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FcεRIγ is an immune-checkpoint of CD8+ T cell function during chronic virus infection

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During virus infection tight regulation of CD8+ T cell functions determine the outcome of disease. Recently others and we determined that regulatory NK cells kill hyper-proliferative CD8+ T cells. Molecules, which are involved in shaping the regulatory capacity of NK cells, remain virtually unknown. Herein we used mice, which lack the Fc-receptor common gamma chain (FcRγ, FcεRIg, Fcer1g –/– mice), to determine the role of Fc receptor and NK-receptor signaling in the process of CD8+ T cell regulation. We found that lack of FcRγ on NK cells limit their capacity to kill hyper-proliferative CD8+ T cells. In line, lack of FcRγ, in Fcer1g –/– mice, lead to enhanced CD8+ T cell responses and fast control of the chronic strain LCMV-Docile. Mechanistically FcεRIγ stabilized expression of NKp46, but not other killing activating receptors on NK cells. While FcεRIγ did not influence development or activation of NK cell during infection with LCMV, it specifically limited their ability to modulate CD8+ T cell functions. In conclusion, we determined that the major role of FcRγ during chronic LCMV infection is the limitation of hyper-proliferative CD8+ T cells.

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