16-12
Proposal #

2016 NERA Planning Grants Program

Project Title: Poor maternal nutrition and its impact on neonatal outcomes

Team Members		
Name	Discipline	Institution/Agency/Other
Steven Zinn	Endocrinology	CT Exp. Stat.
Kristen Govoni	Growth Biology	CT Exp. Stat.
Sarah Reed	Muscle Biology	CT Exp. Stat.
Sabrina Greenwood	Metabolism	VT Exp. Stat.
Ryan Arsenault	Signal Transduction	DE Exp. Stat.
Carol Bagnell	Neonatal Development	NJ Exp. Stat.
Kim Vonnahme	Fetal Programming	ND Exp. Stat.
Joel Caton	Developmental Programming	ND Exp. Stat.
Larry Reynolds	Placental Physiology	ND Exp. Stat.
Caleb Lemley	Placental Development	MS Exp. Stat.
Derris Burnett	Muscle Biology	MS Exp. Stat.
Ryan Ashley	Placental Development	NM Exp. Stat.
Min Du	Muscle Biology	WA Exp. Stat.
Guoyao Wu	Amino Acid Metabolism	TX Exp. Stat.
Stephen Ford	Fetal Programming	WY Exp. Stat.
Sean Limesand	Metabolic Endocrinology	AZ Exp. Stat.
Stephanie (Thorn)	Fetal Metabolism	Univ. Colorado School of Medicine (CO)
Wesoloski		
Rachael Gately	Ultrasound/Fetal Development	Tufts Cummings Veterinary School (TVM)

(Attach an additional sheet if more space is needed.)

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Rationale. With the global population approaching 9.5 billion by 2050 (FAOSTAT, 2015), there is a critical need to produce an adequate supply of high quality animal protein, to feed the growing population. In addition, management systems must maintain animal health and production efficiency. In livestock, exposure to poor maternal nutrition, resulting from restricted and over-feeding, during gestation alters prenatal and postnatal growth of the offspring. Specifically, these animals exhibit undesirable changes to body composition (increased fat, reduced muscle), metabolic disorders, and organ dysfunction (Ford et al., 2007; Long et al., 2009, 2011; Hoffman et al., 2016a). Consequently, these changes can lead to poor health, poor reproductive status, reduced production efficiency, and reduced quantity and quality of meat and milk products (Wu et al., 2006; Reynolds et al., 2010). Therefore, the effects of poor maternal nutrition during gestation on offspring development are detrimental to animal agriculture and ultimately food security. Poor maternal nutrition can be caused by nutrient restriction or overfeeding, with negative phenotypic outcomes observed in the offspring. Importantly, these negative effects can persist into adulthood (Yan et al., 2011; Huang et al., 2012) and can be multi-generational (Dunn et al, 2009; Ford et al., 2012). To date, several studies have demonstrated the connection between poor maternal nutrition and persistent negative effects in the offspring. However, the mechanisms mediating the long-term and multigenerational implications on health, growth, and development are not well characterized (Grandjean et al., 2015). Furthermore, evaluation of therapeutic agents or management regimens that mitigate the negative effects of poor maternal nutrition are limited. Identifying strategies is critical to provide opportunities to improve production practices, which will further improve the health and well-being, production efficiency and food security.

Significance to the Northeast (NE). Controlling maternal diet during gestation can be challenging due to various management, environmental and economic factors. For example, the management practice of flushing or increasing nutrient intake to increase the number of (oocytes) ovulated (Shad et al., 2011) can result in over-feeding of the dam during early and mid-gestation. In addition, the pasture-based management systems used in the NE (Steinberg and Comerford, 2009) can also contribute to poor maternal nutrition during gestation, primarily because quality and quantity of pasture varies greatly throughout an average year with spring and summer months exhibiting the greatest quality and quantity. However, by late summer and early fall this tends to decrease with pasture reaching the poorest quality in the fall. This period of decreasing pasture quality and quantity corresponds to the gestation period for many agricultural species used in the NE (e.g., beef cattle, sheep and goats). Further variations with year, season, temperature and rainfall exacerbate this problem when pasture quality and quantity are limited. Therefore, under current management practices used within the NE, many offspring are born to dams that were poorly nourished (restricted or over-fed) during gestation which impacts offspring health, development and growth, thereby reducing the sustainability and profitability of NE livestock operations.

