Hormonal Responses to Maximal and Submaximal Exercise in Trained and Untrained Men of Various Ages

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Neuroendocrine adjustments to maximal and submaximal exercise were investigated in men as a function of age and training status. Twenty-four trained cyclists and 23 sedentary men constituting a young (M = 22.9 yrs, n = 16), middle-aged (M = 44.9 yrs, n = 16), and old (M = 65.5 yrs, n = 15) group performed both a maximal (GXT) and a 45-minute submaximal exercise test (cycle ergometer) at the workload corresponding to their lactate threshold. Plasma lactate, glucose, growth hormone, cortisol, norepinephrine, and epinephrine concentrations were analyzed both at rest and during exercise. Peak oxygen consumption (\(V_{O2}\) peak) was lower with age; however, all trained groups had higher values for \(V_{O2}\) peak compared to sedentary groups, regardless of age. Lactate threshold, when expressed in absolute terms, was lower with advancing age (sedentary \(9 \text{ yrs} \times 26\%\); trained \(19 \text{ yrs} \times 35\%\) for middle-age and old, respectively, when compared to young). Pre-exercise plasma norepinephrine levels were higher with age in both trained and sedentary subjects. Cortisol levels were lower with age for sedentary subjects and were significantly elevated in trained subjects across all age groups. Endurance training resulted in increased hormonal responses, as measured by plasma concentrations, to both maximal and submaximal exercise across all age groups. However, regardless of training status, age-related declines were observed in peak responses for lactate, growth hormone, and cortisol during the GXT. During the 45-minute submaximal exercise test, these age-related differences that had been present in the GXT were abolished. This submaximal test represented a lower absolute work load for old compared to young as well as sedentary compared to trained subjects; however, individuals were working at similar relative exercise intensities. We conclude that older individuals are capable of similar hormonal responses to submaximal exercise of identical durations and intensities as their young and middle-aged counterparts, and that chronic endurance training can enhance the hormonal response to exercise in all age groups.

Aging has been associated with numerous declines in neuroendocrine function, which results in a reduced capacity to respond to disruptions in homeostasis (for review see Sonntag, 1987; Lakatta, 1993). Endurance training in young individuals has been shown to increase neuroendocrine responsiveness and the ability to adapt to stress (Hagberg et al., 1985, 1988; Kjaer et al., 1985; Lehmann and Keul, 1986). However, little is known regarding the ability of older individuals to respond to acute stressors and the extent to which exercise training can improve neuroendocrine responsiveness in elderly populations.

Acute exercise is a form of physiological stress that requires hormonal and metabolic changes in order to adapt to disruptions in homeostasis. Previous research has shown declines in cortisol (Friedman et al., 1969; Pavlov et al., 1986), growth hormone (Finkelstein et al., 1972; Ho et al., 1987; Vermeulen, 1987; Hagberg et al., 1988) and catecholamine (Ziegler et al., 1976; Fleg et al., 1985; Lehmann and Keul, 1986; Hagberg et al., 1988) regulation both at rest and during exercise with advancing age. A decline in target organ responsiveness, as well as lower plasma levels of key hormones during exercise, have been reported in older sedentary men compared to their younger counterparts (Shepherd and Sidney, 1975; Fleg et al., 1985; Lehmann and Keul, 1986; Hagberg et al., 1988). However, it has been shown that trained older distance runners are capable of similar hormonal and metabolic responses to trained men 40 years younger in response to 1 hour of exercise at 70% of \(V_{O2}\) max (Hagberg et al., 1988). When comparing groups across different ages and training status, it is important to compare them at a similar metabolic stress, as the exercise intensity is the primary stimulus for hormone activity. Therefore, the subjects in the present study were compared both during a progressive exercise test to exhaustion (\(V_{O2}\) peak) as well as during a submaximal exercise test at the workload corresponding to each individual’s lactate threshold.

The primary objective of this investigation was to examine the influence of age and training on the responsiveness of key hormones responsible for regulating important metabolic and physiologic adjustments required during acute exercise. It was hypothesized that chronic endurance training would positively influence hormonal responses to maximal and submaximal exercise so as to provide the best maintenance of homeostasis and the most responsive adjustments from the stress of exercise.

