

Effects of short-term exposure to heat stress on splanchnic metabolism in sheep

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ABSTRACT

Four wethers with chronic indwelling catheters in the portal, hepatic and mesenteric veins as well as in the abdominal aorta were housed in temperature-controlled rooms at 20°C or 35°C. The sheep consumed a diet at the level of maintenance requirements with both temperatures. Hepatic blood flow tended to be greater at 35°C than 20°C. The absorption of acetate and propionate as well as the oxygen uptake by the portal-drained viscera were significantly greater at 35°C than at 20°C. The increased oxygen uptake may be one cause of the heat load under hot temperature.

KEY WORDS: heat stress, nutrient absorption, oxygen consumption, splanchnic tissue, sheep

INTRODUCTION

Animal exposed to excessive heat load reduces feed intake to maintain homeothermy. Ambient temperature, relative humidity and metabolic heat with maintenance and production are important factors relating to the heat load. The portal-drained viscera (PDV) and liver use much oxygen due to the high metabolic activity, and the proportion of oxygen uptake by the total splanchnic (TS) tissues accounts for over 40% of total oxygen use by the whole body (Huntington, 1999). McGuire et al. (1989) have reported that the net flux of alpha amino nitrogen across the PDV of lactating cows was reduced by heat stress. However, few data on net flux of the other metabolites and oxygen consumption by PDV and liver under heat stress are available. We investigated the influence of hot temperature on metabolite net flux and oxygen consumption across the splanchnic tissues of sheep.

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MATERIAL AND METHODS

Four wethers (average body weight 64.9 kg) were surgically fitted with chronic indwelling catheters in the hepatic and hepatic portal vein, mesenteric vein and caudal aorta. All sheep housed in metabolic crates were offered the diet consisting of 70% alfalfa hay cubes and 30% rolled barley in 12 equal daily meals using automatic feeders. The daily amount of the diet was restricted at the maintenance energy level to exclude any influence associated with different feed intake. The room temperature as treatment was controlled at 20°C and 35°C with a relative humidity of 64 and 48%, respectively. From the aspects of animal welfare, the confinement in 35°C room as heat stress treatment was restricted to three days. On day 3, thus, blood samples of the artery and the portal and hepatic vein at 35°C were taken every 30 min for three hours. The blood samples at the comfortable room temperature (20°C) were taken after 14 days for the preliminary period. *p*-Aminohippurate was continuously infused (2% w/v, 1 ml/min) into the mesenteric vein catheter for measuring blood flow. Treatment means were compared by paired-t test.

RESULTS AND DISCUSSION

There were no refusals of the offered diet with any of the temperature treatments. Table 1 summarizes the blood flow, oxygen consumption and metabolite net flux across the splanchnic tissues with both temperature treatments. Hepatic blood flow tended to be greater ($P=0.15$) at 35°C compared with 20°C. Oxygen consumption by PDV at 35°C increased by 15% compared with 20°C, which resulted in increased oxygen consumption by TS ($P<0.05$). Net absorption of acetate and propionate across PDV were greater ($P<0.01$) at 35°C than at 20°C. The PDV net flux of isobutyrate and isovalerate also tended to be greater at 35°C ($P<0.14$). Consequently, the absorption of total short chain fatty acids increased by 42% at 35°C compared with 20°C. These results suggest that as heat stress promotes ruminal fermentation of ingested substrates, the increased short chain fatty acids as fermentation products need more oxygen for the absorption metabolism by the ruminal tissue. Heat stress reduces the gut motility of ruminants (Christopherson and Kennedy, 1983). In this study, reduced gut motility at 35°C might cause the prolongation of digesta retention time in the rumen, resulting in the increase absorption of fermentation products from same dietary substrates.

Although hepatic uptake of propionate at 35°C compared with 20°C increased ($P<0.05$), the net glucose production by the liver did not differ. Because the liver can regulate gluconeogenesis under rigid glucose homeostasis, the increased propionate uptake at 35°C was used for other metabolic processes or fueled through the TCA cycle. Net release of propionate by TS was not different between the treatments. On

the other hand, little uptake of acetate by the liver resulted in a tendency of the greater release by TS ($P < 0.15$). Heat stress did not affect the net fluxes of butyrate and β -hydroxybutyrate across the splanchnic tissues.

Table 1. Blood flow, oxygen consumption and net flux of metabolites across portal, hepatic and total splanchnic tissues in sheep exposed to 20 and 35°C

Item	20°C	35°C	SE	P ¹
Portal blood flow, L/h	145	155	7.8	0.460
Hepatic blood flow, L/h	169	184	9.6	0.150
<i>Oxygen consumption, mmol/h</i>				
portal	131	151	4.2	0.017
hepatic	98	104	4.7	0.610
total splanchnic	229	255	6.3	0.030
<i>Net portal flux, mmol/h</i>				
acetate	60.5	88.1	7.67	0.001
propionate	18.3	25.1	1.73	0.006
isobutyrate	0.8	1.3	0.16	0.138
butyrate	3.7	3.3	0.61	0.553
β -hydroxybutyrate	17.5	17.0	1.31	0.668
glucose	-4.6	-6.5	4.09	0.821
NEFA	2.6	3.4	1.30	0.759
<i>Net hepatic flux, mmol/h</i>				
acetate	-0.6	43.2	18.3	0.310
propionate	-17.1	-23.0	1.53	0.036
isobutyrate	-0.4	-1.2	0.21	0.077
butyrate	-2.2	-1.8	0.42	0.608
β -hydroxybutyrate	13.3	15.7	1.92	0.641
glucose	19.3	21.5	4.07	0.847
NEFA	-6.3	-6.0	1.04	0.876
<i>Net total splanchnic flux, mmol/h</i>				
acetate	59.8	131.3	22.95	0.147
propionate	1.2	2.2	0.40	0.230
isobutyrate	0.4	0.1	0.14	0.400
butyrate	1.5	1.4	0.26	0.910
β -hydroxybutyrate	30.8	32.7	2.47	0.672
glucose	14.7	15.0	2.76	0.970
NEFA	-3.7	-2.6	0.89	0.451

¹ probability that treatments differ

CONCLUSIONS

Hot temperature compared with comfortable environment at a same level of feed intake increased short chain fatty acids absorption from the digestive tract and PDV oxygen consumption. The increased oxygen consumption by PDV could be one cause of heat load driven from metabolic process under heat stress.

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