ADDITIVES IN POULTRY NUTRITION: PROBIOTICS, PREBIOTICS, AND TOXIN BINDERS. PRACTICAL ASPECTS

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1. OVERVIEW

Removal of sub-therapeutic antibiotics from poultry diets in Europe and recent pressure to reduce or removal of these compounds in other parts of the world has amplified corporate and academic interest in improving intestinal health, improving nutrient utilization, and reducing endogenous nutrient loss due, in part, to innate immune responses. Probiotic (direct-fed microbials) and pre-biotic (undigested carbohydrates, e.g. β-glucans or fructo-oligosaccharides), organic acids, short/medium chain fatty acids, and plant extract feed additives are gaining market presence. Hesitation by nutritionists to incorporate these categories feed additives are due in part to a) unfamiliarity, b) overselling of plausible effects, c) documented physiological and microbiological effects in vivo, and d) documentation of persistence from the feed and within the intestinal tract. For review, please see Applegate et al. (2010). Different feed additives and nutritional strategies have shown promise in changing and mediating the response and recovery of the bird to various stressors and challenges and thus influencing flock performance and uniformity. Better understanding and applications of these strategies is imperative in order to reduce the variability in maintenance and/or feed efficiencies.

As we look to the recent focus of research, it solely has focused on the initial response of the bird to particular challenge and/or acquired immune capacity. Largely, it has lacked the dynamics of the responsiveness required to fully grasp the productive “cost” of the response, encompassing immunological reaction, reduction and recovery of food
intake, as well as restoration and healing of damaged tissue. Thus, we do not have a full vision of how the aforementioned products can play in the course of response, pathogen clearance, and tissue repair/recovery. Recent work from our lab also suggests that recovery from an early stressor is different between commercial broiler strains, such that partial compensatory growth is plausible in some, but not all strains.

The gastro-intestinal tract (GIT) comprises a large nutrient and energy to support digestive and barrier functionality. The functions of the intestine are vast and become pliable to changing needs and demands. Stressors (temperature, pathogens, bacteria, toxins, etc.) to the intestine, increase the demands put on it to maintain barrier, digestive, and absorptive roles. For example, when exposed to a coccidial challenge, the energetic efficiency of the bird greatly declines. Recent research from our lab has also demonstrated an energetic and nutrient “cost” model utilizing sub-clinical (no visual lesions present) challenges with a coccidial vaccine. Responses of the animal to intestinal challenges, therefore, encompass a large loss, mainly due to nutrient and energy maintenance needs of the animal.

Beyond the intestine, other factors may be influencing the variability in bird response we see within and between flocks. For example, factors such as toxicological responses may influence the efficiency to which individual birds respond. One toxicological response, would be to that of mycotoxins. The predominate mycotoxins poultry are exposed to, aflatoxin (AF) is readily absorbed in the proximal GIT (greater than 80%), while ochratoxin (OTA) is moderately absorbed (40%), and deoxynivalenol (DON) and fumonisin (FUM) are minimally absorbed (5 to 20 and 1%, respectively; Grenier and Applegate, 2013b). Recent literature and results from our work began to implicate that the accumulative effects of low and routine concentrations of major mycotoxins, primarily DON, FUM, and AF, can lead to effects on intestinal tight junction functionality, active nutrient absorption, and pro-inflammatory cytokine markers. These responses particularly to DON and FUM explain research from different labs wherein coccidial lesion severity is increased and recovery is prolonged, and in other studies where necrotic enteritis lesions have developed (e.g. Antonissen et al., 2014; Grenier et al., 2016).

To cope with dietary mycotoxin contamination, the typical means has been to feed nutritionally inert sorbents. These adsorbent materials include silicates, activated carbons, complex carbohydrates and others. It is very well known that adsorbents are very effective to counteract the effects of aflatoxin (AF), especially clays (inorganic adsorbents). However, there are few reports of these adsorbents being able to bind and eliminate other mycotoxins in animals. The adsorption (and desorption in the GIT) is highly related to the physical structure (i.e., the total charge and charge distribution, the size of the pores and the accessible surface area) of the adsorbents, but also to the properties of the adsorbed molecules like polarity, solubility, size and shape of mycotoxins. These latter properties account for why some binders have little success in efficiently adsorbing other mycotoxins besides AF (Huwig et al., 2001; Kabak et al., 2006).
In summary, maintenance of the barrier functions of the GIT comes at a considerable price. Further work is required to identify conditions permitting us to fully maintain barrier functionality across the severity and virulence of challenges/stressors experienced while minimizing nutritional “cost” associated with maintenance of functions therein. Our knowledge gap has only partially been filled towards elucidating roles and modes of action of individual feed additives can play; however, we lack scientific approaches in many areas including that of combination of approaches and where they can be applied.

2.- REFERENCES


