Absence of CCL2 is sufficient to restore hippocampal neurogenesis following cranial irradiation

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Author(s) Lee, Star W; Haditsch, Ursula; Cord, Branden J; Guzman, Raphael; Kim, Soo Jeong; Boettcher, Chotima; Priller, Josef; Ormerod, Brandi K; Palmer, Theo D
Author(s) at UniBasel Guzman, Raphael ;
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Cranial irradiation for the treatment of brain tumors causes a delayed and progressive cognitive decline that is pronounced in young patients. Dysregulation of neural stem and progenitor cells is thought to contribute to these effects by altering early childhood brain development. Earlier work has shown that irradiation creates a chronic neuroinflammatory state that severely and selectively impairs postnatal and adult neurogenesis. Here we show that irradiation induces a transient non-classical cytokine response with selective upregulation of CCL2/monocyte chemoattractant protein-1 (MCP-1). Absence of CCL2 signaling in the hours after irradiation is alone sufficient to attenuate chronic microglia activation and allow the recovery of neurogenesis in the weeks following irradiation. This identifies CCL2 signaling as a potential clinical target for moderating the long-term defects in neural stem cell function following cranial radiation in children.
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