Recently, there was a reinvigoration of the concept of “gut health.” Field practitioners often correlate bad production outcomes, including diminished health and reduced growth efficiency, with poor gut architecture, as well as gut microbiota proliferation. This interference in production by the gut environment is surprising because the gut has long been considered a poorly refined organ solely equipped for the digestion and absorption of nutrients, with more noble functions, including homeostasis, resistance to disease, and growth efficacy, being performed by other internal organs. Interestingly, other biological disciplines have made quantum leaps in this field and have shown that the gut acts as an intelligent sensory organ in permanent communication with its continuously changing environment and that the gut is capable of substantial adaptation in response to changes in its internal environment. This begets the question of how we can make progress in the field of animal production and nutrition if we do not understand the molecular mechanisms driving the response of the gut to the diet, which seems to govern, more than we previously realized, the productive response of the animal in terms of growth, efficiency, and health.

Comparative physiology is an interesting approach to extrapolate findings from 1 species and apply it to another, especially at the molecular and cellular levels and even more in the postgenomic era. To help facilitate this process, the goal of the first Comparative Gut Physiology Symposium at the 2014 Joint Annual Meeting in Kansas City, MO, was to expose animal nutritionists to the current molecular and cellular discoveries that have been made in gut biology across animal species. The talks explored 2 methods of triggering an improvement in the productive response of farm animals using both 1) the gut epithelium as a specialized translator of messages coming from the lumen and 2) the interplay between the diet, the gut microbial ecosystem, and the immune response.

Furness et al. (2015) presented the specialized gut epithelial cells (i.e., enteroendocrine cells), which translate the complex gut lumenal messages directly or indirectly in response to the diet into a language that the body can understand. These cells are functionally connected by networks that magnify the messages and trigger a local response or activate the response of a distal organ. Additionally, they pointed out the plasticity of the gut and, particularly, its adaptability in relation to the diet with, for example, the induction of enzymes and transporters and growth of the mucosa. This knowledge was gained through studies in humans and rodents, thus allowing the authors to emphasize an interesting viewpoint. Although the appearance of the buildings may be different at the level of the organization of the digestive system (e.g., among ruminants, nonruminant mammals, birds, and fish), the individual building blocks (e.g., at a tissue component, cellular, or molecular level) are most often shared across species. What’s more, when different species are exposed to the same compound, their physiological responses are very comparable. This result opens the possibility of extrapolating insights from heavily investigated animal models to those species for which the research is scarce. Then, the question of whether a certain physiological response is converted to an improvement in production could be investigated in the target animal. At the very least, this comparative approach could help in the design of new and innovative feeding practices.

Interestingly, Connor et al. (2015) exemplified the comparative physiology approach by presenting...
a gut hormone involved in the response of the gut to its environment: the glucagon-like peptide-2 (GLP-2). This hormone triggers intestinal crypt cell proliferation, reduces epithelial cell apoptosis, and decreases gut mucosal inflammation, thereby improving gut integrity. Using comparative aspects and insights from humans, rodent models, and pigs, they demonstrated the potential of GLP-2 in ruminants, especially during transition periods, such as weaning. Then, the search for the GLP-2 secretagogues, via a rational feeding strategy or additive technology, could constitute a new molecular way to counteract the negative aspects of weaning in modern farm practices. Furthermore, there are other gut peptides that drive the response of the gut itself or of other distal organs. An improved knowledge of what promotes their secretion could open new ways of designing diets that improve productivity for farm animals.

Not only does the gut sense and react to its environment, but it also hosts a complex microbial ecosystem with which the host lives in symbiosis and whose contribution goes beyond the gut. Considering the surface of exchange with its ecosystem (100 m² in an adult human), the gut also represents a major lymphoid organ. The interplay between the immune system, the diet, and the gut microbiota was discussed in several presentations. Two speakers gave complementary views on calves. McFadden (2014) explained that not only do specific colostrum-derived factors impact the physiology of the young animal, but they also have long-lasting effects evident in the adult animal. Knowing the underlying mechanisms triggering the secretion of these bioactive compounds in the mother could allow specific diets to be created to enhance the secretion of these bioactive factors, thereby inducing the beneficial response in the progeny. Griebel et al. (2014) also linked the composition of the calf gut microbiota with gene expression in the gut and the function of the mucosal immune system. Kau (2014), from the laboratory of the distinguished Professor Jeffrey I. Gordon at Washington University School of Medicine (St. Louis, MO), extended this observation of the influence of changes in the gut microbiota by demonstrating clear links between the composition of the gut microbiota and the phenotype of its host. They recovered IgA binding fecal microbes from humans affected by severe acute malnutrition. This microbiota reproduced the phenotype of barrier dysfunction and mortality when transplanted in germ-free mice. Furthermore, symptoms were mitigated when gut microbes from healthy mice, which were not bound by IgA, were administrated to the sick animals. The demonstration of the cause-consequence relationship between specific microbial communities and the phenotype of the host and the beneficial nature of bacterial transplants from healthy animals are intriguing. If this relationship could be explored in animal nutrition, it could possibly be the basis of a new generation of probiotics and could trigger the creation of specific diets with optimized composition that could affect certain bacterial populations. How a dietary ingredient affects gut microbial population was the topic of Wlodarska’s (2014) presentation. She demonstrated that the low dietary inclusion of a phytonutrient (i.e., eugenol) promoted the thickening of the inner mucus layer in the colon and stimulated the development of specific beneficial clostridia species, resulting in protection against enteric infection. This protective effect was not the consequence of a direct in situ killing effect of eugenol on the pathogen. This result shatters the traditional vision of using essential oils as direct-killing agents and could be the basis of an alternative approach whereby phytonutrients would trigger host-mediated responses. The host response to phytonutrients was further explored by Lillegard (2014), who reviewed several additive technologies that successfully interfere with the immune response of the host and protect poultry against enteric infections. As lesion formation is often the consequence of immune-mediated damage but not from the pathogen per se, she noted that the protective response is often correlated with a significant downregulation of local proinflammatory cytokines.

In conclusion, the first Comparative Gut Physiology Symposium exposed animal nutritionists to the current progress that has been made in gut cellular and molecular physiology across animal species. It advocated that insight gained in 1 animal model can be reasonably extrapolated to other species, but the final validation needs to be done in the target animal. Interestingly, it also sketched what could be the future direction in nutrition and feeding. Indeed, the feed given to farm animals is designed for the host, but it could also be designed to optimize the microbiota (as done in ruminants, for example). This optimization would utilize the fact that the enzymatic capacity of the gut microbiota is complementary to that of the host and would clearly exploit the essence of the microbiome. Moreover, the progress made in our understanding of gut health continues to shatter the dyadic vision of biology, with physiology on one hand and microbiology and immunology on the other. At the gut level, some gut hormones are anti-inflammatory substances or can directly communicate with the gut ecosystem; metabolites from the microbial ecosystem trigger host physiological responses in distal organs. Thus, the role of nutrition should not be to look at each single response individually but to integrate a vision of how the gut responds to the diet and, subsequently, how this response shapes the productive response of farm animals.
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