Testosterone and cortisol in relationship to dietary nutrients and resistance exercise

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Volek, Jeff S., William J. Kraemer, Jill A. Bush, Thomas Incledon, and Mark Boetes. Testosterone and cortisol in relationship to dietary nutrients and resistance exercise. J. Appl. Physiol. 82(1):49–54, 1997.—Manipulation of resistance exercise variables (i.e., intensity, volume, and rest periods) affects the endocrine response to exercise; however, the influence of dietary nutrients on basal and exercise-induced concentrations of hormones is less understood. The present study examined the relationship between dietary nutrients and resting exercise-induced blood concentrations of testosterone (T) and cortisol (C). Twelve men performed a bench press exercise protocol (5 sets to failure using a 10-repetitions maximum load) and a jump squat protocol (5 sets of 10 repetitions using 30% of each subject’s 1-repetition maximum squat) with 2 min of rest between all sets. A blood sample was obtained at preexercise and 5 min postexercise for determination of serum T and C. Subjects also completed detailed dietary food records for a total of 17 days. There was a significant (P ≤ 0.05) increase in postexercise T compared with preexercise values for both the bench press (7.4%) and jump squat (15.1%) protocols; however, C was not significantly different from preexercise concentrations. Significant correlations were observed between pre-exercise T and percent energy protein (r = −0.71), percent energy fat (r = 0.72), saturated fatty acids (g·1,000 kcal−1·day−1; r = 0.77), monounsaturated fatty acids (g·1,000 kcal−1·day−1; r = 0.79), the polyunsaturated fat-to-saturated fat ratio (r = −0.63), and the protein-to-carbohydrate ratio (r = −0.59). There were no significant correlations observed between any nutritional variables and preexercise C or the absolute increase in T and C after exercise. These data confirm that high-intensity resistance exercise results in elevated postexercise T concentrations. A more impressive finding was that dietary nutrients may be capable of moderating resting concentrations of T.

METHODS

Subjects. Twelve healthy men with at least 1 yr of resistance training experience volunteered to participate in this investigation. Descriptive data for the 12 subjects are presented in Table 1. The subjects had been involved with resistance training ~5 yr, and they trained, on average, five sessions per week. Their workouts involved multiple sets (15–25 per workout) and moderate repetitions (6–15 per set) comprising exercises for two to three muscle groups per session. None of the subjects were coming off any type of high-volume and/or high-intensity cycles, and their workouts were characterized by relatively consistent training volumes 6–10 wk before the study. All subjects were informed as to the possible risks of the investigation before giving their written informed consent in accordance with The Pennsylvania State University Institutional Review Board for use of human subjects.

Exercise protocol. All subjects completed an identical bench press exercise protocol and a jump squat exercise protocol (performed on consecutive days) on two occasions separated by 1 wk. Both testing protocols were performed on a Plyomet
of the forearm veins with a 20-gauge needle, syringe, and
each of the four exercise testing sessions, subjects were seated for
each experimental subject. Descriptive characteristics

Dietary analyses. Before the first exercise session each

Table 1. Descriptive characteristics

| Variable            | Mean ± SE
|---------------------|-----------
| Age, yr             | 23.8 ± 1.1|
| Resistance training, yr | 5.6 ± 0.9 |
| Height, cm          | 172.3 ± 2.2|
| Weight, kg          | 75.6 ± 2.4 |
| Body fat, %         | 13.3 ± 1.2 |
| 1-RM squat, kg      | 145.4 ± 11.3|
| 10-RM bench press, kg | 80.7 ± 4.2 |

Values are means ± SE for 12 subjects. RM, repetition maximum.

vacutainer setup. Although we were unable to schedule all

Figure 1 shows the pre- and postexercise serum T

RESULTS

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At room temperature before being centrifuged for 15 min at 1,500 g at

Immediate after the exercise protocol the subject was

immunoassay procedures. Immuneoreactivity values

Diet and hormones

50 DIET AND HORMONES

Hormonal concentrations. T and C concentrations used for simple regression were not significantly different from preexercise values for both exercise protocols.

Mean values and ranges for dietary energy and

Because of the variation in nutrient intake from day to
day within individuals (especially dietary cholesterol

The following table provides a summary of the

A 2-min rest period between all sets.

Bench press protocol consisted of five sets using a resistance
equal to each subject's pretest 10-repetitions maximum (RM)
bench press. The jump squat protocol involved performance of five sets of 10 continuous repetitions with a resistance equal to 30% of the subject’s 1-RM squat. Thirty percent of the 1 RM was chosen as the resistance because mechanical power is maximized near this value (30). Starting in an upright position, subjects were instructed to jump repeatedly as high as possible without pausing between repetitions within a set. There was an exactly 2-min rest period between all sets.

