# **PHYSIOLOGY**

# The Effect of Environmental Temperature and Thyrotropin Releasing Hormone on the Concentration of Thyroxine in Steers

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### Story in Brief

Twenty-four steers  $(342\pm8 \text{ lb})$  were exposed in environmental chambers to either  $40\pm1$ ,  $65\pm1$  or  $90\pm1^\circ\text{F}$  for 4 days (eight steers/group) following a 3-day adjustment period at  $65\pm1^\circ\text{F}$ . Ambient temperature was changed at a rate of  $3.6^\circ\text{F}$  per hour for 7 hours, and steers were treated with thyrotropin releasing hormone (TRH) at 1 and 72 hours after exposure to their respective environmental temperatures. There was no difference in the concentration of thyroxine during the change in ambient temperature. There was a tendency (P<.1) for the concentration of thyroxine after treatment with TRH to be less with increased environmental temperature ( $84.3\pm3.7$ ,  $78.4\pm2.4$  and  $61.9\pm1.2$  ng/ml for 40, 65 and 90°F, respectively). The concentrations of thyroxine at all environmental temperatures were greater (P<.001) after the first treatment with TRH compared to the second treatment ( $87.0\pm2.7$  vs.  $62.5\pm1.6$  ng/ml, respectively).

## Introduction

Environmental temperature has been demonstrated to alter the function of the thyroid gland and output of thyroid hormones in many species. In general, increased environmental temperatures decrease the concentration of thyroxine, whereas cool environmental temperatures increase thyroxine. Alterations in the concentration of thyroxine regulate the rate of body metabolism. Thyrotropin releasing hormone (TRH), normally produced by the hypothalamus, causes a release of thyroid-stimulating hormone from the pituitary gland, which in turn causes a release of thyroxine from the thyroid gland. The objectives of this study were to determine the effect of environmental temperature on the concentration of thyroxine and to evaluate the influence environmental temperature has on the concentration of thyroxine in response to treatment with TRH in steers.

### **Materials and Methods**

Twenty-four Angus x Hereford steers weighing an average of  $342\pm8$  lb were assigned to environmental temperatures of  $40\pm1$ ,  $65\pm1$  or  $90\pm1^\circ$ F. Two environmentally controlled chambers with two steers per chamber were used in six replicates with two different environmental treatments represented in each replicate. The steers assigned to each replicate were penned indoors at a temperature of  $65\pm5^\circ$ F two weeks prior to being moved into the environmental chambers. Steers were acclimated to confinement in the environmental chambers at a temperature of  $65\pm1^\circ$ F three days prior to exposure to the environmental treatments. Steers were fed 3 lb of ground alfalfa hay, 3 lb of cottonseed hulls and 1 lb of a 12-percent protein grain supplement per day with water available free choice. On the third day of acclimation to the environmental chambers, cannulae were inserted into the jugular vein of each steer. The morning following cannulation the temperature was changed at a rate of  $3.6^\circ$ F per hour until the steers were exposed to their preassigned environmental treatments (40, 65 or 90°F). Blood samples were collected via cannulae every hour 2 hours prior to and during the change in environmental temperature. One hour after steers were exposed to either 40, 65 or 90°F, they were treated with 50 µg of thyrotropin releasing hormone (TRH) administered intravenously. Blood samples were collected immediately prior to the first treatment with TRH, every 15 minutes for the first hour, hourly for the next 8 hours and every 8 hours for 3 days. Three days after the first treatment with TRH, steers were treated a second time with TRH. Serum blood samples were quantified for thyroxine by radioimmunoassay validated in our laboratory.