Introduction. Maternal restricted and over-nutrition results in reduced lean-to-fat ratio (Zhu et al., 2006), reduced muscle cross-sectional area (Bayol et al., 2005; Reed et al., 2014), and increased fat deposition (Bee, 2004; Reed et al., 2014) in the offspring. Nutrient restriction during early or late gestation results in fewer muscle fibers in lambs (Costello et al., 2008) and an increased number of glycolytic myofibers (Zhu et al., 2006). Moreover, fetal muscle in lambs from obese ewes had decreased diameter of primary muscle fibers and increased collagen content (Huang et al., 2010; Yan et al., 2011) which can negatively impact meat tenderness (Oury et al., 2009; Kang et al., 2011). These alterations to body composition are persistent into adulthood (Yan et al., 2011; Huang et al., 2012). In addition to changes in muscle, fat and connective tissue, maternal nutrient restriction and overfeeding alters concentrations of key circulating factors (eg; insulin, IGFI, IGFBP3, and leptin; Ford et al., 2007; Long et al., 2011; Hoffman et al., 2014, 2016a) that are critical for regulating animal growth and

metabolism. Poor maternal nutrition during gestation can have long-term negative effects on the metabolism of the offspring as determined by reduced insulin sensitivity (Ford et al., 2007), altered cellular metabolism (Thorn et al., 2011, 2013), and increased expression of pro-inflammatory mediators (Yan et al., 2011; Ge et al., 2013). In an effort to understand what factors may be mediating these observed phenotypic changes in muscle, our research group using next generation sequencing, determined that genes involved in cell proliferation, cellular metabolism and signal transduction are reduced in the muscle tissue of offspring born to restricted and over-fed dams (Hoffman et al., 2016b). Furthermore, we were able to identify that despite a common phenotype (Reed et al., 2014) the mechanisms by which the muscle tissue development was altered appear to be different (Hoffman et al., 2016b). While these findings are novel, additional research is needed to better understand how poor maternal nutrition causes the physical changes observed in the offspring and the molecular mechanisms mediating these changes. In turn, this information can be used to develop effective intervention strategies to address the problem that poor maternal nutrition poses to animal agriculture and food security.

This group of scientists from these 11 experiment stations, University of Colorado Medical School (UCO) and Tufts Cummings School of Veterinary Medicine (TVM) have developed experimental models using livestock (primarily sheep) that focus on molecular, cellular, and whole animal response to poor maternal nutrition during gestation both in the dam and in the offspring during pre-, peri-, and post-natal periods of development, with additional expertise in high throughput peptide analyses, next-generation sequencing, metabolism, and signal transduction. Thus, the participants in this planning grant bring diverse expertise to the field of poor maternal nutrition and its impact on neonatal outcomes with major areas of focus on the dam and the offspring.

Areas of research focusing on the dam include development of the placenta (MS, ND, NM, WY) and maternal blood supply (ND) to the fetus, alterations in the endocrine system (AZ, CT, UCO) and inflammatory status (CT, WY), as well as potential management tools to mitigate the negative effects to the offspring (MS, ND).

Areas of research focus in the offspring include developmental and metabolic changes in muscle, satellite cells, bone, adipose, liver, pancreas and mesenchymal stem cells (AZ, CT, DE, NJ, VT, TX, UC, WA) as well as changes in pre- and postnatal changes in body weight and body composition (CT, MS, ND, NJ, NM, TX, WY, TVM).

Scientists from NE experiment stations have specific expertise in muscle and bone physiology nextgeneration sequencing, endocrinology and growth biology (CT), high throughput peptide analyses and signal transduction (DE), developmental biology and peri-natal growth (NJ), and ruminant metabolism and nutrition (VT). Therefore, given their specific areas of expertise, these scientists will have a central role in the implementation of this planning grant.

Overall, this group of scientists is uniquely qualified to investigate mechanisms that contribute to poor growth and development of offspring as a result of poor maternal nutrition at the molecular, cellular and whole animal level, as well as evaluate therapeutic intervention strategies that mitigate the negative effects of poor maternal nutrition. Importantly, the collaboration established with this planning grant will increase the opportunities to collaborate on specific experiments, apply for regional and federal grants, and therefore utilize animal resources more efficiently.

The overall goals of this planning committee proposal are:

- 1. To bring together scientists from NE Experiment Stations (CT, DE, NJ, VT) and veterinarians (TVM) with scientists from experiment stations outside the NE (AZ, MS, ND, NM, TX, WA and WY) and UCO that have diverse expertise to foster multi-institutional collaborations to address research questions focused on addressing the effects of poor maternal nutrition on offspring growth and metabolism.
- 2. To develop cross disciplinary multi-state research proposals that integrate a variety of expertise to enhance our understanding of the mechanisms by which poor maternal nutrition during gestation alters multi-generational growth and development that will provide opportunities to improve production practices and identify therapeutic interventions that mitigates the negative effects of poor maternal nutrition.

