PPARα regulates endothelial progenitor cell maturation and myeloid lineage differentiation through a NADPH oxidase-dependent mechanism in mice

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Résumé en anglais	Peroxisome proliferator-activated receptor-alpha (PPARα) is a key modulator of lipid metabolism. Here, we propose that PPARα regulates the maturation and function of bone marrow (BM) progenitor cells. Although PPARα deletion increased the number of BM-resident cells and the differentiation of endothelial progenitor cells (EPCs) and monocytic progenitor cells, it impaired re-endothelization of injured carotid artery that was associated with reduced circulating EPCs. Also, PPARα deletion diminished the in vivo pro-angiogenic effect of PPARα agonist without affecting EPC differentiation markers. Macrophage colony-stimulating factor (M-CSF) treatment increased the population of monocytic progenitor cells as well as secretome of BM-derived cells in PPARα wild-type but not in knock-out mice. In addition, PPARα-null mice displayed reduced lymphocytes and increased monocytes and neutrophils in the blood. Furthermore, PPARα-null mice exhibited increments in the number of total cells (as well as of phenotypically distinct subpopulations of lymph node cells) but also a significant alteration in the number of various subpopulations of splenocytes and thymocytes. Finally, PPARα negatively regulated reactive oxygen species (ROS) derived by NADPH oxidase in BM-resident progenitor cells. Taken together, our data provide evidence that PPARα is a critical regulator of recruitment, homing and maturation of BM-derived progenitor cells.
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