (kg)	$\dot{V}O_2$ max (L $\cdot$ min $^{-1}$ )	$\dot{V}O_2$ max (ml $\cdot$ min $^{-1}$ )	Actual $\dot{V}O_2^*$ (L $\cdot$ min $^{-1}$ )	O $_2$ Deficit** (L.)	Actual* work load (KPM .min $^{-1}$ )
1. GB	30	80.7	3.70	45.70	1.866	.863	800
2. TW	26	93.0	4.38	46.26	2.305	.990	1000
3. GG	25	81.5	4.70	57.95	2.238	1.244	1100
4. MH	29	84.0	3.75	43.10	1.913	1.581	800
5. SH	25	79.8	3.80	48.40	2.052	1.565	800
6. CD	23	92.6	3.56	39.06	2.001	1.763	800
7. RR	28	66.1	3.60	54.50	1.791	1.104	800
8. OC	23	88.6	3.55	40.90	1.960	1.185	700
9. ND	21	73.2	3.75	51.20	1.934	1.213	800
10. SS	22	66.8	3.65	54.00	1.808	1.066	800
Mean	25.2	80.64	3.84	48.11	1.990	1.257	840
SEM	.9	3.00	.12	1.97	.054	.045	37.1

\* This work rate was for continuous exercise only. Work rates during intermittent exercise bouts were 2x these values.

\*\* O $_2$  deficit calculated for CE only.

Legends To Figures

- Figure 1: Plasma growth hormone concentration ( $\bar{X} \pm \text{SEM}$ ) during rest, exercise and recovery.
- Figure 2: Blood lactate concentration ( $\bar{X} \pm \text{SEM}$ ) as a function of time during rest, exercise and recovery.
- Figure 3: Blood pyruvate concentration ( $\bar{X} \pm \text{SEM}$ ) during rest, exercise and recovery.
- Figure 4: Blood glucose concentration ( $\bar{X} \pm \text{SEM}$ ) during rest, exercise and recovery.
- Figure 5: Blood alanine concentration ( $\bar{X} \pm \text{SEM}$ ) during rest, exercise and recovery.
- Figure 6: Plasma free fatty acid concentrations ( $\bar{X} \pm \text{SEM}$ ) during rest, exercise and recovery.
- Figure 7: Rectal temperature ( $\bar{X} \pm \text{SEM}$ ) during rest, exercise and recovery.

