The Role of C/EBP-Homologous Protein (Chop) in Tumor-induced Tolerance

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Article

The Stress-Response Sensor Chop Regulates the Function and Accumulation of Myeloid-Derived Suppressor Cells in Tumors

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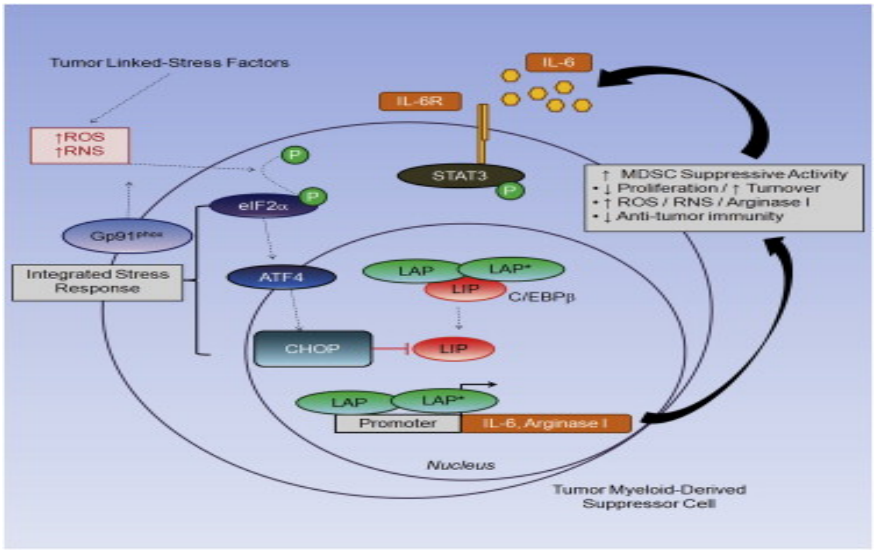
Background

Adaptation of malignant cells to the hostile milieu present in tumors is an important determinant of their survival and growth. However, the interaction between tumor-linked stress and antitumor immunity remains poorly characterized.
The study aimed to determine the role of the cellular stress sensor C/EBP-homologous protein (Chop) in the accumulation and immune inhibitory activity of tumor-infiltrating myeloid-derived suppressor cells (MDSCs).

Highlight

- Chop regulates immune-suppressive activity and accumulation of MDSCs in tumors
- Deletion of Chop in tumor stroma leads to effective antitumor T cell immunity
- Reactive oxygen and nitrogen species in tumors trigger Chop in MDSCs through Atf4
- Stromal cell Chop promotes MDSC activity through induction of C/EBPβ-IL-6 axis
- ***Findings suggest the role of Chop in tumor-induced tolerance and the therapeutic potential of targeting Chop in MDSCs for cancer immunotherapy***



Results show the checkpoints modulating the interaction between tumor-induced stress and MDSCs in the suppression of antitumor immunity and suggest targeting stromal Chop as a means to overcome tumor-induced tolerance and to enhance the efficacy of immunotherapy in cancer.