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The influence of diet, bacteria and bacterial metabolites on epithelial cell responses in intestinal inflammation and cancer

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Westernized diet, defined by high contents of saturated fats and sucrose, is associated with the development of several diseases including metabolic syndrome, obesity and cancer. Intestinal inflammatory responses are mediated by a complex crosstalk between the environment, microbiota and the immune system. Alterations in any of these systems can lead to development of gastrointestinal conditions such as inflammatory bowel diseases (IBD). Triggering factors for IBD and colitis-associated cancer (CAC) include environmental factors (e.g. stress), gut microbiota composition and diet. Recent reports indicate that a specific pathobiont outgrowth in IL-10-/- mice fed with a milk derived fat diet aggravated colitis. Epidemiological data have also identified processed meats and saturated fat as risk factors for IBD and colon cancer. Prebiotics are selectively fermentable ingredients that can change the composition and/or activity of the intestinal microbiota, which can lead to beneficial effects on the host. Short chain fatty acids (SCFAs) are the fermentation products of prebiotic digestion by the colonic commensal microbiota, with the most abundant SCFAs being butyrate, acetate and propionate. SCFAs, especially butyrate, act as source of energy for epithelial cells as well as being immune modulatory and helping dampening inflammation. In this talk, I'll discuss our findings emanating from *in vitro* and *in vivo* studies investigating the effect of diets, bacteria and bacterial metabolites on intestinal epithelial cell responses and in experimental models of colitis and CAC. To date, we have data showing that high fat diets can positively and negatively affect the outcome of colitis and CAC by regulating the microbiota, microbial metabolites and host epithelial and immune responses. In addition, we have generated mechanistic insights on the role of individual SCFAs and IBD-associated pathobionts such as adherent and invasive Escherichia coli (AIEC) on intestinal epithelial cell responses.

Recent Publications:

1. Nilaweera K N et al. (2017) Whey protein-effects on energy balance link the intestinal mechanisms of energy absorption with adiposity and hypothalamic neuropeptide gene expression. American Journal of Physiology Endocrinology and Metabolism. 313(1):E1-E11.
2. Russell S E et al. (2016) IL-36α expression is elevated in ulcerative colitis and promotes colonic inflammation. Mucosal Immunol. 9(5):1193-1204.
3. Yang S et al. (2013) Pellino3 ubiquitinates RIP2 and mediates NOD2-induced signaling and protective effects in colitis. Nature Immunology. 14(9):927-936.
4. Hall L J et al. (2013) Natural killer cells protect mice from DSS-induced colitis by regulating neutrophil function via the NKG2A receptor. Mucosal Immunology. 6(5):1016-1026.
5. McCarthy J et al. (2013) Gene silencing of TNF-α in an murine colitis model using a modified amphiphilic cyclodextrin delivery vector. Journal of Control Release. 168(1):28-34.

Biography

Silvia Melgar received her PhD in Immunology from Umeå University in Sweden, followed by a Postdoctoral Fellowship and a Senior Research Scientist position at AstraZeneca R&D Mölndal, Sweden. She is currently an APC Faculty Investigator under the Host - Microbe Dialogue Research theme in the APC Microbiome Ireland Research Institute of University College Cork (UCC), Ireland. She joined the APC in 2008 as a Principal Scientist under the GlaxoSmithKline-APC collaboration and became an Investigator in 2012. Her research interests include 1) the identification of novel molecular mechanisms associated to diet - host - bacteria interactions and their relevance to health and intestinal disorders such as inflammatory bowel disease (IBD) and colorectal cancer; 2) identification and pre-clinical evaluation of novel therapies for inflammatory and malignant conditions in the gastrointestinal tract in animal models and in *in vitro* cell systems.

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