The symposium titled “Gut chemosensing: integrating nutrition, gut function, and metabolism in pigs” preceded the 12th International Symposium on Digestive Physiology of Pigs that was held in Keystone, CO, from May 29 to June 1, 2012. The objectives of the preconference symposium were to provide a comprehensive overview of the state of the art and thereby foster discussion and research aiming to elucidate the role that chemical (i.e., nutrient) sensing by the gastrointestinal (GI) tract plays on regulating gut integrity and body metabolism. The ability of the GI tract to sense ingested nutrients and other chemicals has been suspected since Bayliss and Starling (1902) first demonstrated that luminal protons stimulate pancreatic secretions through a neurally independent mechanism. Almost 100 yr later, the discovery of the expression of taste signaling elements in cells interspersed in the gastric and intestinal mucosa supported the hypothesis that GI chemosensing is a cell-mediated phenomenon (Höfer et al., 1996). In recent years, scientific efforts have increased remarkably to gain a closer understanding of the GI chemosensory system and its involvement in the normal and pathological functioning of the gut and body metabolism. As a result, a number of nutrient-activated guanosine-protein coupled receptors (GPCR) have been de-orphanized and shown to be expressed in different segments of the GI tract. Subsequent functional studies revealed that these putative chemosensors are localized in hormone-secreting enteroendocrine cells that function along with visceral afferent neurons as the interface between the GI lumen and local or distant targets. In this fashion, enteroendocrine cells appear to operate as chemosensory transducers that respond to ingested nutrients and other chemicals by releasing regulatory peptides to initiate humoral and vagal signaling cascades that convey information to the brain and neighbor cells regarding the luminal milieu. As a consequence, a wide array of physiological responses, ranging from stimulation of gastric, intestinal, and pancreatic secretions to inhibition or stimulation of appetite and feed intake, are triggered. For these reasons, the relationship between GI chemosensing, gut integrity, and body metabolism in pigs emerges as an interesting subject of functional importance.

The symposium program consisted of invited speakers from outside, related disciplines that brought a unique view to the context of the presented science including implications for swine species. As the first speaker of the symposium, A. P. Liou (Massachusetts General Hospital, Charlestown, MA) provided an overview of the complex biology of enteroendocrine cells, focusing on the ability of different cell subtypes to directly sense lipids, proteins, and carbohydrates through GPCR-mediated mechanisms that are linked to the release of GI hormones (Liou, 2013). Reviewed studies evidenced that the scarcity and scattered distribution of endocrine cells in the GI mucosa and the coexistence of direct (i.e., via endocrine, cell-surface GPCR) and indirect (i.e., via nonendocrine, neighbor enterocytes) chemosensing pathways are limiting our capacity to clarify the mechanistic functioning of enteroendocrine cells. For these reasons, experimental approaches to address the contribution of enteroendocrine cells to GI chemosensation were proposed and briefly discussed.

At the present time, most of the scientific evidence on nutrient sensing has been garnered for glucose and glutamate (Glu). The second speaker of the symposium, K. Torii (Institute of Innovation,