METHODS

Subjects. — Forty-seven healthy male volunteer subjects read and signed an informed consent approved by the Human Subjects Committee of the University of Colorado–Boulder.
Subjects consisted of young (sedentary = 22.9 ± 1.0 yrs; trained = 22.6 ± 0.8 yrs), middle-aged (sedentary = 43.6 ± 1.1 yrs; trained = 46.5 ± 0.9 yrs), and old (sedentary = 67.0 ± 2.2 yrs; trained = 63.9 ± 1.8 yrs) individuals. Subjects were either chronic endurance-trained cyclists or sedentary individuals who did not participate in any endurance or resistance training. The trained subjects had been engaged in cycling training for a period of = 20, 10, and 5 yrs for the old, middle-aged, and young groups, respectively.

\( \hat{V}O_2 \) peak protocol. — All subjects reported to the Human Performance Laboratory on the University of Colorado–Boulder campus the morning following an overnight fast. All subjects had refrained from strenuous activity 24 hours prior to testing. Resting supine systolic and diastolic blood pressure was obtained upon entering the laboratory. Body fat percentage was estimated by the 6-site skinfold method (scapularis, triceps, iliac crest, abdomen, chest, and thigh) in all subjects (Golding et al., 1982). Middle-aged and old sedentary as well as old trained subjects were prepared with a standard 12-lead electrocardiogram, and all young as well as middle-aged trained subjects were prepared with a modified \( V < 5 \)-lead electrocardiogram prior to participation in a progressive \( \hat{V}O_2 \) peak test on a Monarch bicycle ergometer. A cardiologist presided over all standard 12-lead exercise tests. Any men with clinical evidence of ischemic heart disease were excluded from the study.

An indwelling catheter was placed in an antecubital forearm vein for serial blood sampling. Following a period of supine rest, after which a pre-exercise blood sample (7 ml) was collected, all subjects began pedaling a Monarch bicycle ergometer at a frequency of 70 rpm at 420 kgm/min. Every 2 minutes the workload was increased by 210 kgm/min until a predetermined heart rate dependent on the subjects’ age-group was reached (young = 160 bpm; middle-aged = 140 bpm; old = 120 bpm). After this point the workload was increased by 105 kgm/min every 2 minutes until subjects had reached maximal exertion. An open circuit indirect calorimetry system was used to collect and analyze expired gases every 30 seconds during exercise. Known gases were used to provide a 3-point calibration regression for oxygen and carbon dioxide fractions, determined by an Applied Electrochemistry S-3A Oxygen Analyzer and a Beckman LB-2 Carbon Dioxide Analyzer, respectively. The ventilatory volumes were measured from inspired samples with a Hans Rudolph Pulmonary tachometer. Final analysis for each 30-second sampling period was determined by computer and provided the following calculations: \( \hat{V}O_2 \) (l/min), \( \hat{V}O_2 \) (ml/kg/min), \( \hat{V}CO_2 \) (l/min), VE (l/min), and respiratory exchange ratio (RER). Blood samples (7 ml) were obtained during the last 30 seconds of each 2-minute stage throughout exercise for measurement of plasma lactate, epinephrine, norepinephrine, growth hormone, and cortisol concentrations.

Submaximal protocol. — Subjects returned to the laboratory at least 72 hours later but not more than 7 days after completing the graded exercise test (GXT) for a 45-minute submaximal exercise bout at the workload corresponding to each individual’s lactate threshold (Tlac, determined from the GXT). Again, all testing was performed the morning after an overnight fast. Heart rate was monitored with a V-5 modified electrocardiogram, and \( \hat{V}O_2 \) was monitored by the on-line indirect calorimetry system as described above. Subjects were allowed a 5-minute warm-up prior to exercising at the determined workload for Tlac. From this point on subjects remained at the workload corresponding to the Tlac until the end of exercise. Blood samples (7.0 ml) were obtained before exercise and during the exercise bout at minute 5, 10, 15, 30, and 45 for analysis of plasma glucose, lactate, epinephrine, norepinephrine, growth hormone, and cortisol concentrations. Only one old sedentary subject was unable to complete the 45-minute submaximal exercise bout (stopped at minute 30).

