EFFECT OF COLD ENVIRONMENT ON PROTEIN SYNTHESIS IN CALVES

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Introduction

Ruminants adapt to cold stress with a variety of hormonal and metabolic responses, one of which may involve a change in protein turnover in one or several tissues. Protein accretion results when protein synthesis exceeds protein degradation; therefore, changes in either parameter in response to cold stress could affect growth. Thompson et al. (1987) reported that when calves are exposed to a cold environment, the rate of N^{τ}-methylhistidine excretion (an index of myofibrillar protein degradation) increases. Effects of environmental temperature on protein synthesis in ruminants have not been reported. However, in chicks, the fractional synthetic rate (FSR) of whole body protein is elevated during chronic cold exposure (Aoyagi et al., 1988). Similarly, cold exposure increases the FSR of liver protein in rats, while it depresses the FSR of soleus muscle protein (McAllister, 1987). The objective of the present study was to examine the effect of chronic cold exposure on the rate of protein synthesis in tissues of the growing calf.

Materials and Methods

Eleven Holstein calves 35 d of age were allocated to one of three treatments: 1) warm environment (20°C), 72 g feed $kg^{-0.75}$ BW d^{-1}; 2) cold environment (- 5°C), 72 g feed $kg^{-0.75}$ BW d^{-1}, or 3) cold environment (−5°C), 90 g feed $kg^{-0.75}$ BW d^{-1}. Calves were allowed to adapt to the treatments for 21 d, during which time they were fed a pelleted alfalfa/barley ration every 2 h from an automatic feeder. At 55 d of age, an infusion catheter was inserted into the right ventricle via the right jugular vein and a sampling catheter was inserted into the left jugular vein. At 56 d of age, each calf received a continuous infusion of L-[ring-2,6-^{3}H] phenylalanine in sterile physiological saline (10 mmol Phe/L; a total of 40 μCi kg BW^{-1} infused) at a rate of 30 ml h^{-1} for 8 h.

During infusion, calves continued to receive feed every 2 h. Blood samples were taken every 20 min and kept on ice in heparinized tubes until centrifugation within 2 h to obtain plasma. At the end of the infusion period, each calf was anesthetized with sodium pentobarbitol and exsanguinated. Tissues were immediately removed, weighed, and homogenized in ice-cold 2% (w/v) perchloric acid. The homogenate supernatant represents the intracellular free Phe pool and the homogenate pellet represents the protein bound Phe pool. Plasma, supernatants, and pellets were stored at −50°C until analysis. The specific radioactivity (SRA) of Phe was determined in plasma, supernatants, and pellets according to the method outlined by McAllister (1987). Assuming that intracellular free Phe is the precursor pool for protein synthesis, the FSR (% d^{-1}) of protein in each tissue was calculated as described by Davis et al. (1981).

Results

The FSR (% d^{-1}) of protein in tissues from calves adapted to the warm and cold environments are shown in table 1. The FSR of protein in kidney, longissimus dorsi and biceps femoris in cold-exposed calves fed the low level of intake were lower ($p < 0.02$) than in cold-exposed calves fed the high level of intake or in warm-exposed calves fed the low level of intake. The FSR of protein in the tissues was similar for the cold-exposed calves fed the high level of intake and warm-exposed calves fed the low level of intake. FSR of protein in skin showed trends similar to that for muscle and kidney, although the difference was not significant ($p = 0.13$). There were no significant differences between treatments for the FSR of protein in the rumen papillae, omasum, rumen, intestine, and liver. Values for the gastrointestinal tissues were quite variable.

Discussion

The FSR of protein in rumen, intestine, and

TABLE 1. PROTEIN FRACTIONAL SYNTHETIC RATE (FSR, % D^{-1}) IN TISSUES FROM CALVES IN THREE TREATMENTS (MEAN ± SEM)

Tissue	Treatment 1	Treatment 2	Treatment 3
Environmental Temperature(°C)	20	−5	−5
Feed Intake (g feed $kg^{-0.75}$ BW d^{-1})	72	72	90
Rumen Papillae	58.9± 8.3	63.7± 9.0	60.0±20.1
Omasum	70.7± 8.5	64.2± 8.4	69.7±17.1
Rumen	42.2± 7.7	47.8± 4.2	48.3±13.8
Intestine	141.6±62.9	101.1±28.4	86.8±39.1
Kidney	32.4± 0.8[a]	28.4± 0.7[b]	31.7± 1.0[a]
Liver	29.5± 0.9	32.3± 2.8	28.5± 2.8
Heart	9.4± 0.6	9.4± 0.3	10.4± 1.0
Long. Dorsi	2.7± 0.6[a]	1.6± 0.7[b]	2.9± 0.2[a]
Biceps Femoris	3.3± 0.0[a]	1.5± 0.2[b]	2.8± 0.4[a]
Skin	12.2± 1.9	6.5± 1.0	11.5± 3.1

[a,b]*Means in a row with different superscripts are significantly different* ($p < 0.02$).

skeletal muscles reported here are similar to values reported for steers by McBride et al. (1989), but the FSR of protein in kidney, liver, and heart are higher. This difference can probably be accounted for by the difference in age of experimental animals used. The decreased FSR of protein in the longissimus dorsi and biceps femoris muscles in cold-exposed calves agrees with results for the soleus in cold-exposed rats (McAllister, 1987), while the decreased FSR of protein in kidney in cold-exposed animals has not been reported before.

Decreased FSR of protein in muscle and kidney was observed in cold-exposed calves on the restricted feed intake, but not in cold-exposed calves on the 25% higher level of intake, when compared with warm-adapted calves on the restricted feed intake, pointing out the role that food intake plays in metabolic responses to cold stress. Thompson et al. (1987) demonstrated that while *ad libitum* feed intake of cold-exposed calves increased by 18%, myofibrillar protein breakdown increased in these calves as well as in calves on a restricted intake. It is likely that increased skeletal muscle protein breakdown made amino acids available for use as gluconeogenic and energy substrates to meet increased energy requirements in the cold. Therefore, even though cold-exposed calves on a high level of intake may have an elevated rate of protein degradation, increased nutrient supply allows them to maintain a level of protein synthesis equal to that of animals in the warm. Because the cold-exposed calves on a restricted intake can not increase nutrient intake, protein synthesis falls in certain tissues. Skeletal tissue is a large and labile protein reserve, and thus can adapt to metabolic demands imposed by cold stress without compromising its function. The significance of the proportionately smaller response in the kidney is not clear.

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(Key Words: Cold, Protein Synthesis)

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