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CONTENTS IN BRIEF

Research News

Cancer immunoediting

The immune system plays a crucial role in shaping cancer development. It is able to detect early lesions through immuno-surveillance and eliminate them. It is thought to maintain tumors in a state of equilibrium. Tumor cells with either reduced immunogenicity or with increased capacity to attenuate the immune response can escape the equilibrium and progress. This whole process is termed cancer immunoediting. Clear evidence for this state has been produced by Mark Smith et al. (Nature, 2007). The authors used C57BL/6 and 129/SvEv mouse colonies and injected with 3′-methylcholanthrene (MCA) to induce sarcoma. As the authors were only interested in the mice that did not develop progressively growing tumors, they eliminated from the study all mice that had expanding tumors by 200 days. Mice that had only small stable mass at the injection site were then treated with control Ig or a mixture of monoclonal antibodies to deplete $CD4^+$ and $CD8^+$ T cells and to neutralize IFN-γ. 60% of these mice developed progressively growing tumors whereas no tumors were seen in the control mice. Importantly, suppression of NK cell function did not induce tumor outgrowth, indicating that suppression of the adaptive immune response is influential. The authors used mice deficient in recombination-activating genes ($Rag1^{-/-}$ or $Rag2^{-/-}$ mice), which have innate but no adaptive immune response. When subjected to the same MCA and monoclonal antibody protocol, very few of these mice developed late-onset tumors. In mice that did develop late-onset tumors, the time to tumor outgrowth was prolonged compared with that of the tumors that grew out in wild-type mice treated with antibodies against $CD4^+$, $CD8^+$ and IFN-γ. The more rapid outgrowth of tumors in the wild-type immunosuppressed mice indicate that the tumors probably have fully transformed cells that can proliferate in the absence of the adaptive immune response. This was further supported by the fact that atypical fibroblast-like cells that grew out of the stable MCA-generated mass from the wild-type mice formed tumors when injected into $Rag2^{-/-}$ mice. However, some of the control wild-type mice developed late-onset sarcoma, indicating that these might arise from cells that had escaped equilibrium. In agreement with cancer immunoediting, cells explanted from these tumors should be able to evade the immune response in syngeneic mice, unlike those with small masses, which should be immunogenic. Indeed, most mice developed tumors when injected with cells from the late-onset sarcomas, whereas up to 51% of the cells from the small masses were rejected. These results clearly indicate that the immune system can maintain tumors in a state of equilibrium and that cells escaping this process are more likely to evade the immune response.

Excerpt from Nature Reviews Immunology, January 2008