Blood analyses. — Blood for glucose and lactate determinations (1 ml) was placed in pre-weighed chilled tubes containing 8% perchloric acid. Blood for growth hormone and cortisol determination (1 ml) was placed in chilled tubes containing heparin. The remaining blood for catecholamine determination (~5 ml) was placed in chilled tubes containing reduced glutathione (5 mM) and heparin. All tubes were centrifuged for 10 minutes at 1200 g in a temperature-controlled centrifuge. The plasma was separated from the supernatant and stored in individual freezer tubes at -70 °C until biochemical analyses.

Plasma glucose concentrations were determined by the enzymatic technique of Bergmeyer and Bernt (1974). Plasma lactate concentrations were determined by the enzymatic technique of Hohorst (1971). Plasma growth hormone and cortisol concentrations were determined by the double antibody technique with kits commercially available from Diagnostic Products Inc. (Los Angeles, CA). Plasma catecholamine concentrations for epinephrine and norepinephrine were determined by high-performance liquid chromatography (HPLC) with electrochemical detection (Mazzeo and Marshall, 1989). An internal standard was prepared by addition of appropriate levels of dihydroxybenzylamine (Sigma) in 50 µl of 0.1 N perchloric acid (PCA) to a 1.5 M tris(hydroxymethyl)aminomethane buffer at pH 8.6 in 2% EDTA. Acid washed alumina (35 mg; Woelm, ICN Pharmaceuticals, Irvine, CA) was added, followed by 10 minutes of vigorous shaking. The alumina was then washed twice with 3.0 ml distilled water with brief centrifugation between washes. The catecholamines were extracted with 100 µl of 0.1 N PCA with 10 minutes of shaking and final centrifugation at 12,000 g. One hundred microliters of sample eluant were injected into the HPLC column (reverse phase; Bio-Sil ODS-SS, Bio-Rad, Richmond, CA) and eluted with mobile phase (6.8 g sodium acetate-anhydrous, 1.0 g sodium heptane sulfonate, 60 ml acetonitrile, 1.0 g Na2EDTA in 1 liter adjusted to a pH of 4.8). The flow rate was 1.1 ml/min at 2,000 psi with a potential of 0.65 V. The chromatogram was analyzed by computer integration (model C-R3A, Shimadzu, Tokyo, Japan).

Statistics. — Values are means ± SE. Differences between groups across all conditions (pre-exercise, \( \hat{V}O_2 \) peak, and submaximal exercise test) were determined by a two-way ANOVA (Age × Training) with Tukey post hoc analysis. Significance was set at \( p < .05 \).
RESULTS

Subject characteristics. — Characteristics of subjects are reported in Table 1. Body fat percentage increased with advancing age and was lower in trained subjects across age groups. No differences existed in resting systolic blood pressure between all groups; however, all trained groups had lower resting diastolic blood pressures than their sedentary counterparts.

Cardiovascular profiles. — Maximal heart rate, obtained during the progressive cycle ergometer VO2 peak test, was lower with advancing age in both the trained and sedentary groups (Table 1). There were no differences in maximal heart rate between trained and sedentary groups of similar age, with the exception of the old subjects, where trained subjects had a greater maximal heart rate.

Peak oxygen consumption (VO2 peak), expressed as l/min or as ml/kg/min, was lower with advancing age in all groups and was higher in all trained compared to sedentary groups of similar age (Table 1).

Lactate threshold profiles. — Lactate threshold (Tlac) profiles determined from the GXT are presented in Table 2 (a minimum of 7 and a maximum of 16 data points were used for Tlac determinations). An aging effect existed for all groups such that the VO2 (l/min or ml/kg/min) eliciting Tlac was lower with advancing age. A training effect was also present, such that all trained groups, regardless of age, had a higher VO2 compared to their age-matched sedentary group. When expressed as % VO2 peak, a significant aging effect occurred within sedentary groups such that the middle-aged and old groups had a higher Tlac compared to the young group. The old trained group had a lower Tlac compared to the young and middle-aged trained groups. A training effect existed for young and middle-aged groups such that Tlac was higher in trained groups compared to their sedentary counterparts.