#### **Results and Discussion**

During the change in ambient temperature there were no differences in the concentrations of thyroxine between the three environmental treatments (Figure 1). The average response of thyroxine after both treatments of TRH decreased (P<.1) as the ambient temperature increased, with thyroxine averaging  $84.3\pm3.7$  ng/ml for  $40^{\circ}$ F,  $78.4\pm2.4$  ng/ml for  $65^{\circ}$ F and  $61.9\pm1.2$  ng/ml for  $90^{\circ}$ F. The response of thyroxine to TRH was significantly greater after the first treatment with TRH compared to the

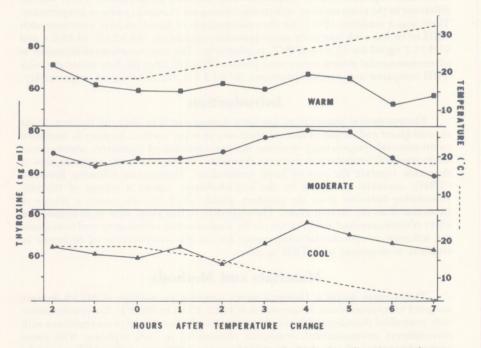
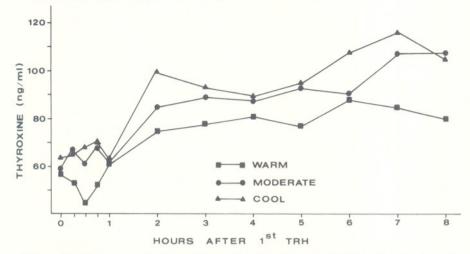
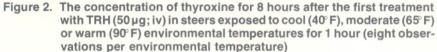


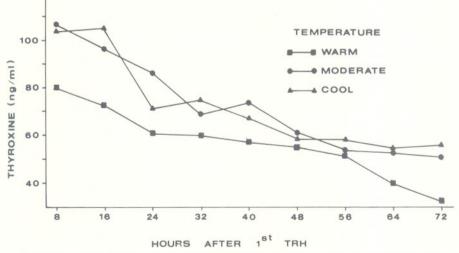
Figure 1. The concentration of thyroxine (ng/ml) during the change in ambient temperature (eight observations per treatment group)

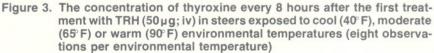
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second treatment  $(87.0\pm2.7 \text{ vs.} 62.5\pm1.6 \text{ ng/ml}; P<.001)$ . The response of thyroxine to TRH was less (P<.05) at the 90°F over the 8-hour period after treatments of TRH compared to the moderate and cool environmental temperatures (Figures 2 and 4). This was particularly evident after the second challenge of TRH (Figure 4; P<.05). The concentration of thyroxine in samples collected every 15 minutes for 1 hour after

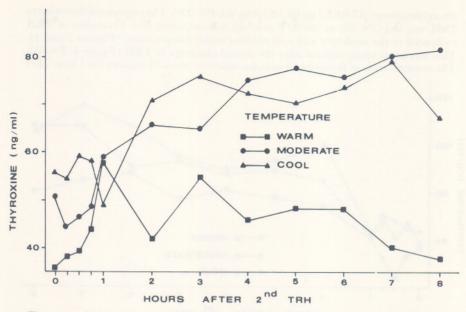


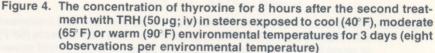






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both treatments with TRH varied over time (P < .05) but was not affected by ambient temperature (Figures 2 and 4). The concentration of thyroxine declined in samples collected every 8 hours for 72 hours after the first treatment with TRH in all environmental treatments (Figure 3).

Rectal temperature, respiratory rate and water intake increased from preexposure values (P<.01) in the steers exposed to 90°F. Hematocrit decreased in all groups throughout the trial (P<.01), but the decrease was more dramatic in the steers exposed to the high environmental temperatures. A more detailed description of the physiological changes in these steers has been reported previously (Pratt and Wettemann, 1980).

These results suggest that the functioning of the thyroid is affected by ambient temperature, with high environmental temperatures suppressing the output of thyroxine. The concentration of thyroxine was not significantly altered by exposure to 40°F compared to 65°F.

The reduced response in release of thyroxine after the second treatment with TRH compared to the first treatment may be due to a negative feedback to thyroxine on the hypothalamus and pituitary from the elevated concentration of thyroxine following the first treatment with TRH. This feedback mechanism may be responsible for the gradual decline of thyroxine observed in the samples collected every 8 hours for 3 days between the treatments with TRH (Figure 3).

### **Literature Cited**

Pratt, B.R. and R.P Wettemann. 1980. Okla. Agr. Exp. Sta. Res. Rep. MP-107:170.

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