


DR. STEVE MARSDEN'S



ESSENTIAL
GUIDE TO
CHINESE
HERBAL
FORMULAS



*Bridging Science and
Tradition in Integrative
Veterinary Medicine*

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SECTION I

THE SAFE PRACTICE
OF VETERINARY
HERBAL MEDICINE

Drugs of the Future: Herbal Formulas in Context, Part I

Introduction

Research into herbal medicine is increasing logarithmically in an effort to identify new pharmaceuticals with enhanced effects relative to the drugs that are relied upon today. The constituents of interest currently occur only in plants, at too low concentrations for pharmacological action. Inside the plant and, by extension inside the herbal medicine patient, the constituents are only one voice among many that together 'sing the song' of the plant's medical influence.

Besides being more concentrated, drugs of the future hoping to preserve and accentuate the benefits of these ingredients will likely have to contain more than one of them. Barbershop quartets will have to be identified within the choir that are singing the song loudest and most clearly. Such drugs do not yet exist. For the moment, veterinarians can access them only in plant form.

However, the effects are worth the effort of learning a new medical discipline. They include:

- ❑ Increasing blood flow to particular tissues and organs
- ❑ Targeted anti-inflammatory effects that do not affect the whole system
- ❑ Insulin sensitization
- ❑ Resolution of chronic inflammation by reversal of endothelial dysfunction
- ❑ Halting and preventing degeneration through targeted increases in blood flow
- ❑ Normalization of smooth muscle contraction
- ❑ True immune modulation, where immunity is strengthened while destructive inflammatory responses are subdued
- ❑ Novel antimicrobials with unlimited distribution and minimal resistance
- ❑ Promotion of cellular differentiation and control of angiogenesis to induce tumor atrophy

These are just a few of the tantalizing medical possibilities arising from integrating herbs into medical practice. The goal of this chapter is to review these possibilities in some more detail and to provide a physiological context by which the reader can easily gain familiarity with the new treatments.

This chapter by no means constitutes an exhaustive list of what all formulas in this text can accomplish for that the reader is referred to the individual monographs making up the bulk of the text. However, it is hoped the reader will gain from the chapter new possibilities to manage existing patients. As yet, many veterinarians will be unaware that one of the most valuable impacts an herb can have in the dog or cat is the induction of insulin sensitivity.

Insulin resistance: its role in acute inflammation

Insulin resistance is of massive importance in small animal medicine, even in non-diabetic animals. Research is emerging to suggest insulin resistance is a prominent cause of acute, recurrent and chronic inflammation in small animals.

Post-prandial hyperglycemia

Cats and dogs share a very similar metabolism to humans, such that they frequently serve as animal models for research into human metabolism and its role in inflammation. This research has disclosed that consumption of highly processed, starch-based or high-fat diets causes the appearance of a transient post-prandial inflammatory response (Margioris 2009). Although the biologic value of such a response is unclear, there is no question it occurs. It is more severe in patients with a higher body score and is mediated by potent pro-inflammatory cytokines such as interleukin-6.

Once elaborated, cytokines do not just produce inflammation but also promote generation of reactive oxygen species. C reactive protein (CRP) is one example. It is a phylogenetically old defense molecule utilized by many animal species to help increase the inflammatory response (Solter and Uhlenbruck 1982). CRP synthesis is induced in the liver by cytokines released in response to diet and has the effect of ramping up any inflammatory responses by promoting complement fixation and opsonization.

Border skirmishes on epithelial surfaces triggered by mild irritants or a few microbes suddenly have the potential to become much more incendiary and destructive under the influence of a processed starchy or high-fat diet. Multiple inflammatory episodes occurring over time at various epithelial surfaces are the hallmark of this post-prandial inflammatory tendency. This is commonly noted in a small animal patient's medical record as a procession of '-itides'. These episodes respond to antimicrobial therapy, although the number of microflora present may not be different from a healthy animal. The difference in response is from a heightened reactivity to their presence.

Just as commonly, however, inflammation remains subclinical, quietly damaging insensate tissues like the kidney and liver. Problems are only recognized on routine blood and urine screening, perhaps once functional reserves have been consumed. Examples include elevated liver enzymes, azotemia, hematuria and crystalluria.