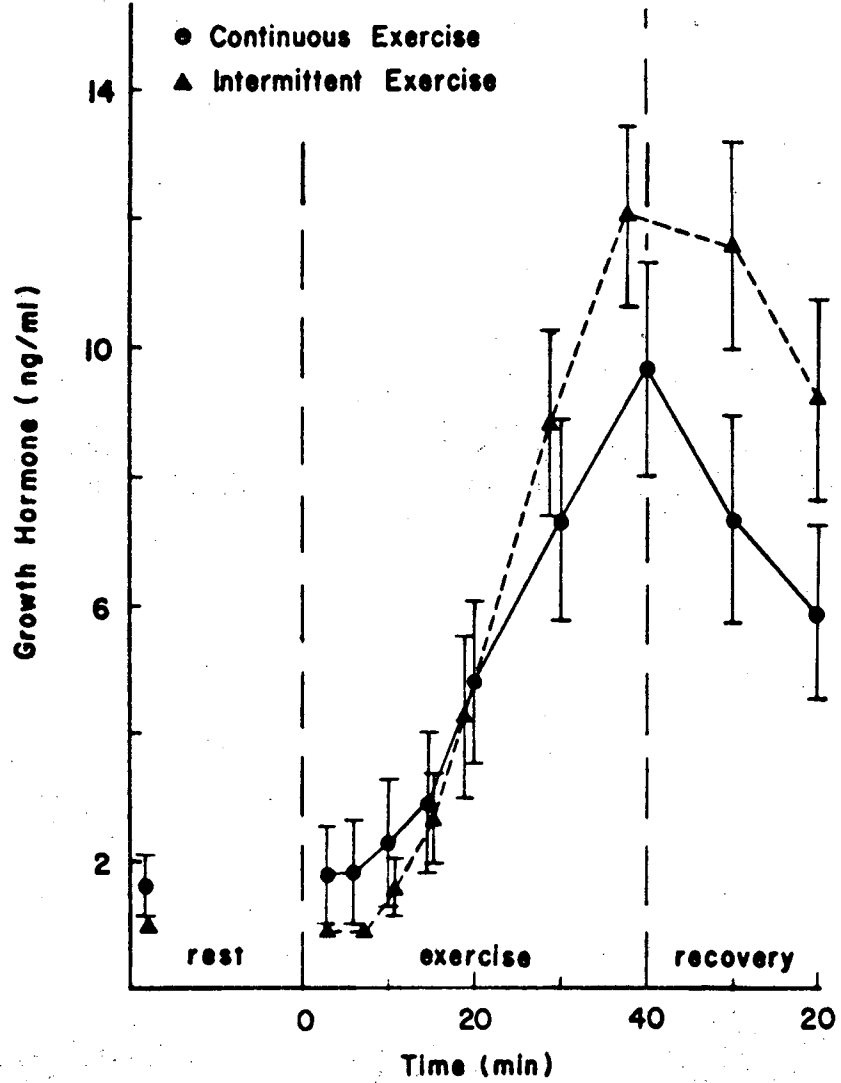


Fig. 1

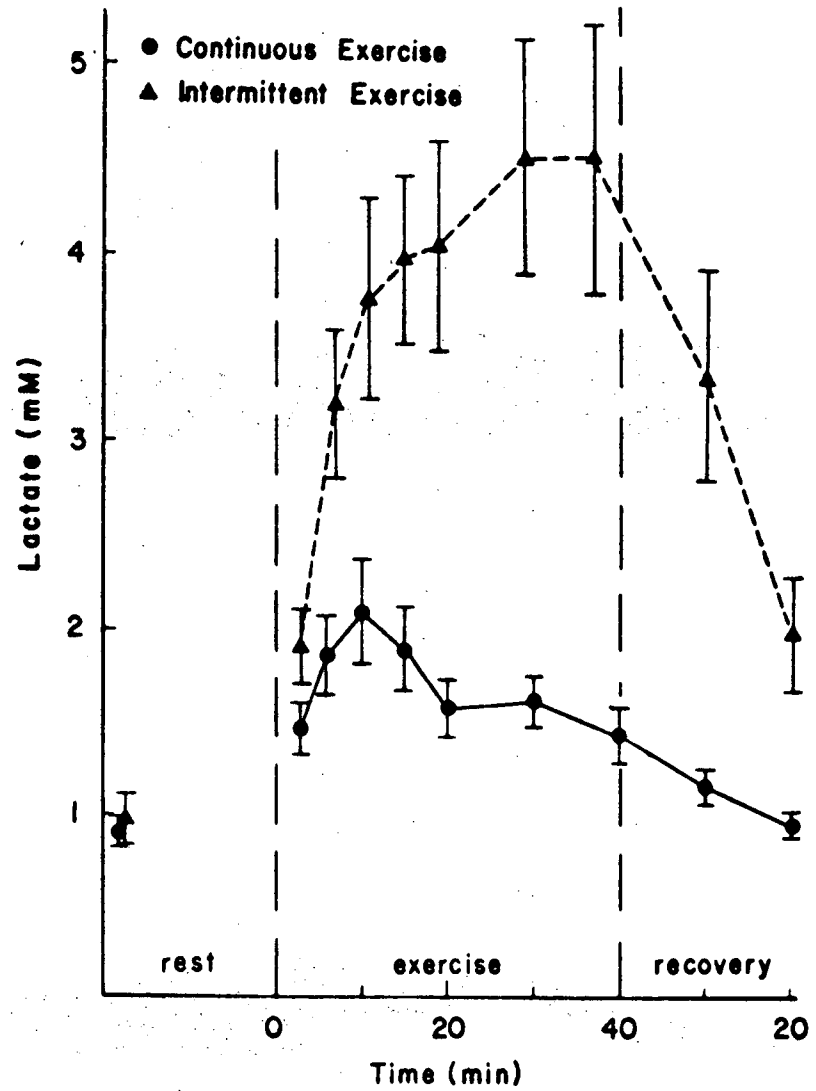


Fig. 2

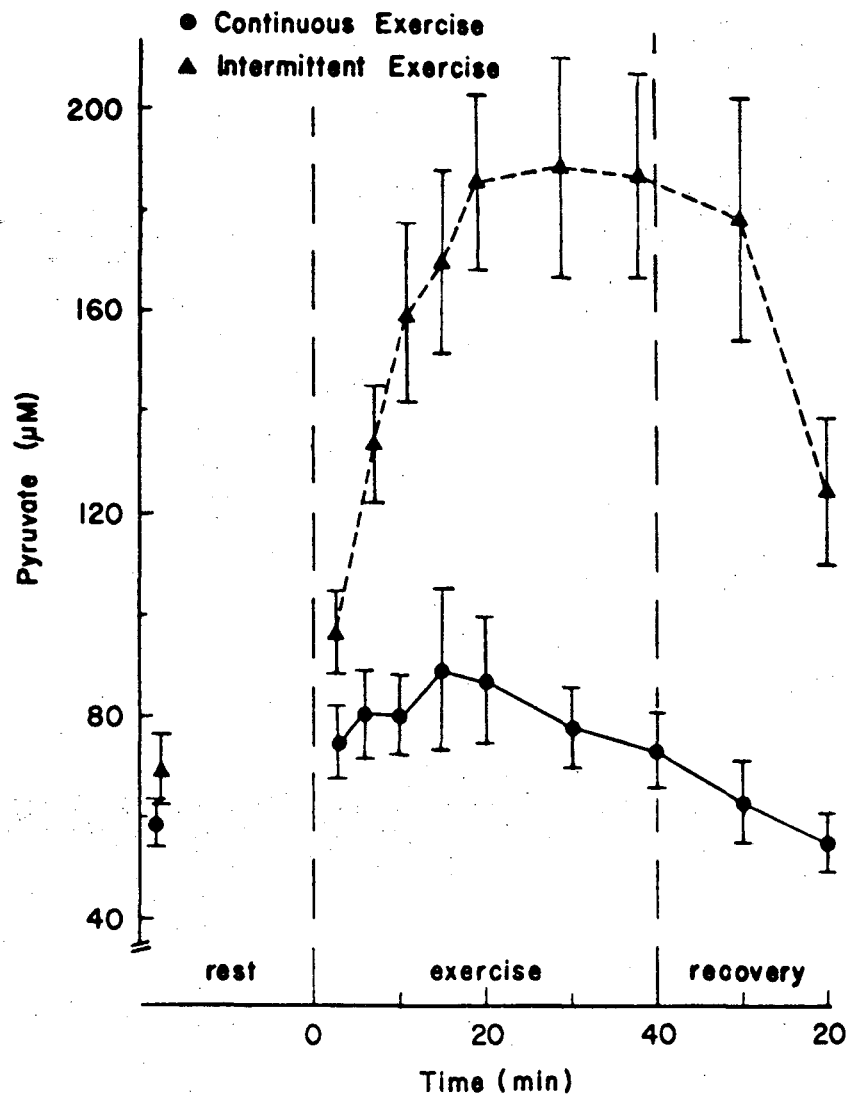


Fig. 3