Achieving these goals will result in 1) the identification of the mechanisms that cause the negative phenotypic changes in offspring born to poorly nourished dams and 2) allow for the development of new management tools to improve livestock production efficiency, product quality and to enhance sustainability of livestock production systems in the NE. Collaboration with Cooperative Extension System (CES) specialists and teaching faculty throughout the NE and the country will provide outreach opportunities to disseminate new technologies and management tools to current and future producers. Moreover, including scientists from UCO and TVM will provide a medical and veterinary perspective to the project and provide additional outreach and educational opportunities at their institutions and in the communities they serve.

Use of the Planning Committee Grant. The first step to address the goals of the project is to organize a meeting for scientists from the NE and other participating experiment stations and associated institutions. CT would serve as the host institution for a 1.5 day meeting. Each Experiment Station would present their experimental approach(es), data, potential for shared samples, and future plans (~45 minutes each). Ample opportunity for discussion around each presentation will be scheduled. The meeting will conclude with a discussion about preparing integrative grant proposals to appropriate agencies (eg., USDA-NIFA, NIH, NSF, USDA-NIH Dual purpose grants, SARE).

The primary products of the Planning Committee will be 1) the integration of scientists from the NE with other scientists from the United States, using their experience and expertise to address an issue that has significant relevance to producers in the NE; 2) the development and publication of a review article updating the 'state of the field' since reviews by Wu et al. (2006) and Du et al. (2010); and 3) the development and submission of grant proposal(s) to fund collaborative projects with participating investigators to identify key mechanisms and develop intervention strategies tailored to NE livestock production systems.

The request for the planning committee grant is \$10,000 which will be used to offset costs of scientists to attend. The grant will cover transportation (\$100 to \$700 per station; \$6,000) and meals at the meeting (\$2,000) with the up to \$2,000 to assist with lodging. Each Experiment Station/PI will be responsible for any additional housing costs and a portion of the travel costs if multiple scientists from a single station participate. The UConn Animal Science Department will match \$1,000 towards meeting rooms, any AV requirements, food, and shuttles from the airport to campus. In addition, the CT Station will match \$1,000 towards costs of the meeting (see letter Appendix 3). If needed, publication charges will be requested from the authors if a manuscript is accepted for publication.

Appendix 1: References

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Hoffman, M. L., K. N. Peck, M. E. Forella, A. R. Fox, K. E. Govoni, and S. A. Zinn. 2016a. The effects of poor maternal nutrition during gestation on postnatal growth and development of lambs. J. Anim. Sci. 94: 789-799.

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Huang, Y., J. X. Zhao, X. Yan, M. J. Zhu, N. M. Long, R. J. McCormick, S. P. Ford, P. W. Nathanielsz, and M. Du. 2012. Maternal obesity enhances collagen accumulation and cross-linking in skeletal muscle of ovine offspring. PloS one 7: e31691.

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Wu, G., F. W. Bazer, J. M. Wallace, and T. E. Spencer. 2006. Board-invited review: Intrauterine growth retardation: Implications for the animal sciences. J. Anim. Sci. 84: 2316-2337.

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BIOGRAPHICAL SKETCH

Steven Zinn			POSITION TITLE: Head and Professor				
Department of Animal Science			Department of Animal Science, UConn				
Unit 4040							
University of Connecticut (UConn)							
Storrs, CT 06268							
EDUCATION/TRAINING							
INSTITUTION AND LOCATION	DEGREE						

INSTITUTION AND LOCATION	DEGREE	YEAR(s)	FIELD
Cornell University, Ithaca, NY	BS	1978	Animal Science
Michigan State University, E. Lansing, MI	MS	1984	Animal Science
Michigan State University, E. Lansing, MI	PhD	1989	Animal Science
Worcester Foundation, Shrewsbury, MA	Post Doc	1989-1990	Molecular Biology

Research Experience:

2005-present - Professor, Department of Animal Science, UConn

1996-2005 - Associate Professor, Department of Animal Science, University of Connecticut

1990-1996 - Assistant Professor, Department of Animal Science, University of Connecticut

1989-1990 - Postdoctoral Associate, Worcester Foundation for Exp. Biology, Shrewsbury, MA

1980-1989 - Graduate Assistant, Department of Animal Science, Michigan State University

Research Interests: The effects of maternal nutrition in growth and development.

