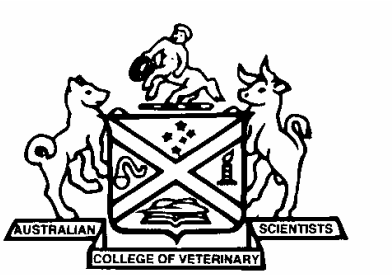

AUSTRALIAN COLLEGE OF VETERINARY SCIENTIST 2008 ANNUAL CONFERENCE

COLLEGE SCIENCE WEEK

GOLD COAST INTERNATIONAL HOTEL

3-5 JULY 2008

EQUINE CHAPTER CONFERENCE PROGRAM AND PROCEEDINGS

Scientific Meeting Equine Chapter Program Coordinator – Lee Morris and John Chopin

Proceedings Editor – John Chopin

PROGRAM

	Tuesday	Wednesday	Thursday	Friday	Saturday
	Surgery Chapter	Surgery Chapter	**Combined with Surgery Chapter**	**Equine Chapter**	**Equine Chapter**
8.30	Registration	Transendoscopic laser assisted ventriculo-cordectomy in roarers Dr Paul Robinson	How To Treat Joint Infections Antibiotic Treatment Dr Jane Axon Regional Perfusion Dr Paul Robinson Interosseus perfusion Dr Jane Axon	8.30 – 8.55 EI – the veterinary/EV A response. Dr James Gilkerson 8.55 – 9.10 The Challenges and Logistics of Containing an EI Outbreak. Dr Nathan Anthony	8.30 – 9.00am Endometritis update Dr Lee Morris
9.00	Equine Head Surgery Professor Tim Greet	Surgery of the Larynx Prof Tim Greet	Arthroscopy Prof Tim Greet	9.10 – 9.25 EI – A practitioners view – the early picture. Dr Rowan Sedgwick	9.00 – 10.00am AGM
			Arthroscopic management of coxofemoral joint sepsis. Dr Tom Russell	9.25-9.40 EI – A practitioners view II – the end result. Dr John Chopin	
				9.40-10.00 EI – The pathology of EI in the field. Dr Joan Carrick.	
10.00	Morning Tea	Morning Tea	Morning Tea	Morning Tea	Morning tea
10.30	Sinus and Dental Surgery Professor Tim Greet	Surgery of the Pharynx Prof Tim Greet	**How to:** place a transphyseal screw Dr Paul Robinson	10.30 – 11.30 The history of EI. Dr Jenny Mumford	10.30-10.50 A new thought for the aetiology of equine placentitis. Dr John Chopin

					10.50 –11.10 The effectiveness of ultrasound monitoring to prevent placentitis. Dr Joan Carrick 11.10-11.30 What do we really know about pregnancy recognition in the horse: Is eCG the same as hCG? Dr Jim McFarlane
11.30	MRI Advances in Lameness Assoc Prof Matt Stewart	Surgical treatment of hoof injuries Dr Todd Booth	Other Surgical Techniques Prof Tim Greet		11.30-11.50 Toxic syndrome in Thoroughbred broodmares associated with the grazing of Fescue pasture. Dr Deb Racklyeft
12.00	Comparing 2 Methods of Pastern Arthrodesis Dr Todd Booth	Sinoscopy Assoc Prof Matt Stewart	Casting for hoof and pastern injuries Assoc Prof Matt Stewart	11.30 - 12.00 The enquiry and legal ramifications of EI. Dr James Gilkerson 12.00 – 12.30 The virology of EI. Dr Jenny Mumford	11.50 – 12.10 The role of leptin in reproduction and fertility Dr Jim McFarlane 12.10-12.30 Open Discussion
12.30	Lunch	Lunch	Lunch	Lunch	Lunch
1.30	Male Urogenital Surgery Prof Tim	New approaches to equine fractures	Resident/Intern Papers NatureVet Prize	**Combined with Epidemiology Chapter**	1.30 – 2.30 EHV update – Dr Jenny Mumford