Blood lactate concentration at the Tlac was similar between all groups (~1.4 mM), and there was also no difference in pre-exercise plasma lactate concentrations across all groups.

Pre-exercise plasma hormone levels. — No differences in pre-exercise plasma glucose, lactate, epinephrine, or growth hormone concentrations were observed across age or training status (Table 3). Plasma norepinephrine levels were higher in the middle-aged and old sedentary groups compared to the younger group. The young and middle-aged trained groups had similar norepinephrine concentrations, whereas the old trained group had a higher pre-exercise concentration. A significant age and training interaction occurred in pre-exercise plasma cortisol concentrations. Within the sedentary subjects, both middle-aged and old groups had lower concentrations compared to the young group. A training effect existed such that all trained groups had higher pre-exercise plasma cortisol levels compared to their sedentary counterparts.

Responses to maximal exercise. — The maximal work load achieved during the GXT averaged 1453 ± 71, 1222 ±

Table 1. Subject Characteristics and Cardiovascular Profiles

<table>
<thead>
<tr>
<th></th>
<th>Young Sedentary</th>
<th>Young Trained</th>
<th>Middle-aged Sedentary</th>
<th>Middle-aged Trained</th>
<th>Old Sedentary</th>
<th>Old Trained</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>85.4 ± 5.3</td>
<td>71.7 ± 2.7†</td>
<td>82.4 ± 4.1</td>
<td>70.0 ± 3.5†</td>
<td>79.3 ± 3.7</td>
<td>73.5 ± 2.4</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>15.6 ± 2.3</td>
<td>11.4 ± 1.0†</td>
<td>21.0 ± 1.8*</td>
<td>15.7 ± 0.7†</td>
<td>23.4 ± 1.3</td>
<td>20.6 ± 1.9*</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>124 ± 4</td>
<td>119 ± 4</td>
<td>125 ± 3</td>
<td>120 ± 3</td>
<td>128 ± 5</td>
<td>119 ± 3</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>87 ± 4</td>
<td>75 ± 3†</td>
<td>88 ± 4</td>
<td>79 ± 3†</td>
<td>85 ± 3</td>
<td>78 ± 3†</td>
</tr>
<tr>
<td>HR max (bpm)</td>
<td>192 ± 2</td>
<td>196 ± 2</td>
<td>174 ± 4*</td>
<td>180 ± 3*</td>
<td>155 ± 3*</td>
<td>163 ± 4*</td>
</tr>
<tr>
<td>VO2 peak (l/min)</td>
<td>3.5 ± 0.2</td>
<td>4.5 ± 0.1†</td>
<td>3.0 ± 0.1*</td>
<td>3.9 ± 0.1†</td>
<td>2.2 ± 0.2*</td>
<td>3.2 ± 0.2*</td>
</tr>
<tr>
<td>VO2 peak (ml/kg/min)</td>
<td>42.1 ± 2.6</td>
<td>62.5 ± 1.2†</td>
<td>35.3 ± 2.5*</td>
<td>51.0 ± 1.7†</td>
<td>27.7 ± 1.6*</td>
<td>43.5 ± 2.2*</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE. For all Tables n = 8/group except for old sedentary group, where n = 7.
*Significantly different from young, p < .05.
**Significantly different from middle-aged, p < .05.
†Significant training effect, p < .05.

