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"Investigating the role of serotonin receptor 2c in regulating helminth-induced inflammation."

by
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M.S. 2018, Rutgers Graduate School of Biomedical Sciences, Newark
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Department of Medicine

Thursday, December 7th, 2023
11:00 A.M.
Cancer Center G1196

Join Zoom Meeting
https://rutgers.zoom.us/j/95669515627?pwd=SCtXa0V2Z2lHZi9CNjhSSHR5bzdwQT09

Meeting ID: 956 6951 5627
Password: 398290

ABSTRACT

A growing body of literature has begun to reveal that neuropeptides and neurotransmitters released in highly innervated peripheral tissues operate as critical regulators of helminth-induced inflammation. Helminth parasites, such as hookworms, can affect several host tissues, including the lung, where they promote strong type 2 cytokine responses that support worm clearance and simultaneously initiate pulmonary wound healing. Our work, and that of others, has recently identified monocyte-derived alveolar macrophages (Mo-AMs) with an alternatively activated, or M2, phenotype as critical regulators of these host protective responses. Mo-AMs populate the lung in response to hookworm-induced inflammation and take on a highly activated M2 phenotype compared to tissue-derived AMs. Further, our work has shown that Mo-AMs change phenotypically the longer they persist in the lung, while they receive tissue-derived signals that suppress their highly activated state. Despite these advances, whether neuron-associated factors participate in this tissue imprinting process remains unknown. To address this, we performed transcriptional profiling of lung macrophage populations following infection with the hookworm *Nippostrongylus brasiliensis* (Nb) and investigated their expression of various receptors for neuron-derived molecules. Interestingly, Mo-AMs were found to gradually acquire high levels of the serotonin receptor 2C (5HTR2c) after entering the lung microenvironment. Serotonin (also known as 5HT) is a neurotransmitter that plays important roles in myriad biological functions beyond its well described roles in the central nervous system. However, whether 5HTR2c-derived signals regulate the activation of monocyte-derived macrophages is unclear. Our work indicates that macrophages lacking 5HTR2c are less able to promote the wound-healing functions needed to mitigate helminth-induced tissue damage. Collectively, this work suggests that 5HT may operate to fine-tune helminth-induced M2 response and provides additional insight into the neuron-associated factors that regulate macrophage responses in the lung.