TOTAL AND FREE TRIIODOTHYRONINE LEVELS ARE NOT ALTERED BY SEVEN DAYS OF ACTIVE HEAT ACCLIMATION

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INTRODUCTION

It has been suggested that thyroid gland activity in humans is depressed in the heat (1). This conclusion is based on findings of seasonal (winter vs. summer) changes in basal metabolic rate, protein bound iodine and serum hormone levels (1,2,3). For example, Gertner et al. (3) reported that total serum triiodothyronine (T3) levels were significantly decreased by 5 to 10% in the summer vs. the winter.

However, it is generally thought that free T3 is the biologically active component and that the bound fraction is inert (4). To our knowledge, no study has examined the response of free T3 to heat acclimation. Furthermore, most seasonal studies, by their very nature, measured thyroid responses several months apart. Conversely, laboratory-based heat acclimation protocols usually consist of only 6 to 10 successive days of heat exposure. Also, each heat exposure usually lasts 2 hours per day, with the remaining 22 hours per day being spent in a more temperate environment.

The above 2 issues led us to wonder if the T3 responses reported during seasonal acclimatization would be similar with shorter heat exposures. Therefore, the purpose of this study was to examine total and free T3 during a typical laboratory-based heat acclimation protocol.

METHODS AND MATERIALS

The subjects for this study were 9 healthy females with a mean age of 24 years. Each subject heat acclimated by exercising at approximately 30% of maximal oxygen uptake for 2 hours per day in an environmental chamber. The exercise bouts consisted of treadmill walking at 1.34 $m \cdot s^{-1}$ at a 3% grade or stationary cycling on a Monark ergometer at 75 W. Both modes of exercise produced absolute oxygen uptakes of approximately 1.2 $L \cdot min^{-1}$. Each subject completed 2 bouts of each mode of exercise, always finishing with treadmill walking. The chamber maintained the air temperature at 35°C with a relative humidity of 75%. During each heat exposure, cold water was allowed on an ad lib basis. Also, core temperature was measured using a rectal probe inserted 10 cm past the anal sphincter. Heart rate was measured using a Polar heart watch. Blood was collected via venipuncture in the morning (7 AM) prior to the heat exposure on days 1, 3 and 8. Total and free T3 was measured, in duplicate, using a radioimmunoassay technique (DPC, California, USA). The test-retest reliability of the total and free T3 assays were 0.96 and 0.95, respectively. Also, the coefficient of variation was approximately 5% for each assay.

Dependent variables were statistically analyzed during heat acclimation using repeated measures ANOVA. Significance was set at the $P < 0.05$ level.

RESULTS

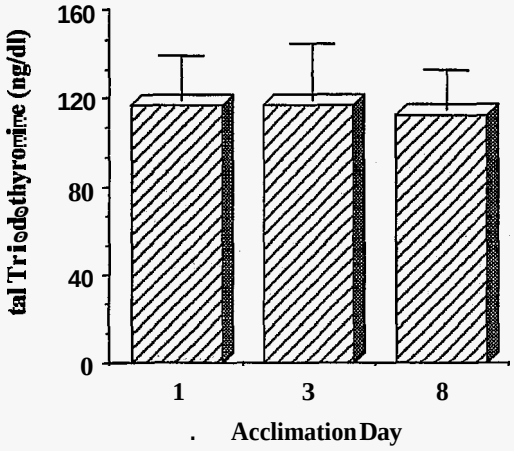
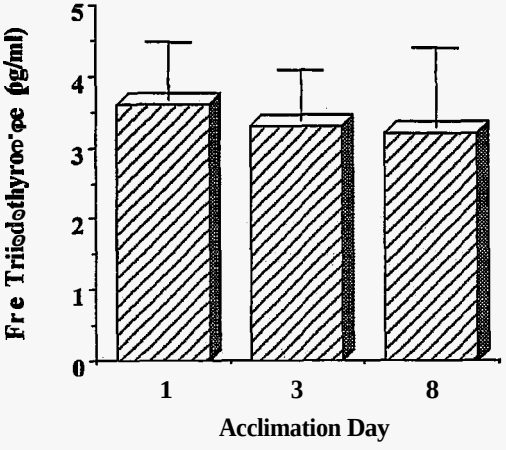


Figure 1. Mean triiodothyronine prior to heat exposure on days 1, 3, and 8.



Firgure 2. Mean free triiodothyronine prior to heat exposure on days 1, 3, and 8.

The mean (SD) ending rectal temperature on day 2, 3 and 7 was 38.9 ± 0.3, 38.8 ± 0.2 and 38.6 ± 0.4°C, respectively. Day 1 values were not presented because some of the subjects could not complete the 2-hour heat exposure and thus the values are low. Mean ending HR for the same 3 acclimation days was 157 ± 22, 153 ± 14 and 145 ± 17 bpm, respectively. Both HR and core temperature were significantly reduced during the 7 days of heat exposure, suggesting that our subjects did successfully acclimate. This is further supported by the magnitude of the reductions, which are consistent with previous studies. For example, Shvartz et al. (5) reported that heat acclimation decreased HR by 15 bpm and core temperature by 0.5°C.

Mean total T3 was 116 ± 21, 116 ± 26 and 112f 18 $ng \cdot dl^{-1}$ on days 1, 3 and 8, respectively. Mean free T3 was 3.6 ± 0.8, 3.3 ± 0.7 and 3.2 ± 1.1 $pg \cdot ml^{-1}$ on days 1, 3 and 8, respectively. Total and free T3 values are presented in Figure 1 and Figure 2, respectively. A repeated measures ANOVA showed non-significant changes for both total and free T3.

DISCUSSION

The non-significant changes in total and free **T3** found in the current study were unexpected.. Previously, it has been reported that chronic, seasonal heat exposure caused a significant **5** to **10%** reduction in total **T3 (3).** Specifically, metal shop workers in Israel had a total **T3** level of **180** ng·dl^{-1} during the summer **(25** to **32°C)** and **200** ngdl-' in the winter **(10** to **17°C).** Interestingly, total **T4** levels were higher in the summer vs. the winter. These results were interpreted to suggest that living in a hot climate reduces the peripheral degradation of **T4** into the metabolically more active T3. Further support for such a hypothesis was reported by Epstein et al. **(6).** They found that 1 hour of light exercise in the heat **(35°C,** 50% RH) caused a significant **20%** reduction in total **T3,** while **T4** levels were unchanged.

The above **2** studies **(3,6)** suggest that both chronic heat exposure and short-term **(1** hour) exercise in the heat can decrease total **T3** levels. Therefore, it seemed logical to hypothesize that similar responses would occur during a typical laboratory-based heat acclimation protocol. Such results, however, were not found in the current study.

Possibly the interval nature of most laboratory-based heat acclimation programs **(2** h·day^{-1} in the heat, **22** h·day^{-1} in temperate environments) does not provide enough stimulus to reduce total and free **T3.** In addition, the current study had a relatively small sample size and the acclimation period was only **7** days, both of which may have influenced the results. Further work in this area seems warranted.

CONCLUSIONS

The results of the current study suggest that a 7-day, laboratory-based heat acclimation protocol does not decrease total or free **T3** levels. Such a result does not agree with seasonal studies that have examined total **T3.**

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