Post-prandial oxidative stress

Post-prandial increases in inflammation lead to oxidative stress, but so too does the meal itself as fats and starch are metabolized by liver mitochondria. If antioxidant levels are insufficient in a diet to quench them (as is typical in canned and dry foods), these reactive oxygen species provoke tissue damage that spawns more inflammation. Inflammation, in turn, adds to any oxidative stress in the body, for instance through the heightened activity of cyclo-oxygenase and lipoxygenase. In the end, a vicious cycle of inflammation and oxidative stress is created and perpetuated with each meal.

Development of insulin resistance

Sustained inflammation and oxidative stress eventually can damage beta islet cells plus insulin receptor and signalling mechanisms. Obesity and insulin resistance commonly ensue.

Although it is uncommon for insulin resistance to result in diabetes, it is of tremendous clinical significance. Insulin resistance impairs gluconeogenesis, promoting heightened food consumption that in turn propagates more inflammation and oxidative stress. It also allows post-prandial hyperglycemia to endure, heightening its inflammatory consequences.

Insulin resistance paves the way for other sequelae besides inflammation and obesity. A pre-cushingoid state can be produced where glucocorticoid receptors increase in number or are otherwise up-regulated, allowing normal cortisol levels to have a heightened effect. This syndrome is common in dogs, producing symptoms of hyperadrenocorticism with no diagnostic increase in serum cortisol.

The pro-inflammatory effect of abdominal fat

Problems deepen further as abdominal fat accumulates in response to insulin resistance. Adipose tissue, previously considered largely inert, is in fact a separate major source of inflammatory cytokines. The evolutionary advantage behind adipose tissue generating significant amounts of inflammatory mediators is not clear, but it is able to be demonstrated consistently in a variety of species. If fat stores are large, the release of cytokines is large. Not surprisingly, weight loss is associated with reversal of tendencies toward accumulation of inflammatory mediators (Bastard et al 2006). Unfortunately, weight loss is progressively more difficult as insulin resistance worsens. In addition, post-prandial inflammatory responses worsen in the obese patient and are more easily produced regardless of the nutrients consumed.

Therapeutic intervention through diet

Satisfactory results in managing inflammation are unlikely to be obtained without addressing an animal's diet. To date, veterinarians focus on specific immune responses to individual food antigens as the leading cause of diet-induced inflammation. Clinic shelves are lined with food containing novel proteins from increasingly creative sources in an effort to address an apparent epidemic of food allergies and sensitivities. In contrast to this perception, clinical experience and research suggest that primary food allergies are relatively uncommon.

Acquisition of food allergies appears driven by chronic inflammation of the digestive tract, leading to a so-called 'leaky gut' that allows larger-sized food molecules premature entry to the lamina propria where they prime the immune system to respond to them. However, gut wall inflammation arises from inflammatory responses that are non-specific and driven by post-prandial hyperglycemia. Elimination of these inflammatory responses through use of an unprocessed, non-starch-based diet allows the gut wall to heal itself, thus eliminating the possibility of food allergies. Non-specific inflammatory responses trump food allergies as the main diet-related problem in dogs and cats.

Current research suggests the ideal dog or cat diet has a low glycemic index, such that surges in post-prandial insulin, inflammation and oxidation are minimized. Additionally, they should be an important source of anti-oxidants. "When measured by these criteria, most commercial diets fail on all accounts, even so-called hypoallergenic diets.

Despite their nutritional completeness, research evidence is emerging that points to most canned and kibble diets as being potential promoters of disease. In response, the public has reached for home-cooked and raw diets for their pets, much to the chagrin of veterinarians. The veterinary profession has likely been too quick to judge, however. "When compared with canned and kibbled diets, home-cooked and raw diets' greater complexity, higher antioxidant levels and longer digestion times undoubtedly lead to reductions in insulin resistance, oxidation and inflammation.

Regardless of whether relying on drugs or herbs to treat patients, a primary goal of the small animal veterinarian must include providing the owner with advice and resources on the creation of nutritionally complete, unprocessed diets.

Given the foregoing, the ideal anti-inflammatory drug should be:

- ❑ Antioxidant
- ❑ Insulin sensitizing
- ❑ Anti-inflammatory