Table 2. Lactate Threshold Profiles

<table>
<thead>
<tr>
<th></th>
<th>Young Sedentary</th>
<th>Young Trained</th>
<th>Middle-aged Sedentary</th>
<th>Middle-aged Trained</th>
<th>Old Sedentary</th>
<th>Old Trained</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tlac (% VO2 peak)</td>
<td>63.1 ± 1.7</td>
<td>75.8 ± 2.7†</td>
<td>68.5 ± 1.6*</td>
<td>74.5 ± 2.7†</td>
<td>69.3 ± 1.9*</td>
<td>69.7 ± 1.2</td>
</tr>
<tr>
<td>VO2 at Tlac (l/min)</td>
<td>2.1 ± 0.1</td>
<td>3.4 ± 0.1†</td>
<td>2.0 ± 0.1*</td>
<td>2.9 ± 0.1†</td>
<td>1.5 ± 0.1*</td>
<td>2.2 ± 0.1*</td>
</tr>
<tr>
<td>VO2 at Tlac (ml/kg/min)</td>
<td>26.2 ± 2.0</td>
<td>47.2 ± 1.0†</td>
<td>23.9 ± 1.3</td>
<td>38.3 ± 1.8†</td>
<td>19.5 ± 1.2*</td>
<td>30.5 ± 2.0*</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE.
*Significantly different from young, p < .05.
**Significantly different from middle-aged, p < .05.
†Significant training effect, p < .05.
Cortisol concentrations compared to their young counterparts. A higher lactate concentration was reached in both the young and middle-aged groups. All trained groups had higher mean norepinephrine and epinephrine concentrations compared to their sedentary counterparts.

An aging effect occurred for maximal cortisol concentrations such that middle-aged and old groups had lower maximal cortisol concentrations compared to their young counterparts (Table 4). A training effect occurred in the young and middle-aged groups.

Responses to submaximal exercise. — The work load employed during the 45-min submaximal exercise was 1000 ± 63, 865 ± 34, and 659 ± 38 kgm/min for sedentary; 1495 ± 67, 1273 ± 50, and 1012 ± 76 kgm/min for trained young, middle-aged, and old subjects, respectively. Mean growth hormone and cortisol concentrations during the 45-minute submaximal exercise test did not differ across age groups or training status (sedentary = 3.7 ± 0.1, 4.1 ± 0.2, and 3.8 ± 0.1 mM for young, middle-aged, and old, respectively; trained = 3.8 ± 0.2, 3.6 ± 0.2, and 4.1 ± 0.2 mM for young, middle-aged, and old, respectively).

Mean lactate concentrations during the 45-minute submaximal exercise test are presented in Figure 1. An aging effect occurred in the old sedentary group only, such that the old group had a higher mean lactate concentration compared to the young and middle-aged groups. All trained groups had lower mean lactate concentrations compared to their sedentary counterparts.

Mean norepinephrine and epinephrine concentrations during the 45-minute submaximal exercise test are presented in Figure 2A and B, respectively. An aging effect occurred in the sedentary groups, such that the middle-aged and old groups had higher mean norepinephrine and epinephrine concentrations compared to the young group. All trained groups had higher mean norepinephrine and epinephrine concentrations compared to their sedentary counterparts.

Mean growth hormone and cortisol concentrations during the 45-minute submaximal exercise test are presented in Figure 3A and B, respectively. Unlike the results from the GXT, no aging effect occurred for mean growth hormone concentrations; however, a training effect existed in all age groups such that all trained subjects had higher mean growth hormone concentrations compared to their sedentary counterparts. For mean cortisol concentration, an age and training interaction existed. The middle-aged and old trained groups had lower cortisol concentrations compared to their