Selected Research Awards (Selected awards since 2000):

- 2000-2003: Novel delivery systems of porcine somatotropin to stimulate growth rate, feed efficiency and carcass composition in growing pigs. Connecticut Innovations, Inc., \$175,393 plus \$35,000 from Drug Smart, Inc.
- 2000-2003: Effects of zinc on nuclear actions of thyroid hormone. USDA NRI Competitive Research Grants Program, Co-PI with Hedley Freake, direct cost, \$ 109,323.
- 2004-2006 Physiological and genetic factors contributing to differences between two genetic lines of IGF-I divergent cattle. University of Connecticut Research Foundation, \$25,102.
- 2005-2010 STRONG-CT: Science and Technology, Reaching Out to New Generations in Connecticut. NSF, Steven A Zinn, Co-PI H. Freake D. Khan, M. Philion, M. Jehnings, direct cost, \$1,999,995.
- 2007-2009 Using the somatotropic axis as a model to predict nutritional status in free-ranging Steller sea lions. University of Connecticut Research Foundation. \$12, 953.
- 2013: Evaluation of the Antigenicity of Novel DNA-based Foot and Mouth Disease Virus Vaccines in Swine, Inovio Pharmaceuticals, Co-PI with K. Govoni, direct cost \$19,000.
- 2014-2016: Effects of intrauterine growth retardation (IUGR) on fetal development in sheep. USDA-NIFA AFRI Foundational Nutrition, Growth and Lactation Program, direct cost \$110,555.
- 2015-2017: Effects of poor maternal nutrition on muscle progenitor cell function and metabolism. USDA-NIFA AFRI Foundational Nutrition, Growth and Lactation Program, Co- with S. Reed, K. Govoni, direct cost, \$150,000.
- Awards (Selected awards since 2008):
- 2016 American Society of Animal Science (ASAS) President Elect
- 2016 University of Connecticut Teaching Fellow
- 2015 Fellow of the American Society of Animal Science
- 2014 H. Allen Tucker H. Allen Tucker Lactation & Endocrinology Award, ASAS
- 2008-2013 Editor-in-Chief, Journal of Animal Science
- 2011-2014 Editor-in-Chief, Animal Frontiers

B. Publications: (selected publications since 2010):

Richmond, J.P., T. Norris, S.A. Zinn. 2010. Re-alimentation in harbor seal pups: Effects on the somatotropic axis and growth rate. Gen. Comp. Endocrinol. 165: 286-292.

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Glynn E.R., A.L. Sanchez, S.A. Zinn, T.A. Hoagland, and K.E. Govoni. 2013 Culture conditions for equine bone marrow mesenchymal stem cells and expression of key transcription factors during their differentiation into osteoblasts. J. Anim. Sci. Biotech. 4:40 (DOI: 10.1186/2049-1891-4-40).

McGonagle, A., H.C. Freake, S.A. Zinn, T. Bauerle, J. Winston, G. Lewicki, M. Jehnings, D. Khan-Bureau, and M. Philion. 2014. Evaluation of STRONG-CT: A program supporting minority and first-generation U.S. science students. J. STEM Ed.: Innovat. Res. 15:52-61

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Zinn, S.A. 2015 H. Allen Tucker Lactation and Endocrinology Award: Graduate Education: Lessons from my mentor. J. Anim. Sci. 93: 12: 5594-5596. doi: 10.2527/jas2015-8869

Jones, A.K., R.E. Gately, K.K. McFadden, S.A. Zinn, K.E. Govoni, and S.A. Reed. 2016. Transabdominal ultrasound for detection of pregnancy, fetal and placental landmarks, and fetal age before day 45 of gestation in the sheep. Theriogenology 10.1016/j.theriogenology.2015.11.002.

Hoffman, M.L., K.N. Peck, M.E. Forella, A.R. Fox, K.E. Govoni, and S.A. Zinn. 2016. The effects of poor maternal nutrition during gestation on postnatal growth and development of lambs J. Anim. Sci. 94: 2: 789-79910.2527/jas2015-9933.

Raja, J.S., M. L. Hoffman, K. E. Govoni, S. A. Zinn, and S. A. Reed. 2016. Restricted maternal nutrition alters myogenic regulatory factor expression in satellite cells of ovine offspring. Animal 10.1017/S1751731116000070.

Hoffman, M.L., K.N. Peck, J.L. Wegrzyn, S.A. Reed, S.A. Zinn, and K.E. Govoni. 2016. Poor maternal nutrition during gestation alters the expression of genes involved in muscle development and metabolism in lambs. J. Anim. Sci. doi: 10.2527/jas.2016-0570.



College of Agriculture, Health and Natural Resources Ratcliffe Hicks School of Agriculture Office of Academic Programs

August 25, 2016

To:

Steven Zinn Head, Animal Science Department

Camer Jauran

From: Cameron Faustman Associate Dean/Director

This is to follow up our recent discussion regarding your 2016 NERA Planning Grant proposal entitled, "Poor maternal nutrition and its impact on neonatal outcomes". The purpose of this memo is to formally confirm that the CAHNR research office will provide you with a match of 10% of the approved budget, up to a total of \$1,000, should your proposal be selected for funding. Best wishes for success!

Cc: L. Grabowski