

Effects of Heat Stress and Plane of Nutrition on Production and Metabolism in Growing Pigs

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Summary and Implications

We heat-stressed growing pigs and evaluated the differential effects of environmental hyperthermia and reduced feed intake. Our results indicate that hyperthermia directly effects production and metabolism and many of these are independent of reduced nutrient intake. Identifying how and why heat stress alters metabolism and physiology are prerequisites in developing ameliorating strategies to prevent the reduction in performance and lost income typically observed during the warm summer months.

Introduction

Suboptimal livestock growth limits the U.S. meat industry's productive competitiveness and marginalizes efforts to reduce inputs into food production. The U.S. swine industry has made efficient production a high priority and, as a result, has realized rapid improvements in the lean growth of market pigs. Unfortunately, heat stress (HS) during both fetal development and postnatal life undermines advances made by the pork industry. The economic burden is primarily due to reduced growth and poor sow performance, but also includes increased veterinary costs, and decreased carcass value. As a consequence HS is currently one of the costliest issues in the U.S. pork industry and compromises the swine industry's capacity to efficiently produce animal protein for human consumption. In addition, earth's climate is predicted to continually warm at an unprecedented rate, and some models forecast extreme summer conditions in most U.S. pig-producing areas. Further, because basal heat production has markedly increased with selection for enhanced lean tissue accretion rates some suggest faster growing animals are more sensitive to HS. Therefore, genetic selection based upon traditional production traits may increase pigs' susceptibility to thermal stress. Consequently, defining the biology and mechanisms of how HS jeopardizes pig performance is critical in developing approaches to ameliorate current production issues and is a prerequisite for generating future mitigating strategies to improve pig well-being, growth performance and agriculture economics.

Materials and Methods

We exposed pigs to 38°C (humidity: 25-35%) for 7 d at the Iowa State University Swine Nutrition Farm. In an attempt to differentiate between the direct effects of HS and reduced nutrient intake (indirect effects of HS) on animal physiology/metabolism, we allowed one group of thermal neutral (TN) pigs to consume feed ad libitum and pair-fed another group of TN pigs (PFTN) to the same level of intake as the pigs experiencing HS. The disruption in the nutrient intake:growth relationship emphasizes the importance of our experimental design because it enables us to evaluate HS while eliminating the confounding effects of dissimilar nutrient intake. This design is also critical in determining the extent to which HS directly or indirectly (i.e. through reduced feed intake), plays a role in physiological and metabolic adaptations occurring during HS.

Results and Discussion

Pigs exposed to HS conditions had an immediate and sustained increase ($P < 0.01$) in rectal temperature (39.3 vs. 40.8°C) and a doubling in respiration rates (54 vs. 107 bpm). Feed intake in HS pigs was markedly reduced; by design, the PFTN controls mirrored that of the HS group (Figure 1A). Along with reduced feed intake, HS pigs had a marked decrease in body weight gain (compared to the TN ad libitum pigs), while the PFTN pigs on average lost body weight (Figure 1B), which can be interpreted to suggest profound metabolic alterations in HS leading to different nutritional needs for maintenance and growth. Thus, reductions in feed intake differentially affect body weight parameters in HS and PFTN controls.

During HS, pigs had an immediate (24 h) increase in plasma NEFA levels, but the NEFA concentrations returned to normal within 3 d and remained low throughout the experiment (7 d: Figure 2A). Plasma insulin in HS pigs was inversely related to NEFA levels and linearly increased during the 7 d trial (Figure 2A). Blood was only collected on the 7th d for the PFTN controls and their basal NEFA and insulin levels were typical (high and low, respectively) of a malnourished animal (Figure 2B,C). The increased basal insulin and decreased NEFA levels agree with our recent data in HS rodent and growing and lactating ruminants. Importantly, we have shown that the stimulated insulin response is higher in HS animals and the lipolytic response to an adrenergic challenge is blunted in HS ruminants. The differential regulation of adipose tissue mobilization and circulating insulin (a key lipogenic hormone) between environments is perplexing given that both groups of pigs were on a lowered plane of nutrition. Taken together, it appears distinct shifts in whole-body metabolism

accompany the onset of HS in growing pigs, and are unexplainable by nutritional status.

