Diet-induced obesity aggravates NK cell-mediated contact hypersensitivity reaction in *Rag1*$^{-/-}$ mice

Paulina Kowalczyk[1], Monika Majewska-Szczepanik[1], Anna Strzępa[1], Dominika Biała[1], and Marian Szczepanik[2]

[1]Uniwersytet Jagiellonski Wydzial Nauk o Zdrowiu
[2]Uniwersytet Jagielloński Wydział Nauk o Zdrowiu

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Abstract

BACKGROUND: Previous studies showed that liver NK cells mediate contact hypersensitivity (CHS) reaction in mice. There are many reports showing that obesity is accompanied with chronic low-grade inflammation and it promotes several inflammatory diseases. It was shown that diet-induced obesity (DIO) aggravates classical T cell-mediated CHS in C57BL/6 mice. Our work sheds light on a poorly explored subject of the influence of DIO on the course of NK cell-mediated CHS reaction in mice. METHODS: We evaluated the effect of DIO on NK cell-mediated CHS reaction using a model of dinitrofluorobenzene (DNFB)-induced CHS in *Rag1*$^{-/-}$ mice. RESULTS: Our data show that mice fed HFD for 8 but not for 4 weeks developed aggravated CHS reaction determined by ear swelling measurement when compared to animals kept on normal diet (ND) prior to DNFB sensitization. The obese *Rag1*$^{-/-}$ mice presented the adipose tissue inflammation. Furthermore, in vitro analysis showed that feeding with HFD significantly increases IFN-γ and IL-12p70 and decreases adiponectin concentration in liver mononuclear cells (LMNC) culture supernatants. The flow cytometry analysis of LMNC revealed that HFD treatment prior to DNFB sensitization increases the percentage of NK1.1^{+}IFN-γ^{+} cell population and affects development and maturation of NK1.1^{+} cells. CONCLUSION: In summary, current results suggest that the DIO significantly modulates the local and systemic inflammatory response, contributing to exacerbation of the CHS response mediated by liver NK cells.

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