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RESEARCH ARTICLE

Effects of high ambient temperature on lipid metabolism in finishing pigs



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Abstract

In this study, we investigated the effects of high ambient temperature on lipid metabolism in finishing pigs. Sixteen pigs ((79.6±1.2) kg) were randomly assigned to two groups: (1) ambient temperature of 30°C with *ad libitum* access to feed (HT; *n*=8) and (2) ambient temperature of 22°C and fed the average amount consumed by eight pigs in HT group on the previous day (PF; *n*=8). After 21 days of constant exposure to different environmental conditions, all the pigs were euthanized, and blood and tissue samples were obtained. High ambient temperature increased the proportion of backfat ($P=0.04$, +21.6%) and flare fat ($P<0.01$, +43.3%). Compared with pair-fed pigs, the activities of fatty acid synthase (FAS) and malic enzyme in backfat and flare fat were lower ($P<0.05$) in heat-stressed pigs, as were the amounts of acetyl-CoA-carboxylase and FAS in the *longissimus* muscle (LM), the amount of FAS in backfat ($P<0.01$), and FAS activity in the liver ($P<0.01$). Ambient temperature did not affect the amount of hormone-sensitive lipase in different tissues. The amount of lipoprotein lipase in flare fat tended to be higher ($P=0.09$, +28.3%), and the activities of β -hydroxyacyl coenzyme A dehydrogenase in front and back of LM were lower ($P<0.01$, -48.3 and -49.8%, respectively) at 30°C than at 22°C. The plasma concentration of high-density lipoprotein tended to be lower ($P=0.08$), but the plasma concentrations of very low-density lipoprotein (VLDL) ($P=0.09$, +50.0%) and nonesterified fatty acid (NEFA) ($P=0.04$, +44.2%) were higher in heat-stressed pigs. We concluded that high ambient temperature depressed *de novo* fatty acid synthesis in both adipose tissues and the liver. However, β -oxidation of fatty acid in skeletal muscles was also inhibited in the high-temperature environment. As a result, more plasma NEFAs were used to synthesize VLDLs in the liver and were absorbed by adipose tissues. This may be one reason that high ambient temperature enhances the accumulation of backfat and flare fat in finishing pigs.

Keywords: β -hydroxyacyl coenzyme A dehydrogenase, lipoprotein lipase, lipogenesis, finishing pigs, high ambient temperature

1. Introduction

It is well documented that heat stress reduces feed intake (Close and Stanier 1984; Verstegen and Close 1994; Quiniou *et al.* 2000) and decreases the weight of flare fat and backfat in pigs (Rinaldo and Le Dividich 1991; Becker *et al.* 1992; Collin *et al.* 2002). However, at a similar level

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of feed intake, high ambient temperature directly increases the proportion of flare fat and the lipid content of backfat (Kouba *et al.* 2001), which indicates that heat stress directly promotes lipid deposition in the flare fat and backfat of pigs.

The fatty acids used for synthesizing triglycerides (TGs) are derived mainly from *de novo* synthesis in adipose tissues in swine (O’Hea and Leveille 1969). Heat stress depresses the activities of malic enzyme (ME) and glucose-6-phosphate dehydrogenase (G6PDH) in the backfat and flare fat of pigs (Rinaldo and Le Dividich 1991). Even at a similar level of feed intake, acetyl-CoA-carboxylase (ACC) activity is lower in heat-stressed pigs (Kouba *et al.* 1999). These findings indicate that *de novo* fatty acid synthesis is inhibited in the backfat and flare fat of heat-stressed pigs.

Plasma TG-rich lipoproteins, i.e., intestinal chylomicrons and hepatic very low-density lipoprotein (VLDL), are other sources of fatty acids that synthesize TGs in adipose tissues by the action of lipoprotein lipase (LPL) (Steffen and Frobish 1978). Kouba *et al.* (2001) found that heat stress increased LPL activity in adipose tissues and plasma VLDL concentrations, and they speculated that heat stress might enhance lipid metabolism in adipose tissues and the liver. However, high temperature depressed *de novo* fatty acid synthesis in the liver (Kouba *et al.* 1999). We speculated that high temperature might inhibit the β -oxidation of fatty acids in muscles, sequentially more plasma nonesterified fatty acids (NEFAs) were to be re-esterified and reassembled as VLDLs in the liver and reabsorbed by adipose tissues. To test this hypothesis, two groups of finishing pigs were maintained at different temperatures and received similar amount of feed in order to examine the influence of heat stress on lipid metabolism.

