Heat Stress and Marbling Development in Beef Cattle

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The effect of heat stress on skeletal muscle depends on its intensity and duration. Short-term moderate exposure can be adaptive. It induces heat shock proteins, increases satellite cell proliferation through HSP27, and maintains protein synthesis via the mTOR pathway. Beyond a protective threshold, the response becomes catabolic, with reactive oxygen species accumulation, mitochondrial dysfunction, and increased protein degradation. Beef-on-Dairy systems are of particular interest because they expose animals during two windows: late gestation and an extended finishing period. We exposed Holstein dams to heat stress or active cooling during the final 54 d of gestation. In utero heat stress lowered offspring birth weight and average daily gain and shifted the semitendinosus toward a Type I oxidative phenotype with higher heat shock protein abundance and satellite cell activity. In feedlot steers, 28 d of heat stress reduced dry matter intake, adjusted final body weight (p = 0.013), and hot carcass weight (p = 0.038), with no change in backfat, ribeye area, or KPH fat. Marbling did not increase. Energy was partitioned toward subcutaneous and visceral fat, and muscle shifted to an oxidative metabolism that draws on adjacent lipid. Heat stress, aggressive stacked implants, and β-adrenergic agonists each divert nutrients from intramuscular adipogenesis, and their effects on marbling are additive. Dietary fat supplementation may help preserve marbling by maintaining intramuscular and intramyocellular lipid reservoirs. These data point to nutrition and management strategies that extend thermal tolerance and protect carcass quality during heat stress.

Keywords : heat stress, skeletal muscle, marbling, intramyocellular lipid, fetal programming