Collectively, our preliminary data and the literature suggest HS alters pre and postnatal development performance in pigs by preventing the enlistment of nutrient partitioning mechanisms, which would normally allow for maximal growth and protein accretion. This is in part driven

by epigenetic events, compromised intestinal integrity and altered tissue growth dynamics.

Acknowledgments

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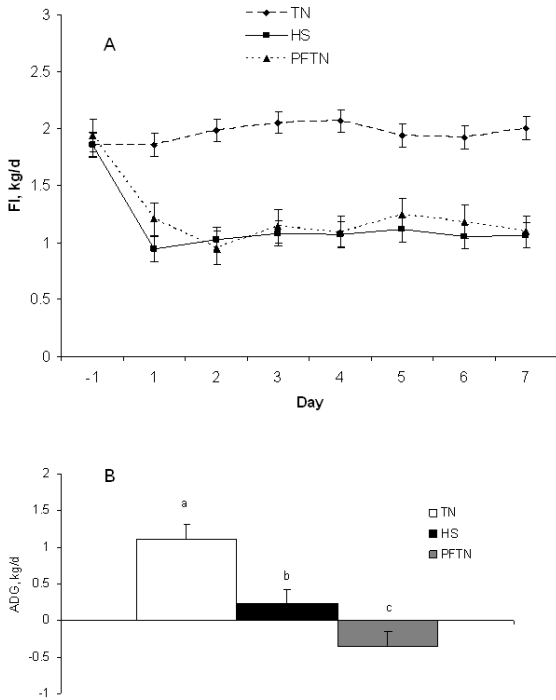


Figure 1. A: Effects of ad libitum feeding in thermal neutral conditions (TN), heat stress (HS) or pair-feeding in thermal neutral conditions on daily feed Intake. B: Effects of ad libitum feeding in thermal neutral conditions (TN), heat stress (HS) or pair-feeding in thermal neutral conditions on average daily gain

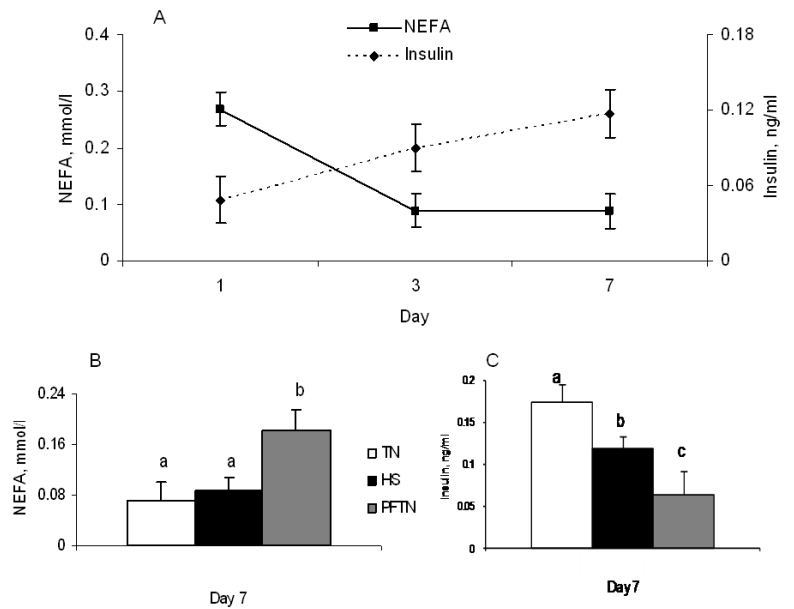


Figure 2. A: Effects of heat stress on the temporal basal plasma levels of NEFA and insulin in growing pigs. B: Basal plasma NEFA concentrations in thermal neutral (TN), heat-stressed (HS) and pair-fed thermal neutral (PFTN) control pigs. C: Basal plasma insulin concentrations in thermal neutral (TN), heat-stressed (HS) and pair-fed thermal neutral (PFTN) control pigs.