	Greet	Prof Tim Greet	1.30-1.45 Cellular response to exercise induced hyperthermia in the equine superficial flexor tendon – Sarah Burrows 1.50-2.05 Review of insulin resistance in equids, association with laminitis, treatment and prevention – Kellie Tinworth 2.10-2.25 Three dimensional microstructure of the metacarpus of Thoroughbred racehorses with catastrophic limb fracture Gareth Trope	1.30 – 2.30 Sense, Science and Compliance in the control of EI. Dr Jenny Mumford	
2.30	Incidence of roarers and risk factors for the development of the disease Dr Paul Robinson	Review of literature relating to epiglottic entrapment Dr Paul Robinson	2.30-2.45 Wendy Goodwin	2.30 – 2.50 Epidemiology of 2007 EI outbreak NSW Dr Barbara Moloney	2.30 – 3.00 Risk factors associated with sore backs in Pony Club Horses. Dr Petra Buckley.
			2.45-3.00 Prognosis for racing following laryngoplasty using the "Securos"suture. Hadley	2.50-3.10. Epidemiology of Qld EI outbreak Dr Nigel Perkins	

			Willsallen		
3.00	Afternoon Tea	Afternoon Tea	Afternoon Tea	Afternoon Tea	Afternoon Tea
3.30	Surgical management of Carpal flexural deformities in foals. Dr Jim Vasey	Advances in equine surgery Randwick Equine Clinic	3.30 Clinical Prize Presentation	3.30 – 3.50. Response of a non-infected state (Vic) to EI outbreak. Dr Roger Raskin	3.30 – 3.50 Tendonitis – fact, fiction (What I don't know). Dr Dick Wright
	Foaling percentages comparing one and three layer repair of cervical lacerations. Dr Jim Vasey			3.50 – 4.10. Epidemiology of the EI outbreak – National perspective. Dr Graeme Garner	3.50 – 4.30 What limits the responses of injury-prone tendons to training Assoc Prof Janet Patterson-Kane
				4.10 – 4.30. NZ response to the EI outbreak in Australia. Dr Naya Brangenberg.	4.30 – 4.50 Synovially-derived stem cells. Assoc Prof Matt Stewart
4.30	Surgical management of jugular vein sepsis. Dr Tom Russell	Advances in equine surgery TBA	Abdominal Surgery in Horses "Are we saving more?" Prof Tim Greet	4.30 – 4.50. Managing the risk of EI in horses from Aus – NZ. Dr Andrew McFadden	4.50-5.10 Analysis of osteochondrosis pathogenesis using molecular methods. Dr Eleanor Mackie
				4.50 Panel discussion	5.10- 5.30 Open Discussion
				5.10 – 5.30. Panel discussion.	5.30 Close
5.30	Close	Close	Close	Close	
7.00	BBQ Patio Room	Cocktail Part Q! Tower	Free Evening	College Dinner	

CONTENTS PAGE

These are printed abstracts from the conference. These and other extended abstracts are available to download from the College Website www.acvs.org.au

THE ROLE OF LEPTIN IN REPRODUCTION AND FERTILITY

JIM MCFARLANE, KATE KAUTER AND SHALINI PANWAR

Centre for Bioactive Discovery in Health and Aging, School of Science and Technology, University of New England, Armidale NSW 2351

Leptin was originally identified as a peripheral satiety signal made by fat cells and it was proposed that as fat accumulates leptin is secreted proportionally. Leptin then crosses the blood brain barrier and signals to the hypothalamus suppressing appetite and increasing energy expenditure as it rises. It is evident that if the leptin gene or its receptor is disrupted them morbid obesity occurs. However, obesity in humans is very rarely caused by a disruption of the leptin gene or its receptor, and in many species a good correlation exists between leptin concentrations in plasma with the degree of obesity. This early finding was unexpected and led to the leptin resistance hypothesis. Further studies have revealed that leptin actually appears to be almost ubiquitously expressed in many tissues. Unfortunately despite the accumulation of a vast literature leptin still appears to be regarded as a peripheral satiety signal although the data to support this hypothesis is not substantial.

There is increasing evidence that, in addition to its action on food intake and energy expenditure, leptin plays an important role in reproduction and development (Cervero *et el.,* 2005). The *ob/ob* mouse is deficient in leptin and is obese and infertile. Fertility can be restored in both female and male *ob/ob* mice by the exogenous provision of leptin, which is characterized by an increase in basal LH and FSH (Mounzih *et al*., 1997). During the menstrual cycle, there are variations in serum leptin levels, with higher concentration reported in the preovulatory and midluteal phases and lower in the early follicular phase (Henson and Castracane, 2005). Indeed, the *ob/ob* mouse exhibits numerous reproductive abnormalities including unusually small ovarian weight in females and a small amount of interstitial tissue in males (Jones & Ainsworth-Harrisson, 1957). Treatment of *ob/ob* mice with leptin results in a normalization of testicular and ovarian weight and function (Mounzih *et al*., 1997), thus suggesting that leptin may be required for normal growth and development of reproductive organs. The restoration of fertility to the female *ob/ob* mouse by the exogenous administration of leptin has demonstrated that leptin is essential for normal preimplantation and/or implantation processes (Malik *et al*., 2001). We and others have reported a beneficial effect of low physiological doses of leptin on early embryo development in culture and that higher doses impeded development. Leptin would also appear to be important during implantation and early pregnancy (Malik et al 2001; Henson and Castracane, 2005)