### Table 3. Pre-Exercise Plasma Hormone Concentrations

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Young Sedentary Mean ± SE</th>
<th>Young Trained Mean ± SE</th>
<th>Middle-aged Sedentary Mean ± SE</th>
<th>Middle-aged Trained Mean ± SE</th>
<th>Old Sedentary Mean ± SE</th>
<th>Old Trained Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine (ng/ml)</td>
<td>0.04 ± 0.01</td>
<td>0.06 ± 0.01</td>
<td>0.06 ± 0.01</td>
<td>0.06 ± 0.01</td>
<td>0.07 ± 0.02</td>
<td>0.10 ± 0.03</td>
</tr>
<tr>
<td>Norepinephrine (ng/ml)</td>
<td>0.4 ± 0.1</td>
<td>0.5 ± 0.1</td>
<td>0.8 ± 0.1*</td>
<td>0.6 ± 0.1</td>
<td>1.0 ± 0.1,**</td>
<td>1.0 ± 0.2,**</td>
</tr>
<tr>
<td>Growth hormone (ng/ml)</td>
<td>1.0 ± 0.1</td>
<td>1.3 ± 0.2</td>
<td>1.2 ± 0.1</td>
<td>1.4 ± 0.1</td>
<td>1.4 ± 0.3</td>
<td>1.2 ± 0.1</td>
</tr>
<tr>
<td>Cortisol (µg/dl)</td>
<td>16.6 ± 2.0</td>
<td>22.8 ± 1.3</td>
<td>12.1 ± 0.7*</td>
<td>16.1 ± 1.1†</td>
<td>13.7 ± 1.7*</td>
<td>17.1 ± 2.4†</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE.
*Significantly different from young, p < .05.
**Significantly different from middle-aged, p < .05.
†Significant training effect, p < .05.

### Table 4. Maximal Plasma Hormonal Concentrations: Graded Exercise Test

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Young Sedentary Mean ± SE</th>
<th>Young Trained Mean ± SE</th>
<th>Middle-aged Sedentary Mean ± SE</th>
<th>Middle-aged Trained Mean ± SE</th>
<th>Old Sedentary Mean ± SE</th>
<th>Old Trained Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate (mM)</td>
<td>4.8 ± 0.3</td>
<td>6.0 ± 0.4†</td>
<td>3.9 ± 0.5</td>
<td>5.2 ± 0.4†</td>
<td>3.7 ± 0.4*</td>
<td>3.8 ± 0.6*,**</td>
</tr>
<tr>
<td>Norepinephrine (ng/ml)</td>
<td>2.9 ± 0.4</td>
<td>7.5 ± 1.3†</td>
<td>3.1 ± 0.3</td>
<td>8.6 ± 2.5†</td>
<td>4.1 ± 1.0</td>
<td>6.0 ± 1.1†</td>
</tr>
<tr>
<td>Epinephrine (ng/ml)</td>
<td>0.4 ± 0.04</td>
<td>1.2 ± 0.30†</td>
<td>0.4 ± 0.12</td>
<td>1.3 ± 0.50†</td>
<td>0.3 ± 0.05</td>
<td>0.7 ± 0.28†</td>
</tr>
<tr>
<td>Growth hormone (ng/ml)</td>
<td>5.6 ± 1.8</td>
<td>21.9 ± 4.0†</td>
<td>8.6 ± 3.2</td>
<td>19.0 ± 4.4†</td>
<td>2.8 ± 1.9*,**</td>
<td>11.3 ± 3.5*,**,†</td>
</tr>
<tr>
<td>Cortisol (µg/dl)</td>
<td>19.8 ± 2.3</td>
<td>26.8 ± 2.2†</td>
<td>14.9 ± 0.9*</td>
<td>21.6 ± 1.8,†</td>
<td>16.3 ± 2.4*</td>
<td>19.5 ± 2.4*</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE.
*Significantly different from young, p < .05.
**Significantly different from middle-aged, p < .05.
†Significant training effect, p < .05.
young group; however, the old sedentary group had higher cortisol concentrations compared to the young and middle-aged groups. A training effect existed in the middle-aged and old groups such that the middle-aged trained group had a higher mean cortisol concentration compared to the sedentary group, while the old trained group had a lower mean cortisol concentration than the sedentary group.

DISCUSSION

The major finding of the present investigation was that endurance training resulted in increased hormonal responses, as determined by plasma concentrations, to both maximal and submaximal exercise across all age groups. Additionally, during 45 min of submaximal exercise at Tlac, old trained individuals were capable of similar responses in all hormones measured as their younger counterparts and significantly greater responses than the young and middle-aged sedentary groups. Similar findings were observed during the maximal exercise testing such that old trained individuals demonstrated greater hormonal responses than the younger sedentary groups. Thus, for a given metabolic stress as imposed in the present investigation, this would suggest that these neuroendocrine responses are enhanced with endurance training and, further, that training throughout life
may attenuate the well documented decline in neuroendocrine function (for review see Sonntag, 1987; Lakatta, 1993).

