Mechanisms of action of Plasmodium infection against cancer

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Video Byte

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Abstract

In cancer, tumor cells secrete chemicals that suppress immune function by upregulating the expression of immune “brakes”. Immunotherapy with checkpoint inhibitors can release these brakes to effectively treat certain types of cancer. However, other types of cancer are resistant to checkpoint inhibitors, so alternate treatments are needed. In mouse models, infection with the malaria parasite *Plasmodium* can activate immune defense against cancer. Similarly, global human epidemiological data indicate that malaria occurrence is inversely associated with cancer mortality. In mice, *Plasmodium* induces proinflammatory molecule production, immune cell activation, and subsequent systemic immune responses while simultaneously upregulating the expression of brake molecules through a feedback mechanism of the immune system to prevent unchecked damage by these immune responses. In contrast to the targeted effects of checkpoint inhibitors, the wide-ranging effects of *Plasmodium* infection make it a suitable “ecological counterattack” therapy for cancer, which is an “ecological disease” involving both tumor cells and their environments. To ensure safety in patients, a certain stage and strain of parasite can be used, and antimalarial drugs can be administered. Clinical trials are currently underway in China, but additional studies are warranted to explore the mechanisms and value of this unique therapy.