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IRF8: Effects on Health and the Mechanism of Action

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INTRODUCTION

A transcription factor belonging to the IRF protein family is called interferon regulatory factor 8 (IRF8). IRF8 was first discovered to be a crucial component in myeloid cell lineage commitment and differentiation. Irf8 deletion causes a significant build-up of CD-11b+Gr1+ immature myeloid cells (IMCs), especially the suppressor cell-like CD11b+Ly6Chi/+Ly6G Polymorphnuclear Myeloid Cells (PMN-MDSCs). Irf8 is repressed under pathological circumstances like cancer by its promoter DNA hyper-methylation, which causes mice to accumulate PMN-MDSCs and CD11b+ Ly6G+Ly6Clo monocytic MDSCs (M-MDSCs). In MDSCs from cancer patients in humans, IRF8 is frequently suppressed. The diverse populations of immune suppressive cells known as MDSCs reduce T and NK cell function to aid in tumour immune evasion and create growth factors to directly promote tumour growth.

DESCRIPTION

The New experimental evidence shows that IRF8 is expressed in non-hematopoietic cells as well. IRF8 is expressed by epithelial cells and controls apoptosis while suppressing osteopontin (OPN). In order to advance cancer through developing apoptosis resistance and up-regulation of OPN, human tumour cells may employ the IRF8 promoter DNA methylation as a strategy to inhibit IRF8 expression. In order to develop cancer, elevated OPN interacts with CD44 to inhibit T cell activation and support tumour cell stemness. Consequently, IRF8 is a transcription factor that controls both immunological and non-immune aspects of human health and disease.

The IRF transcription factor family includes the interferon regulatory factor 8 (IRF8), also known as interferon consensus sequence binding protein (ICSBP). IRFs were initially discovered to be a type I interferon (IFN-I) response regulator for the activation of IFN-stimulated genes necessary for immune response to viruses and other pathogens. Today, it is understood that IRFs also play a key role in translating pathogen-associated molecular patterns into chromatin changes, which in turn lead to the activation of immune cells. IRF8 was initially identified as a gene encoding an IFN-inducible nuclear protein that binds to a particular IFN-responsive DNA pattern in the MHC I genes. Since then, it has been discovered that IRF8 is constitutively expressed, IFN-inducible, and essential for the IFN response pathways.

Function to encourage immune evasion of colon tumours. In order to suppress OPN expression, IRF8 binds to the ISRE elements at the Spp1 promoter, which is expressed in human and animal gastric and colon epithelial cells. Human colon and pancreatic tumours. Model of the IRF8-OPN axis's mode of action in tumour promotion and immune suppression. IRF8 acts as a regulator, inhibiting Spp1 transcription in colon epithelial cells by binding to the two ISRE sites in the Spp1 promoter. Irf8 is repressed during the growth of tumours as a result of its promoter's DNA methylation and H3K9me3 build-up, which causes tumour cells, MDSCs, and ILCs to express more OPN [1-5].

CONCLUSION

OPN attaches to CD44 to prevent T cell activation as well as to CD44 on tumour cells to encourage the stemness and growth of tumour cells. Thus, the IRF8-OPN axis regulates tumour development and progression by acting on both immune and non-immune cell components.

More than 30 years ago, the transcription factor IRF8 was discovered to be a member of the IFN transcription factor family. The loss of IRF8 expression or activity results in an increase of CD-11b+Gr1+IMCs that phenotypically and functionally resemble tumor induced MDSCs. IRF8 is crucial for myelopoiesis. As a means of preventing haematological malignancies, IRF8 is also essential for immune cell homeostasis and turnover.

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