donors through JIVET to produce 75% dairy F2 offspring and then inter-crossing the lines to generate homozygous sires. Gene editing of the "slick" mutation directly into dairy sires is not a practical option in NZ at the current time.

Further improvements in tropical dairy cow development are possible if more genetic variations associated with heat tolerance are found, although introgression of additional variations (for example, coat color) will be challenging in a breeding program such as that outlined here.

Key Words: Senepol, thermoregulation, dairy

0405 Genetic solutions to infertility caused by heat stress. P. J. Hansen*[1], S. Dikmen[2], J. B. Cole[3], M. S. Ortega[1], and G. E. Dahl[1], *[1]Department of Animal Sciences, University of Florida, Gainesville, [2]Uludag University, Faculty of Veterinary Medicine, Department of Animal Science, Bursa, Turkey, [3]Animal Genomics and Improvement Laboratory, USDA-ARS, Beltsville, MD.*

Reproductive function in mammals is very susceptible to disruption by heat stress. In lactating dairy cows, for example, pregnancy rates per insemination can be as low as 10–15% in the summer vs. 25–40% in cool weather. Reduced fertility is caused by a combination of (1) the negative consequences of the physiological adjustments engaged to minimize hyperthermia during heat stress and (2) direct deleterious effects of elevated body temperature on the gamete and embryo (i.e., heat shock). There is genetic variation body temperature regulation during heat stress as well as in cellular resistance to elevated temperature. Thus, opportunities exist for improving reproduction during heat stress by modifying livestock genetically to improve body temperature regulation and cellular resistance to heat shock. Genetic improvement can be achieved by identifying genetically superior animals within a breed (heritability for rectal temperature during heat stress is 0.17) as well as by transferring genes from thermotolerant breeds to thermosensitive ones. A successful example of gene transfer is for a mutation in *PRLR* causing the slick hair phenotype. Holstein cattle inheriting this mutation have increased ability to regulate body temperature during heat stress and are less likely to experience a decrease in milk yield during summer than other Holsteins. Among the genes conferring cellular resistance to heat shock is a mutation in the promoter of *HSPA1L* identified in cattle. Selection for the beneficial allele of this gene, as well as other genes controlling cellular resistance to heat shock, might reduce the damage to the oocyte and embryo caused by elevated body temperature.

Key Words: heat stress, infertility, reproduction, body temperature

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0406 Resilience and lessons from studies in genetics of heat stress. I. Misztal*, *University of Georgia, Athens.*

Production environments are expected to change, mostly to hotter climates but also possibly more extreme and drier. This raises a question whether the current generation of farm animals can cope with the changes or should they be specifically selected for changing conditions. In general, genetic selection produces animals with smaller environmental footprint but also with smaller environmental flexibility. Some answers are coming from heat stress research across species, with heat tolerance partly understood as a greater environmental flexibility. Specific studies in various species show complexities of defining and selecting for heat tolerance. In Holsteins, the genetic component of heat stress on production approximately doubles in second and quadruples in third parity. Best production under heat stress is by cows with elevated body temperature, probably at a risk of increased mortality. In hot but less intensive environments, the effect of heat stress on production is minimal although the negative effect on fertility remains. Mortality peaks under heat stress and increases with parity. In Angus, the effect of heat stress is stronger only in selected regions, probably due to adaptation of calving seasons to local conditions and crossbreeding. Genetically, while the direct effect shows variability due to heat stress, the maternal does not, probably due to dams shielding calves from environmental challenges. In pigs, the effect of heat stress is strong in commercial but almost none in nucleus farms. This is partly due to lower pig density and better heat abatement in nucleus farms. Under intensive management, heat stress is less evident in drier environments because of more efficient cooling. A genetic component of heat stress exists but it is partly masked by improving management and selection based on data from elite farms. Genetic selection may provide superior identification of heat-tolerant animals but a few cycles may be needed for clear results. Also simple traits exist that are strongly related to heat stress, e.g., slick hair in dairy and shedding intensity in Angus. Defining resilience/robustness may be difficult especially when masked by improving environment. Under climate change, the current selection may be adequate if it (1) is accompanied by constantly improving management, (2) uses commercial data and (3) includes traits important under climate change such as mortality.

Key Words: G x E interaction, animal stress, robustness