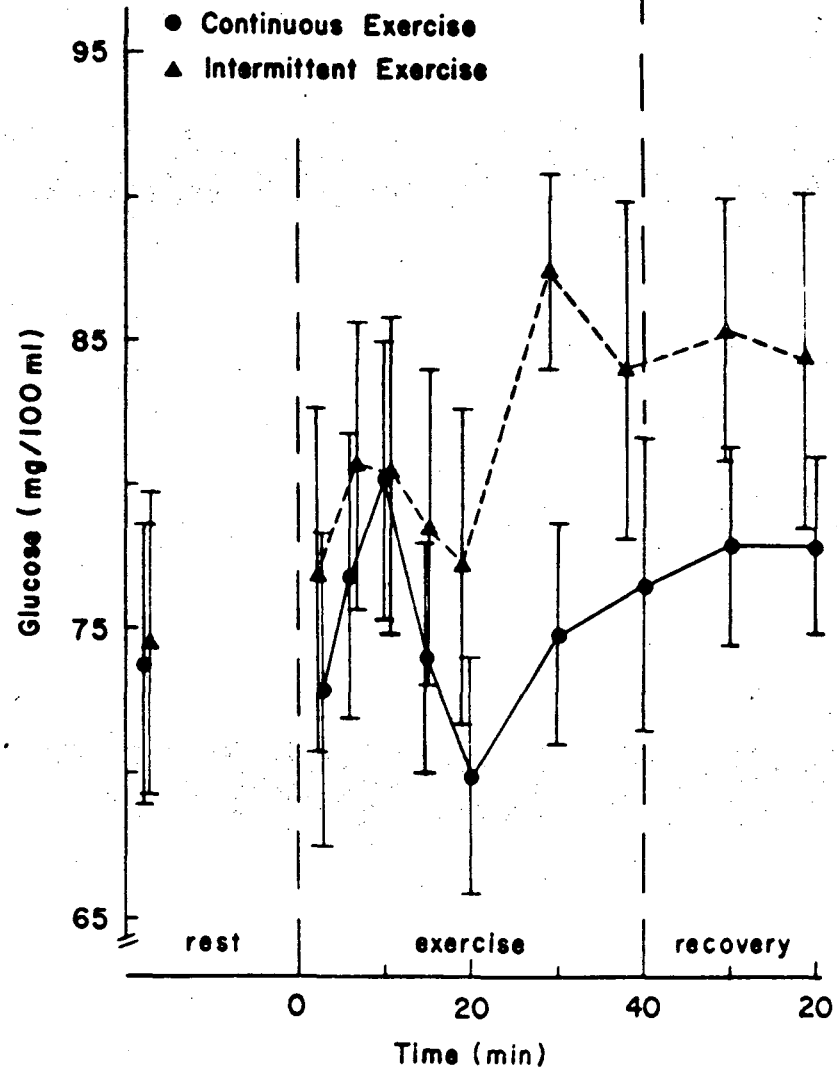


Fig. 4

00004803011

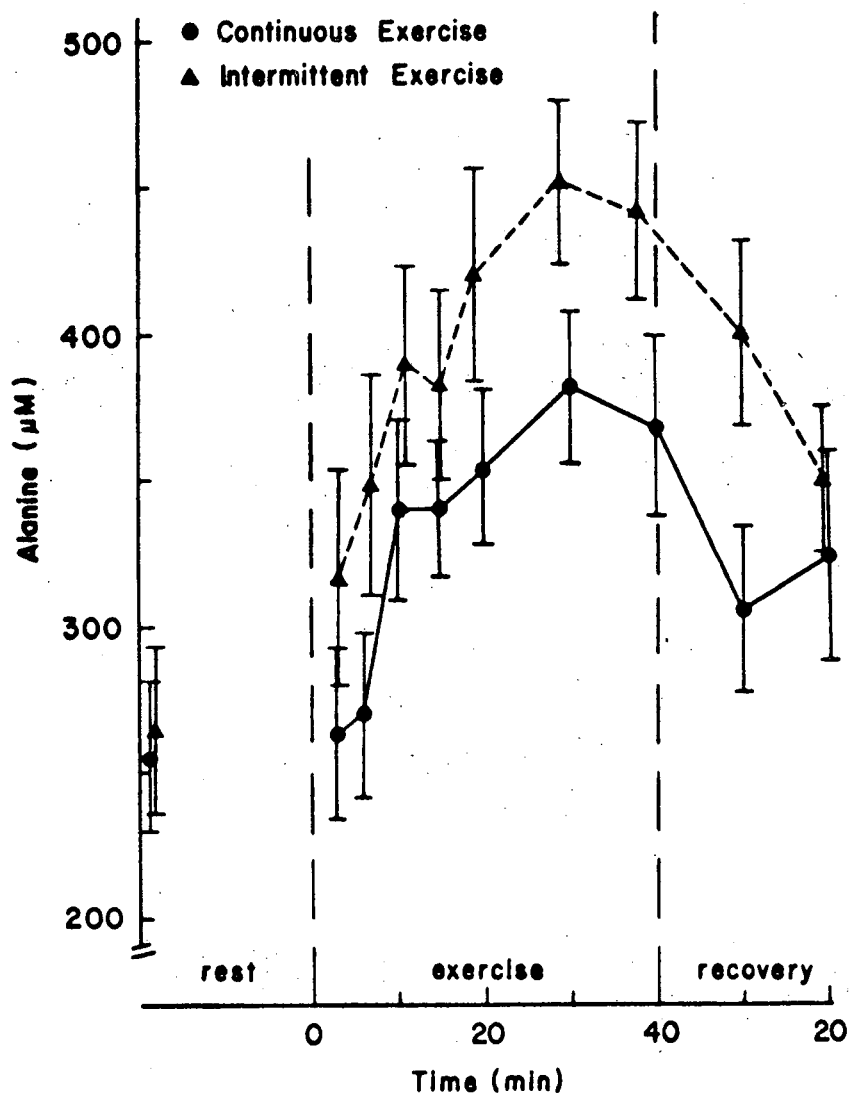


Fig. 5

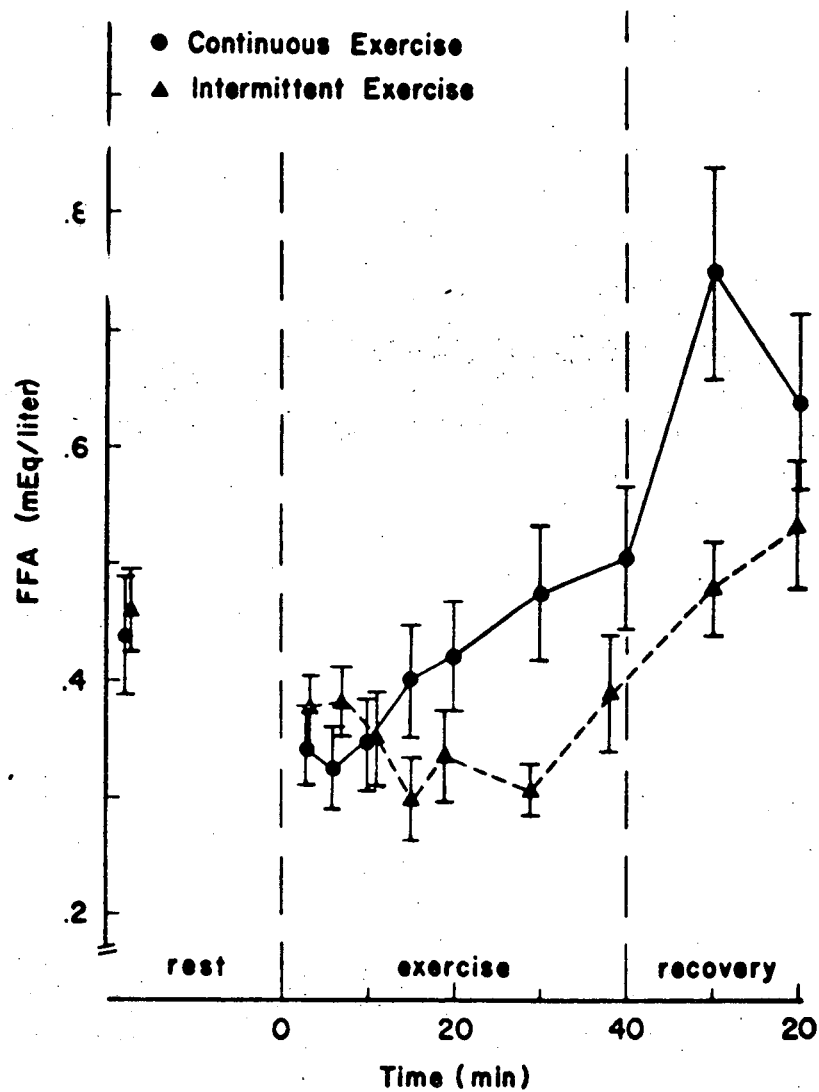


Fig. 6

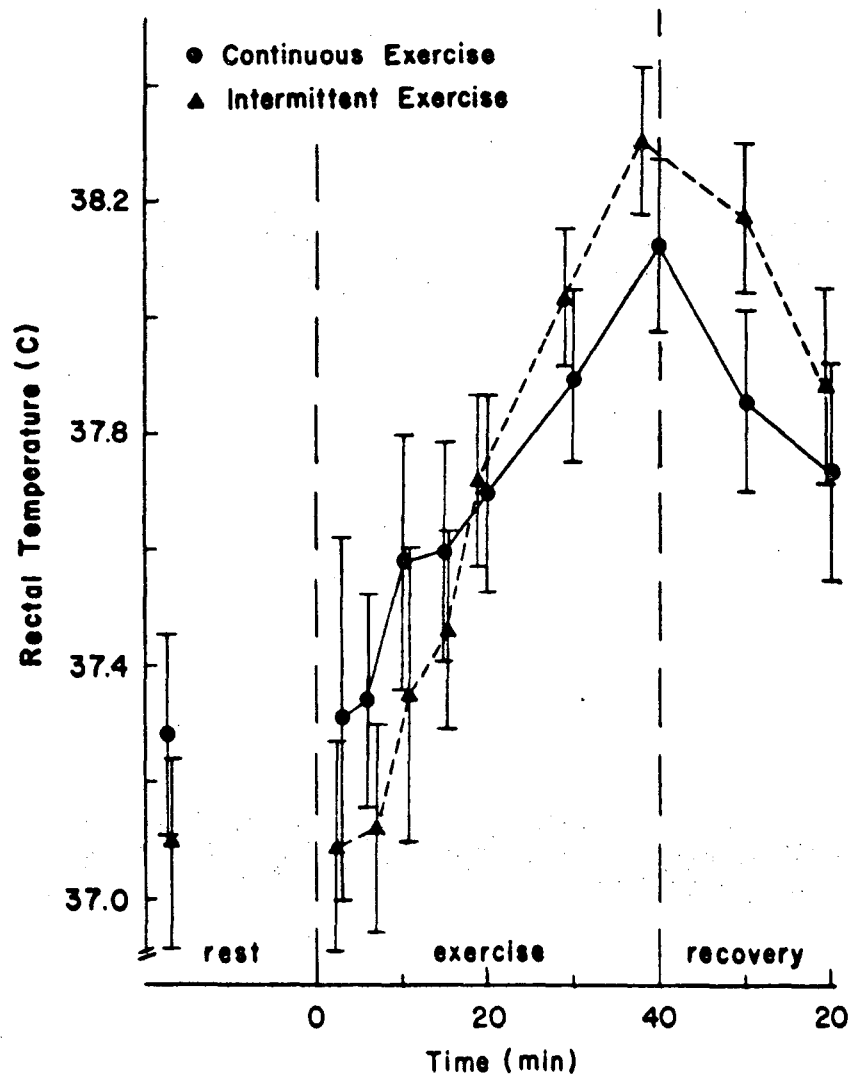


Fig. 7



This report was done with support from the United States Energy Research and Development Administration. Any conclusions or opinions expressed in this report represent solely those of the author(s) and not necessarily those of The Regents of the University of California, the Lawrence Berkeley Laboratory or the United States Energy Research and Development Administration.

TECHNICAL INFORMATION DIVISION  
LAWRENCE BERKELEY LABORATORY  
UNIVERSITY OF CALIFORNIA  
BERKELEY, CALIFORNIA 94720