The gastrointestinal (GI) tract is home to a large number and vast array of bacteria that play an important role in nutrition, immune system development and host defense. In inflammatory bowel disease (IBD) there is a breakdown in this mutualistic relationship resulting in aberrant inflammatory responses to intestinal bacteria. Studies in model systems indicate that intestinal homeostasis is an active process involving a delicate balance between effector and immune suppressive pathways. The cytokine IL-23 plays a pivotal role in orchestrating intestinal inflammation and several genes in the IL-23/Th17 pathway confer risk to IBD. We have recently shown that IL-23 acts directly on T cells to promote pathological Th17 type responses at the expense of immune suppressive regulatory T cells. In addition IL-23 drives a novel population of RORγt-dependent innate lymphoid cells (ILC) that mediate colitis through the production of Th17 associated cytokines. In this presentation I will discuss the multiple pathways through which IL-23 promotes tissue inflammatory responses in some cases leading to tumorigenesis.