Prenatal Inflammation Disrupts Murine Foetal Hematopoietic Development and Alters Postnatal Immunity

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Abstract

Adult hematopoietic stem and progenitor cells (HSPCs) respond directly to inflammation and infection, changing their quiescence, mobilisation, and differentiation in both acute and chronic ways. We show that murine foetal HSPCs respond in utero to prenatal inflammation, and that this response shapes postnatal hematopoiesis and immune cell function. Divergent responses of heterogeneous foetal HSPCs to maternal immune activation (MIA) include changes in quiescence, expansion, and lineage-biased output [1]. In response to MIA, single-cell transcriptomic analysis of foetal HSPCs reveals specific upregulation of inflammatory gene profiles in discrete, transient hematopoietic stem cell (HSC) populations that propagate expansion of lymphoid-biased progenitors. MIA causes inappropriate postnatal expansion and persistence of foetal lymphoid-biased progenitors, as well as increased cellularity and hyperresponsiveness of fetal-derived innate-like lymphocytes. By reshaping foetal HSC establishment, we show how inflammation in utero can direct the output and function of fetal-derived immune cells [2].

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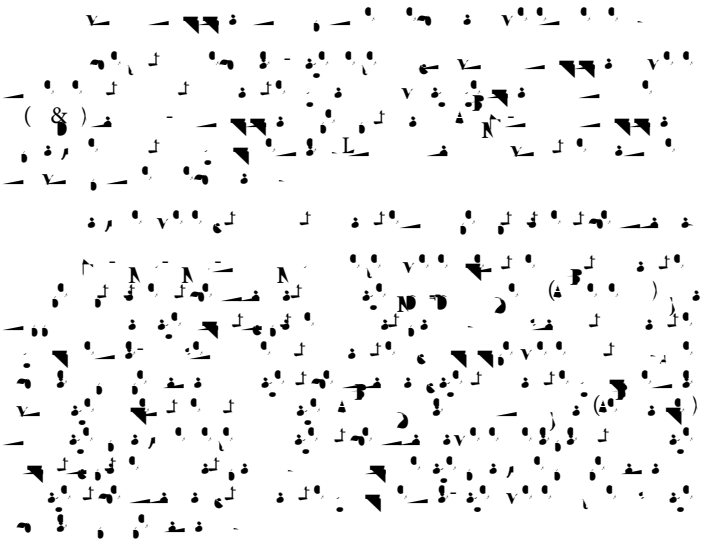

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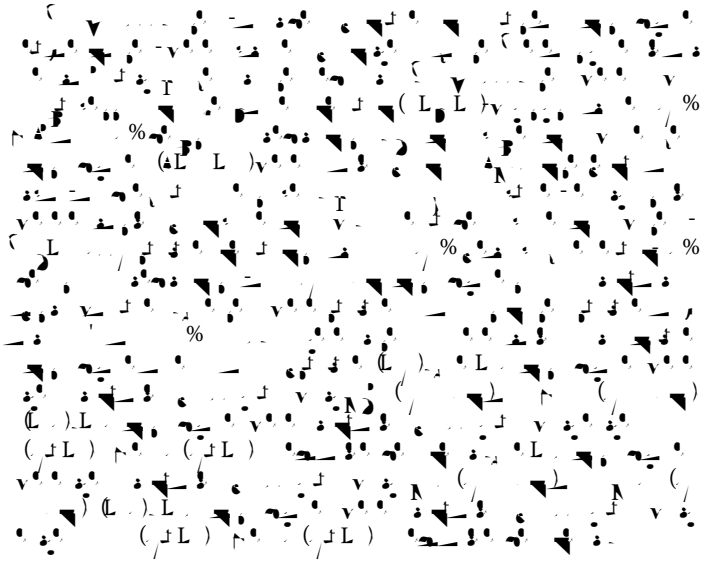

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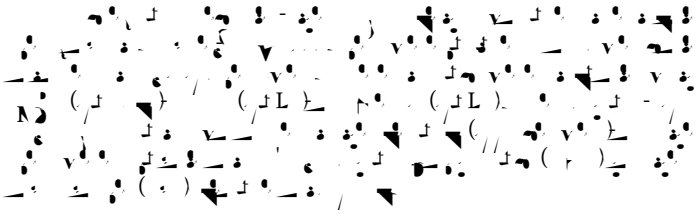

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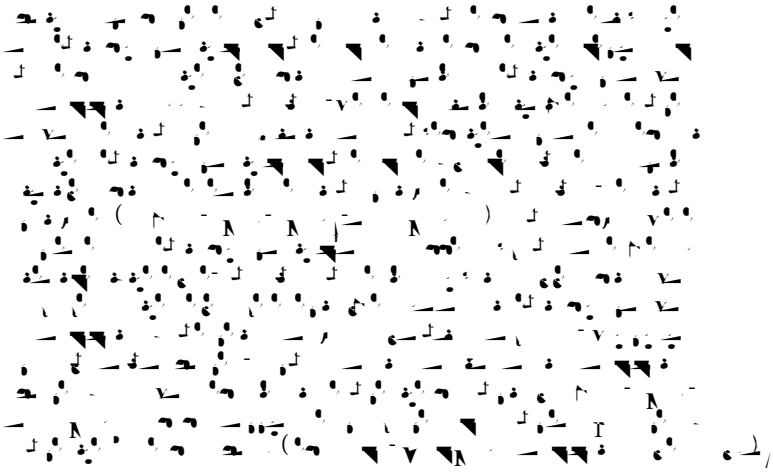

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