594 Stress-induced intestinal barrier dysfunction and its effects. G. P. Lambert*, *Creighton University*.

The intestinal barrier is formed by enterocyte membranes, tight junctions, secreted mucus, and immunologic factors like tissue macrophages. Dysfunction of this barrier can be caused by different types of stress (e.g., physiological, pathological, psychological, pharmacological) and can lead to increased intestinal permeability. Increased permeability to endotoxin, a component of the walls of Gram-negative bacteria, causes local and/or systemic inflammatory reactions. The immune response(s) can then promote more serious conditions. Exertional heat stroke is an example of such a condition. During severe exercise-heat stress, possibly combined with other stresses, reductions in intestinal blood flow and/or direct thermal damage to the intestinal mucosa can cause intestinal barrier disruption and endotoxemia. The resulting inflammatory response is believed to be involved in altered thermoregulation and multiple-organ dysfunction. Possible means for preventing and/or attenuating many stress-induced intestinal barrier problems include environmental, pharmaceutical, and/or nutritional approaches.

Key Words: Intestine, Heat, Stress

595 Dietary plasma proteins and the barrier functions of the intestinal mucosa. M. Moretó* and A. Pérez-Bosque, *Universitat de Barcelona*, *Barcelona, Spain*.

The intestinal mucosa contributes to homeostasis by preventing the passage of biological and chemical agents across the epithelium, which could alter the stability of the system. This protective function is important at weaning, when animals are exposed to infectious agents and to stresses such as changes in diet composition. Diets supplemented with spray dried plasma or immunoglobulin concentrates improve growth and performance of farm animals and have been proposed as an alternative to antibiotics. We summarize our findings on the mechanism of action of dietary plasma proteins. We used a rat model of intestinal inflammation based on the administration of Staphylococcus aureus enterotoxin B (SEB). SEB activates the gut-associated lymphoid tissue (GALT), increasing T-lymphocytes in Peyer's patches and the number of activated T lymphocytes in mesenteric lymph nodes (organized GALT). In the lamina propria SEB increased cytotoxic Tγδ and NK cells populations of the diffuse GALT. SEB significantly increased IFN-γ, TNF-α, IL-6 and LTB4 concentration in both Peyer′s patches and mucosa. Plasma protein supplements modulated the mucosal immune response in both organized and diffuse GALT, protecting GALT from excessive activation by the SEB challenge. These effects were accompanied by a reduction of pro-inflammatory cytokine production and increased expression of IL-10 in the mucosa, supporting the view that changes in cytokine production mediate the effects of dietary plasma proteins during intestinal inflammation. The increase in mucosal permeability and intestinal secretion induced by SEB was associated with decreased expression of mucosal tight-junction and adherent-junction proteins. Both plasma and immunoglobulin supplements could prevent the effects of SEB on intestinal permeability, thus reducing the probability of microbial and food antigens entering the interstitial space. These findings indicate that dietary plasma proteins modulate both functional and structural properties of the intestinal mucosa.

Key Words: Barrier function, Intestinal inflammation, Spray-dried animal plasma

596 Strategies to minimize inflammatory taxation on animal performance. M. E. Cook*, *University of Wisconsin*, *Madison*.

Collateral damage of inflammatory activation is measured as decreased gain, product production, reproduction, feed efficiency, and increased mortality. A decline in efficient animal performance (>10%) associated with microbial colonization or after vaccination of an animal suggests that current management systems fail to realize the animal's genetic potential for performance. The mechanisms by which pro-inflammatory cytokines redirect nutrient use at the expense of performance have been extensively studied. At least three strategies are available for improving efficient performance of the conventional (as opposed to germ-free) animal: 1. Minimize exposure to environmental immune stimulants: 2. Suppress the immune/inflammatory response: and/or 3. Erect barriers against inflammation-induced collateral damage. The success of a strategy employed may be case dependent, but is likely additive when used in combination. Traditional and successful methods to minimize immune stimulants and hence inflammation of the epithelial surfaces have included the use of antibiotics, pre- or pro-biotics and sanitation. Suppression of the inflammatory response, while seemingly problematic, has long been a pharmacological approach in human medicine. Since the domestic animal maintains the immune capacity of its wild ancestor, but is housed in an environment where diseases are well managed, an acute inflammatory reaction, even to benign agents, is often counterproductive. Barriers to the collateral damage caused by inflammatory responses constitute well-defined and novel approaches to improve efficient animal performance. Barriers erected are designed to target key biochemical or physiological responses to pro-inflammatory mediators in an attempt to assure resources are maintained for efficient animal performance. In this latter approach, neither the animal's environment nor its inflammatory response to the environment is the focus of improving efficient animal performance. Barrier examples to be presented include orally delivered egg antibodies to key targets in the gastrointestinal tract and conjugated linoleic acids that systemically reduce the collateral damage of inflammatory responses.

Key Words: Inflammation, Immunity, Growth