Consequently in the complete absence of leptin / leptin signalling the reproductive system is seriously impaired. However despite considerable research it remains very unclear as to whether a distinction can be made between central and peripheral actions of leptin and indeed the exact role leptin plays in the reproductive axis. The aim of the following experiment was to investigate the effect of leptin withdrawal in the peripheral circulation using active and passive vaccination on reproductive performance in the mouse and sheep. Therefore in the present study, we looked at the effect passively immunizing mice against leptin on ovarian weight. We also examined the effect of neutralizing leptin on early embryo development and implantation in mice.

The purified polyclonal antibody JMCK#43 has previously been shown to increase food intake in rats when administered centrally. In the first experiment, prepubutal mice were randomly divided into 4 treatment groups of 15 animals; antibody (50ug) with or without PMSG (1iu), and non immune antibody (50ug) with or without PMSG (1iu). The mice received daily injections (IM) for 4 days and were killed on the fifth day and the ovaries and uterus dissected out and weighed. The results of this experiment showed that the paired ovarian weight (12.0 ± 0.5 mg) in those mice treated with the antileptin Ig were significantly heavier than those treated with non immune Ig (9.8 ± 0.6mg). PMSG significantly increased ovarian weight (15.9 ± 0.7mg) over both the control and antileptin treated animals. Interestingly, the combination of antileptin and PMSG significantly increased ovarian weight (21.3 ± 0.8) over all other treatment groups. In the 2nd experiment adult mice were divided into 2 groups of 10 adult female mice and antibody (50ug) or non immune antibody injections were given daily for 7 days after mating. At birth the pups were counted and the weight of the pups and mothers at weaning (3 weeks) was recorded. The number of pups born to the mice which had been injected with antileptin (16.4 ± 0.5) was significantly greater that those treated with the non immune Ig (13.1 ± 0.4). Although pups born to the antileptin treated mothers was heaver (12.1 ± 1.6 g) at weaning than those born to untreated mothers (10.3 ± 1.8) this not significant.

In a longer term study a group of merino ewes were vaccinated against leptin six months before the breeding season. The ewes were kept in open paddocks and only supplied supplemental feed as required. Unexpectedly vaccinated ewes did not become obese or infertile but rather over the two year experimental period pregnancy rate was 100% while in controls pregnancy rate was 80%. More interestingly the lambing rate (at weaning) was significantly higher in the vaccinated ewes (150%) compared to the control ewes (102%).

While it is clear that in the complete absence of leptin the reproductive systems does not function. The data from the above study suggests that peripheral leptin may act as an inhibitor of both follicular development in the ovary and of early embryo development or implantation. It is interesting to speculate that leptin may have a permissive role when acting centrally and an inhibitory role in the reproductive tract, thus acting as a responder to over nutrition rather than under nutrition. Thus it would appear that reducing peripheral leptin enhances reproduction in both mono and poly ovulatory species. Further work is underway to investigate this in other species including the horse.

References

Cervero A, Horcajadas JA, Dominguez F, Pellicer A and Simon C (2005) Leptin system in embryo development and implantation: a protein in search of a function. *Reproductive Biomedicine Online* **10:** 217-223

Henson MC and Castracane VD (2005) Leptin in Pregnancy: An Update. *Biology of Reproduction* **105:** 45-120

Jones N and Ainsworth-Harrisson G (1957) Genetically determined obesity and sterility in the mouse *Study of Fertility* **9:** 51-64

Malik NM, Carter ND, Murray JF, Scaramuzzi RJ, Wilson CA and Stock MJ (2001) Leptin requirement for conception, implantation, and gestation in the mouse *Endocrinology* **142**: 5198-5202

Mounzih K, Lu RH and Chehab FF (1997) Leptin treatment rescues the sterility of genetically obese ob/ob males *Endocrinology* **138:** 1190-1193