Maximal exercise. — As has been previously shown (Astrand et al., 1973; Hagberg et al., 1988), while $\dot{V}_O_2$ peak was significantly improved with endurance training across all age groups, a clear aging effect still remained regardless of training status (Table 1). It was generally observed that maximal lactate and hormone concentrations achieved during the GXT were lower for the old subjects when compared to the younger age groups (Table 4). This was particularly noticeable in the growth hormone and cortisol responses. Explanations for this observation may stem from a reduced capacity to synthesize and release these hormones from their respective glands or an enhanced capacity for hormone removal and clearance from plasma. It is doubtful that the latter mechanism is responsible, as we are unaware of any studies indicating that removal/clearance of these hormones is increased with age. However, a number of studies have reported a reduced capacity for growth hormone (Hagberg et al., 1988; Craig et al., 1989; Pyka et al., 1992) and cortisol (West et al., 1961; Serio et al., 1969; Romanoff and Baxter, 1975) secretion in the elderly both at rest and after a variety of stimuli including fasting and exercise. Twenty-four hour monitoring of the frequency and the amplitude of growth hormone pulses indicates a clear decline in secretory capacity with age (Finkelstein et al., 1972; Ho et al., 1987; Vermeulen, 1987). Further, with advancing age, variations in indices of growth hormone circadian rhythm patterns (Finkelstein et al., 1972; Prinz et al., 1983; Ho et al., 1987; Vermeulen, 1987), alterations in growth hormone messenger RNA and releasing hormone (Takahashi et al., 1990), and increases in somatostatin levels (Casad and Adelman, 1992; D’Costa et al., 1993) have all been reported.

A third possible explanation for lower growth hormone levels found in the elderly subjects relates to the total exercise time required to reach maximal work capacity by these subjects. The old sedentary subjects achieved the lowest peak hormone levels during the GXT; however, they also had the shortest exercise duration times required to reach peak $\dot{V}_O_2$ (10.3 ± 2.7 vs 16.1 ± 2.8 and 21.6 ± 3.1 min for middle-aged and young, respectively). As the hormonal response is dependent upon the exercise duration as well as the intensity, it is quite possible that the short exercise time observed for the old subjects is, in part, responsible for the lower hormonal responses demonstrated by this age group. This is particularly true for the growth hormone and cortisol responses observed, as these hormones are generally recognized as having a delayed/prolonged response time to a given exercise stimulus (Few, 1974; Shephard and Sidney, 1975; Bonen, 1976). As discussed below, when exercise duration was fixed and consistent across all groups, as during the 45 min of submaximal exercise, the aging effect witnessed during the GXT demonstrated for growth hormone and cortisol is abolished. Additionally, it is difficult to determine at this time whether the training effect observed across all age groups during the GXT is a product of enhanced capacity to synthesize and release these hormones or simply a function of the exercise duration as trained subjects exercised significantly longer than their age-matched sedentary controls.

Submaximal exercise. — In an attempt to control for the influence of the varied exercise durations during the GXT on hormonal responses with age and training, a second exercise test was performed at a fixed duration of 45 min. As hormonal responses to exercise are more dependent upon the level of metabolic stress imposed rather than an absolute work intensity, $Tlac$ was chosen as the submaximal work intensity in order to make similar comparisons between individuals across age and training status (Mazzeo and Marshall, 1989; Belman and Gaesser, 1991). Thus, this submaximal test represented a lower absolute work load for old compared to young as well as sedentary compared to trained subjects.

