Stressing hematopoiesis and immunity: an acetylcholinesterase window into nervous and immune system interactions.

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Abstract:

Hematopoietic stem cells (HSCs) differentiate and generate all blood cell lineages while maintaining self-renewal ability throughout life. Systemic responses to stressful insults, either psychological or physical exert both stimulating and down-regulating effects on these dynamic members of the immune system. Stress-facilitated division and re-oriented differentiation of progenitor cells modifies hematopoietic cell type composition, while enhancing cytokine production and promoting inflammation. Inversely, stress-induced increases in the neurotransmitter acetylcholine (ACh) act to mitigate inflammatory response and regain homeostasis. This signaling process is terminated when ACh is hydrolyzed by acetylcholinesterase (AChE). Alternative splicing, which is stress-modified, changes the composition of AChE variants, modifying their terminal sequences, susceptibility for microRNA suppression, and sub-cellular localizations. Intriguingly, the effects of stress and AChE variants on hematopoietic development and inflammation in health and disease are both subject to small molecule as well as oligonucleotide-mediated manipulations in vitro and in vivo. The therapeutic agents can thus be targeted to the enzyme protein, its encoding mRNA transcripts, or the regulator microRNA-132, opening new venues for therapeutic interference with multiple nervous and immune system diseases.

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