2. Results

The high ambient temperature had no significant effect on carcass weight, backfat thickness, or lipid content of *longissimus* muscle (LM) ($P=0.21$, 0.80, 0.67, respectively). The relative weight of backfat was 21.6% higher ($P=0.04$) and flare fat was 43.3% higher ($P=0.07$) in the HT group than in the PF group, the pigs in the HT group were maintained at 30°C with *ad libitum* access to feed, the pigs in the PF group were maintained at 22°C and were fed the average amount consumed by eight pigs in HT group on the previous day (Table 1).

The effects of a high environmental temperature on *de novo* synthesis of enzymes of fatty acid in different tissues are shown in Fig. 1. Heat stress decreased the amount of fatty acid synthase (FAS) ($P<0.01$) and the activity of FAS ($P<0.05$) and ME ($P=0.03$) in backfat. The activities of FAS and ME in flare fat ($P<0.03$), amounts of ACC and FAS in LM ($P<0.01$), and the activity of FAS in the liver were lower

Table 1 Effects of high ambient temperature on fat accumulation in finishing pig¹⁾

Item ²⁾	HT ³⁾	PF ⁴⁾	SEM ⁵⁾	P-value
Carcass weight (kg)	77.28	79.95	2.04	0.21
Backfat thickness (cm)	3.17	3.10	0.26	0.80
Relative backfat weight (%)	17.09	14.06	1.31	0.04
Relative flare fat weight (%)	7.58	5.29	0.07	0.07
Lipid content of LM (%)	1.89	2.01	0.08	0.67

¹⁾ Values are means of eight repeats.

²⁾ Relative backfat weight and relative flare fat weight were expressed as % of the empty body weight (BW); lipid content of *longissimus* muscle (LM) was expressed as g 100 g⁻¹ LM tissue.

³⁾ Means from pigs maintained at an ambient temperature of 30°C, with *ad libitum* access to feed.

⁴⁾ Means from pigs maintained at an ambient temperature of 22°C and fed the average amount consumed by the 30°C group.

⁵⁾ SEM, stand error of mean.

The same as below.

($P<0.01$) in the HT group than in the PF group.

High ambient temperature had no significant effect on the plasma concentrations of cholesterol (CHOL), TG, or low-density lipoprotein cholesterol (LDL-C) ($P>0.10$). The plasma concentration of high-density lipoprotein cholesterol (HDL-C) tended to be lower ($P=0.08$), VLDL tended to be higher ($P=0.09$, +50.0%), and NEFA was higher ($P=0.04$; +44.2%) in the HT group (Table 2).

Heat stress tended to have increased the amount of LPL in flare fat ($P=0.09$, +28.3%) and markedly decreased β -hydroxyacyl coenzyme A dehydrogenase (HAD) activity in the front ($P<0.01$; -48.3%) and back ($P<0.01$; -49.8%) of LM. High ambient temperature did not affect the amount of HSL in different tissues (Table 3).

3. Discussion

The upper critical temperature of the thermoneutral zone for finishing pigs is 28.5°C (Verstegen and Close 1994), so we chose 30°C for the heat-stressed group. As reported by Hao *et al.* (2014), we found that a high temperature of 30°C had no significant effects on gain-to-feed ratio (G:F) and average daily feed intake (ADFI) compared with the PF group, which was not consistent with Song *et al.* (2011). This might be because the high temperature they chose was much higher than ours (37°C vs. 30°C). However, average daily gain (ADG) was numerically lower ($P=0.21$; 0.65 vs. 0.75 kg d⁻¹) and final weight was significantly lower ($P=0.03$; 97.5 vs. 102.8 kg) in the HT group. At the end of the experiment, pigs maintained at 30°C had a higher rectal temperature than their pair-fed counterparts maintained at 22°C, which was in accordance with the results of Fernandez *et al.* (2014) and Mendoza (2014).

Heat stress reduces feed intake (Le Dividich *et al.* 1987; Katsumata *et al.* 1996; Cruz 1997), decreases metabolic energy intake (Renaudeau *et al.* 2012) and decreases

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