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Investigating molecular mechanism regulating *EGR2* expression and its role in the immune response

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The immune system is evolved to defend the body against pathogens and is composed of thousands of complicated and intertwined approaches, which are highly controlled by regulatory processes such as transcription and repression of cellular genes. Early growth response gene (EGR2) is important for maintaining immune stability and it has a vital role in controlling inflammation and preventing the development of autoimmune diseases. A recent study by our group demonstrated the function of *EGR2* as a checkpoint molecule controlling the proliferation and differentiation of the T cells, confirming its regulatory role, which is essential for optimal immune responses against pathogens; however the *EGR2* expression mechanism is still unclear. *EGR2* and *EGR3* play indispensable roles in T cell immune response, but its role in tumor regression is less well known. In this study, we have found that *EGR2* expression is regulated by antigens and cytokines, including INFγ and IL-6 which is mediated by INFγ/STAT1 and IL6/STAT3 signaling pathways. Furthermore, it is shown that *EGR2* can be significantly expressed in tumor infiltrating lymphocytes (TILs) as we observed in a mouse melanoma tumor model. Also, tumor growth is delayed in WT mice model in comparison with EGR2/3 KO counterpart, which is followed by higher TILs expansion in WT model. This was supported by IL-2 and Ki-67 significant expression in WT CD8+ TILs, suggesting the positive role of *EGR2* in CD8+ TILs expansion and tumor regression. Collectively, our results demonstrate that *EGR2* is an intrinsic regulator of adaptive immunity and tumor microenvironment.