Dietary analyses. Before the first exercise session each

Table 2. Correlation coefficients obtained between preexercise T concentrations and dietary nutrients. Preexercise T was significantly positively correlated with percent energy fat, SFA (g·1,000 kcal−1·day−1), and MUFA (g·1,000 kcal−1·day−1) and was significantly negatively correlated with the percent energy protein, the PUFA/SFA ratio, and the protein-to-carbohydrate ratio (Fig. 2). There were no significant correlations observed between any nutritional variables and preexercise C or the absolute increase in T and C after exercise.

DISCUSSION

The primary finding from this investigation was that
dietary nutrients may influence resting concentrations of T in young athletic men. However, the resistance
other studies have used much shorter time periods to obtain individual food intake information; thus their reliability and accuracy may be questionable. Our results demonstrated that dietary protein, fat, SFA, MUFA, PUFA/SFA ratio, and protein-to-carbohydrate ratio were all significantly correlated with preexercise T concentrations. However, none of these dietary variables were significantly correlated with C concentrations. These data are consistent with the findings of several other investigations that have reported a decrease in T in individuals consuming a diet containing ~20% fat compared with a diet containing ~40% fat (7, 9, 13, 25). Vegetarians also consume less fat, SFA, and a higher PUFA/SFA ratio compared with omnivores, and vegetarians exhibit lower concentrations of T compared with omnivores (3, 11, 12, 15, 24). These data suggest that alteration in dietary energy and/or dietary composition has the potential to modify T concentrations. The results from several investigations strongly suggest that dietary fat has a significant impact on T concentrations; however, the influence of different types of lipids on T is not as clear. In the present investigation, dietary fat, SFA, and MUFA were the best predictors of resting T concentrations. Interestingly, Tegelman et al. (28) observed a significant positive correlation (r = 0.76) between percent energy fat and T in young athletic men, which is very similar to the correlation (r = 0.72) obtained in this study. Also, Adlercreutz et al. (1) reported significant positive correlations between T and dietary fat, SFA, MUFA, and cholesterol in postmenopausal women. The same nutrients were positively correlated with T in the present investigation except for cholesterol, which showed a correlation of r = 0.53 (P = 0.07) with T. In contrast to the results obtained in this study, Key et al. (15) reported a significant positive correlation (r = 0.37) between PUFA and T in male vegetarians and omnivores. Our results showed a nonsignificant correlation between PUFA and T and a significant negative correlation between the PUFA/SFA ratio and T. Thus dietary lipids appear to have a significant influence on resting T concentrations; however, the effect of different types of lipids on T regulation and metabolism is complicated and most likely influenced by a complex interaction of several...

### Table 2. Calculated daily intake of dietary energy and nutrients

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Mean</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy, kJ</td>
<td>9,899</td>
<td>4,962</td>
<td>13,364</td>
</tr>
<tr>
<td>Protein, %</td>
<td>20</td>
<td>14</td>
<td>33</td>
</tr>
<tr>
<td>CHO, %</td>
<td>56</td>
<td>48</td>
<td>69</td>
</tr>
<tr>
<td>Fat, %</td>
<td>23</td>
<td>10</td>
<td>32</td>
</tr>
<tr>
<td>SFA, g·1,000 kcal⁻¹·day⁻¹</td>
<td>7.6</td>
<td>2.9</td>
<td>12.6</td>
</tr>
<tr>
<td>MUFA, g·1,000 kcal⁻¹·day⁻¹</td>
<td>8.3</td>
<td>3.1</td>
<td>12.6</td>
</tr>
<tr>
<td>PUFA, g·1,000 kcal⁻¹·day⁻¹</td>
<td>4.6</td>
<td>2.3</td>
<td>7.4</td>
</tr>
<tr>
<td>Cholesterol, mg·1,000 kcal⁻¹·day⁻¹</td>
<td>109</td>
<td>66</td>
<td>168</td>
</tr>
<tr>
<td>PUFA/SFA</td>
<td>0.65</td>
<td>0.32</td>
<td>0.99</td>
</tr>
<tr>
<td>Dietary fiber, g·1,000 kcal⁻¹·day⁻¹</td>
<td>9.0</td>
<td>4.0</td>
<td>27.5</td>
</tr>
<tr>
<td>Protein/CHO</td>
<td>0.36</td>
<td>0.26</td>
<td>0.59</td>
</tr>
<tr>
<td>Protein/fat</td>
<td>2.46</td>
<td>1.02</td>
<td>6.91</td>
</tr>
<tr>
<td>CHO/fat</td>
<td>6.47</td>
<td>3.36</td>
<td>15.89</td>
</tr>
</tbody>
</table>

Nutrient percent values are percentage of total energy per day. CHO, carbohydrate; SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.

