partially hindering the phage receptors on the cell surface. Moreover, it is possible that the production of EPS increases the viscosity of the medium and perhaps, this molecular crowding slightly affect the spread of the phage infection. It remains to be seen if the sugar composition or the structure of some EPS produced by LAB may be involved in phage sensitivity or insensitivity. Nonetheless, it is now clear from the above evidences that we should not solely rely on the EPS production to protect starter cultures against phages.

Key Words: Exopolysaccharide (EPS), Bacteriophage, Lactic acid bacteria (LAB)

Physiology
Basic Mechanisms Regulating Anovulatory States

465 Neuroendocrine mechanisms underlying seasonal breeding in the ewe. RL Goodman*[1], GM Anderson[1], VL Adams[1], SL Hardy[1], JM Connors[1], and MN Lehman[2], [1]*West Virginia University,* [2]*University of Cincinnati.*

It is now clear that an increase in response to the negative feedback action of estradiol (E) is responsible for the inhibition of ovarian function in anestrous ewes. In early work, we demonstrated that A15 dopaminergic (DA) neurons play a key role in this response. These neurons mediate E negative feedback in anestrus and their response to E varies seasonally. Because A15 cells do not contain estrogen receptors (ER), other neurons most likely provide information on E levels to them in anestrus. In more recent work, we have focused on the E-responsive component of this circuitry and have identified two important areas: the ventromedial preoptic area (vmPOA) and retrochiasmatic area (RCh). ER-positive cells in both areas project to the A15, and local administration of E to either area inhibits LH secretion in anestrus, but not during the breeding season. Furthermore, the inhibition of LH in anestrus can be overcome by a DA-receptor antagonist. We thus postulated that the neural circuit mediating E negative feedback in anestrus includes E-responsive perikarya in the vmPOA and RCh that project to the A15 and stimulate these DA neurons, which in turn inhibit GnRH release. This hypothesis raises the possibility that structural changes within this circuit may contribute to the seasonal alterations in response to E negative feedback. Therefore, we tested if there is a seasonal variation in synaptic input to A15 perikarya using dual immunocytochemistry to stain for synaptic varicosities (synapsin I) and DA perikarya (tyrosine hydroxylase). Confocal microscopic analysis indicated a significant increase in synaptic close contacts on A15 dendrites in anestrous ewes. This increase in synaptic input correlated with a significant increase in the dendritic arborization of these neurons. Thus seasonal morphological changes within the neural system mediating E negative feedback may well play an important role in the mechanisms responsible for seasonal breeding in the ewe. Supported by NIH HD-17864

Key Words: Seasonal breeding, estrogen negative feedback, sheep

466 Nutrition and suckling mediated anovulation in beef cattle. R.P. Wettemann*, C.A. Lents, N.H. Ciccioli, F.J. White, and I. Rubio, *Oklahoma Agricultural Experiment Station, Stillwater.*

Nutrient intake, body energy reserves, and suckling are major regulators of reproductive performance of beef cows. Inadequate body energy reserves at parturition increase the interval to first estrus and ovulation, and postpartum nutrient intake can influence length of the interval in cows with thin to moderate body condition (BCS). Suckling can increase the postpartum anestrous interval in thin cows but has little effect on mature cows with adequate body energy reserves. Reduced nutrient intake can delay the onset of puberty and cause cessation of estrous cycles. The objective of this presentation is to evaluate signals by which nutrient intake and body energy reserves may regulate ovarian function. Nutritional restriction causes decreased secretion of LH, reduced follicular growth, and decrease concentrations of estradiol in plasma. Pituitary concentrations of LH were reduced and concentrations of FSH were greater in nutritionally induced anovulatory cows that were ovariectomized compared with proestrous cows. Acute energy restriction of postpubertal heifers resulted in decreased IGF-I in plasma, inhibition of the proestrus increase in estradiol and the ovulatory surge of LH, and anovulation. In addition to direct and indirect effects of decreased energy intake on the hypothalamus and pituitary, nutrition may influence ovarian function. Metabolic signals such as insulin, IGF-I, and leptin may regulate functions of the pituitary and ovary. Concentrations of IGF-I in plasma during late gestation are correlated ($r = 0.33$; $P < 0.01$) with BCS, and concentrations of IGF-I and leptin in plasma are greater in postpartum cows that have increased energy intake. Concentrations of IGF-I in plasma increase preceding ovulation when nutritionally induced anovulatory cows are realimentated. Nutrient intake and BCS alter metabolic signals at the hypothalamus and pituitary that control secretion of LH, and reduced stimulation by metabolic signal at the ovary may compromise the ability of follicles to respond to gonadotropins.

Key Words: Anovulation, Nutrition, Beef Cattle

467 Nitric oxide and the ovary. Carlo Tamanini*, Giuseppina Basini, and Francesca Grasselli, *Dip. Prod. Anim., Biotec. Vet., Qual. Sic. Alim., University of Parma-Italy.*

Nitric oxide (NO) is synthesized from L-arginine by NO synthase (NOS), an enzyme with three isoforms; two of them, neuronal and endothelial (n and eNOS) are constitutive, while the third one, iNOS, is inducible. NO is effective in mediating multiple biological effects, at least in part through the activation of soluble guanylate cyclase (cGMP); among these, smooth muscle cell tone, platelet aggregation and adhesion, cell growth, apoptosis and neurotransmission. Being that these mechanisms are associated with the pathophysiology of several reproductive processes, it became clear that NO could play a key role in reproduction. Apart from its effects through the modulation of LHRH release, NO has been proven to act directly at the ovarian level, where it has been demonstrated to be produced by the vasculature and neurons as well as by various cell types, including granulosa, theca and luteal cells; its production is modulated by several hormones (P4, LH, FSH and hCG) and cytokines which interfere with either eNOS or iNOS expression and activity. Experiments performed with NO donors and/or NO synthase inhibitors have demonstrated that NO reduces apoptosis and inhibits both E2 and P4 production by granulosa cells (at least in part via cGMP). NO is possibly involved in follicle growth. In fact, it is a potent mitogen in the presence of basic fibroblast growth factor (bFGF), it increases the receptors for epidermal growth factor on granulosa cells and, as mentioned above, it regulates the programmed cell death (which is an important part of folliculogenesis). The gonadotropin-stimulated eNOS and iNOS expression as well as the inhibition of ovulation by NOS inhibitors suggest that NO participates in the ovulatory process. After ovulation, iNOS is expressed in luteal cells but its activity diminishes with the corpus luteum development; during the luteolysis phase NO stimulates PGF2α synthesis while reducing P4 secretion. The overall information provides convincing evidence that NO plays a critical role in the ovarian physiology with regard to follicle growth, ovulation and corpus luteum function, even if its clinical implications have not been clarified yet.

Key Words: Folliculogenesis, Ovulation, Corpus luteum