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Immune Landscape in Chronic Inflammation: Complications and Risks

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Abstract

Controlled inflammatory responses are essential for restoring homeostasis following an insult. However, unresolved stimuli or dysregulated responses can lead to chronic inflammation, resulting in profound alterations in hematopoiesis, particularly within the myeloid lineage (myelopoiesis). These disruptions destabilize immune balance, triggering systemic shifts in immune cell phenotypes and functions that affect both local inflammation sites and peripheral tissues, with significant implications for disease progression. Our studies explore how chronic inflammation reshapes immune functions and disrupts niches of the bone marrow, gut, and peripheral tissues, creating harmful environments that drive complications. In the gut, chronic inflammation during inflammatory bowel disease (IBD), such as ulcerative colitis, compromises the epithelial barrier and facilitates dysbiotic bacterial invasion into the tissue. This process, along with the recruitment of immune cells such as myeloid-derived suppressor cells (MDSCs) to the intestine, disrupts homeostasis and heightens the risk of colitis-associated colorectal cancer (CAC). Intriguingly, the intricate interactions between MDSCs and dysbiotic microbiota and their outcomes in CAC remain poorly understood. Using a murine model of CAC, we demonstrate that CAC-bearing mice exhibit significantly elevated levels of highly immunosuppressive MDSCs alongside microbiota alterations. Within the tumor microenvironment, recruited MDSCs and invading bacteria localize in close proximity, revealing a complex MDSC-microbiota crosstalk. Our findings show that bacteria directly interact with and engulfed by MDSCs, enhancing their suppressive activities and inhibiting their differentiation into mature, beneficial myeloid cells, further impairing immune responses.
Our findings underscore the plasticity of MDSCs and their pivotal role in disease progression, highlighting MDSC-microbiota interactions as critical drivers of immune dysregulation and tumor growth in CAC. By unraveling these mechanisms, we provide new insights into potential therapeutic strategies targeting MDSC-microbiota crosstalk to mitigate inflammation-driven cancer progression.