### Table 3. Correlation coefficients between preexercise testosterone concentration and selected nutritional variables

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Correlation With Testosterone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy, kJ</td>
<td>-0.18</td>
</tr>
<tr>
<td>Protein, %</td>
<td>-0.71*</td>
</tr>
<tr>
<td>CHO, %</td>
<td>-0.30</td>
</tr>
<tr>
<td>Fat, %</td>
<td>0.72*</td>
</tr>
<tr>
<td>SFA, g·1,000 kcal⁻¹·day⁻¹</td>
<td>0.77†</td>
</tr>
<tr>
<td>MUFA, g·1,000 kcal⁻¹·day⁻¹</td>
<td>0.79†</td>
</tr>
<tr>
<td>PUFA, g·1,000 kcal⁻¹·day⁻¹</td>
<td>0.25</td>
</tr>
<tr>
<td>Cholesterol, mg·1,000 kcal⁻¹·day⁻¹</td>
<td>0.53</td>
</tr>
<tr>
<td>PUFA/SFA</td>
<td>-0.63†</td>
</tr>
<tr>
<td>Dietary fiber, g·1,000 kcal⁻¹·day⁻¹</td>
<td>-0.19</td>
</tr>
<tr>
<td>Protein/CHO</td>
<td>-0.59†</td>
</tr>
<tr>
<td>Protein/fat</td>
<td>0.16</td>
</tr>
<tr>
<td>CHO/fat</td>
<td>0.16</td>
</tr>
</tbody>
</table>

Correlation coefficients are Pearson product-moment correlation. Nutrient percent values are percentage of total energy per day. *P ≤ 0.01. †P ≤ 0.005. ‡P ≤ 0.05.

MUFA, PUFA/SFA ratio, and protein-to-carbohydrate ratio were all significantly correlated with preexercise T concentrations. However, none of these dietary variables were significantly correlated with C concentrations. These data are consistent with the findings of several other investigations that have reported a decrease in T in individuals consuming a diet containing ~20% fat compared with a diet containing ~40% fat (7, 9, 13, 25). Vegetarians also consume less fat, SFA, and a higher PUFA/SFA ratio compared with omnivores, and vegetarians exhibit lower concentrations of T compared with omnivores (3, 11, 12, 15, 24). These data suggest that alteration in dietary energy and/or dietary composition has the potential to modify T concentrations.

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nutritional and metabolic factors. This complexity is illustrated by the findings of Sebokova et al. (26, 27), who reported that alteration in the testicular plasma membrane and changes in the responsiveness of Leydig cells and subsequent T synthesis occur as a result of ingestion of different compositions of lipids.

The significant negative correlation between protein and resting T concentrations is consistent with the findings of Anderson et al. (2), who demonstrated that a low-protein diet (10% of total energy) was associated with higher levels of T compared with a diet higher in protein (44% of total energy). The authors postulated that it was the protein-to-carbohydrate ratio in the diet that influenced either T metabolism or the liver-derived protein sex hormone-binding globulin (2, 14). Interestingly, the protein-to-carbohydrate ratio in the present study was significantly negatively correlated with resting T concentrations. Also, the source from which the protein is derived may influence T concentrations. Raben et al. (24) compared the effects of two diets differing only in the source of protein in male athletes. Results showed a reduced resting and postexercise increase in T concentrations in athletes consuming protein derived mainly from vegetable sources compared with a diet with protein derived mainly from animal sources. Thus not only the percent energy derived from protein in the diet but also the source of protein may influence T homeostasis.

The reason for a lack of a significant relationship between dietary nutrients and resting or resistance exercise-induced changes in C concentrations remains unknown. A number of factors related to the more dynamic nature of this hormone responding to stress and the differential storage, release, and synthesis mechanisms in glands along with differences in regulatory factors (e.g., blood flow) compared with T may partially explain our findings.

The fact that the absolute resistance exercise-induced increases in T and C concentrations were not significantly correlated with any nutritional variables
indicates that other mechanisms are responsible for the acute hormonal responses to exercise stress. The significant increase in T after both the bench press and jump squat exercise protocols confirms that high-intensity resistance exercise results in elevated concentrations of T (5, 8, 18, 19, 29). The fact that T was increased by ~15% after the jump squat exercise compared with ~7% after the bench press exercise was most likely due to the greater muscle mass used in the jump squat (16, 20). The lack of a significant C response to the resistance exercise protocols may have been due to the time of blood sampling or the amount of rest periods between sets (17). Finally, if blood samples had been obtained further into recovery, the possibility still exists that dietary nutrients may influence testosterone or cortisol concentrations.

In summary, the primary finding of this study was that resting concentrations of T may be partially explained by the amount and composition of dietary macronutrients. Our data suggest that the percentages of energy-providing macronutrients in the diet are important determinants of T homeostasis in healthy athletic men. Also, the type of lipid appears to influence circulating T concentrations. In this study, MUFA (g·1,000 kcal−1·day−1) and SFA (g·1,000 kcal−1·day−1) were the strongest predictors of T, accounting for 62 and 59% of the shared variance in T concentrations, respectively. These findings are particularly important for athletes training intensely who may experience a decline in T concentrations due to overtraining. Furthermore, this scenario may be exacerbated by a diet very low in fat, which many athletes (e.g., wrestlers, gymnasts, etc.) consume.

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REFERENCES


