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Immune System/ Bone/ Fat cross-talk: the role of LIGHT/ TNFSF14

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LIGHT/TNFS14 is a cytokine produced by immune cells. We demonstrated its role in regulating basal and pathological bone remodeling whereas other authors showed its pro-adipogenic role. Based on the "immune system/bone/fat" cross-talk, in this study we investigated whether LIGHT could be a new linker of this interaction. In bone marrow cell extracts of Tnfsf14-/- mice (KO), in which we proved the reduced trabecular bone, here we firstly detected a reduced expression of PPARγ, the key pro-adipogenic transcription factor. Consistently, we detected a lower weight of visceral and inguinal white adipose (iWAT) tissues respect to the WT mice, suggesting an impairment of adipocyte precursors in LIGHT deficient mice. Moreover, in the iWAT of these mice, we detected a lower number of brown adipocytes and lower mRNA levels of Wnt10b, involved in browning response, respect to WT mice, indicating that LIGHT-deficiency alters the adipose phenotype together with the bone one. These effects are mediated by immune cells, indeed, by using Rag-/Tnfsf14-mice lacking mature B/T-cells and LIGHT expression, the levels of PPARγ in bone marrow extracts and the number of brown adipocytes in iWAT are rescued respect to KO mice. These findings indicate LIGHT as new linker in immune system/bone/fat cross-talk and a potential target in obesity.