Similar to the results from the GXT, trained subjects across all age groups demonstrated significantly greater plasma hormone levels during the 45 min of submaximal exercise when compared to sedentary controls. Of equal importance is the finding that when the exercise duration was identical for all groups, the age-related decline in hormonal responses, as measured for the GXT, was eliminated. In response to the submaximal bout of exercise, plasma growth hormone concentration increased compared to resting values and achieved greater levels for all trained subjects than sedentary age-matched controls. However, for both trained and sedentary subjects, values did not decline with age, suggesting that for a given training status older individuals are capable of similar growth hormone responses to submaximal work if the exercise duration and intensity are equal. In general, similar results were observed for the other hormones measured such that across trained and sedentary groups no declines in plasma hormone levels to submaximal exercise were found with age. The notable exception existed in the sedentary subjects for both catecholamines measured. Both middle-aged and old sedentary subjects had significantly greater plasma epinephrine and norepinephrine levels during the 45 min submaximal exercise bout when compared to the young controls. This may be related to the observation that (1) the old subjects had elevated norepinephrine levels prior to the exercise session, and (2) there is a well-documented age-related decline in adrenergic receptor responsiveness (Sonntag, 1987; Lakatta, 1993) that can result in a compensatory increase in catecholamine release during periods of stress. Lehmann et al. (1986) has also reported greater catecholamine levels in older subjects for a given exercise intensity and concluded that, in addition to β-adrenergic receptor desensitization, age-dependent changes in fitness and rates of catecholamine clearance may account for differences observed with age. Fleg et al. (1985) also found that, in response to maximal and submaximal exercise at the same relative exercise intensity, older men demonstrated elevated plasma catecholamine levels.

Training resulted in significantly lower blood lactate levels during the 45 min submaximal exercise at $Tlac$, however, a consistent finding was that trained subjects achieved greater plasma hormone levels across all age groups. Endurance training in young populations has been shown to alter the pulsatile patterns for growth hormone release (Bunt et
physiological adaptations. Growth hormone administration to elderly as aging has been documented to result in a diminished capacity for growth hormone synthesis and release, this training adaptation could result in a number of beneficial physiological adaptations. Growth hormone administration in elderly subjects has been reported to increase lean muscle mass, decrease body fat, increase cardiac output, and improve maximal exercise performance (Rudman et al., 1990; Cuneo et al., 1991a, 1991b).

Training also augmented the plasma catecholamine response to submaximal exercise, and this was true for all age groups. The elevated levels found for the trained subjects compared to controls may be related to: (1) trained subjects were exercising at higher absolute and relative workloads, and (2) enhanced adrenal medulla responsiveness and secretory capacity with endurance training as demonstrated by Kjaer et al. (1985 and 1986) in young male subjects. Regardless of the mechanism(s) involved for this training effect it is apparent that, as with growth hormone, a lifetime of endurance training allows an individual to maintain a plasma catecholamine response to a given metabolic exercise stress that is similar to their younger trained counterparts and significantly greater than their age-matched controls.

Finally, a training effect was observed for the plasma cortisol response to submaximal exercise for the young and middle-aged groups only. Previous literature addressing the effect of endurance training on the cortisol response to acute exercise has been inconsistent. Some investigators have reported no change in plasma cortisol levels as a result of endurance training (Hartley et al., 1972; Kraemer et al., 1989), while others have observed elevated levels in trained subjects for a given work intensity (Severson et al., 1977). The reason why we did not observe a training effect in the old group, as was seen for both the young and middle-aged groups, is unknown and beyond the scope of the present study. Care was taken to control for variations in circadian rhythms known to occur for the hormones measured in the present study. Subjects were always tested the same time of day with a controlled period of fasting prior to experimentation. The lack of training effect witnessed for the old subjects may be related to a number of factors including: (1) the young and middle-aged trained subjects had significantly greater plasma cortisol levels prior to exercise; (2) alterations with age and/or training of the normal circadian rhythm patterns; and (3) other yet to be determined mechanisms involving the interaction of age and training.

In summary, the results of the present study indicate that a lifetime of endurance training can enhance the hormonal response to a similar bout of acute exercise in older individuals and allow them to maintain levels similar to their younger trained counterparts. Additionally, these old trained individuals are capable of significantly greater hormonal responses than young and middle-aged sedentary controls in response to the stress of a single bout of intense exercise. The mechanisms responsible for these findings as well as the physiological and clinical significance remain to be determined.

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