

Chronic alcohol consumption inhibits peripheral NK cell development and maturation by decreasing the availability of IL - 15

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First published: 11 November 2016

<https://doi.org/10.1189/jlb.1A0716-298RR>

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Abstract

NK cells are innate immune cells and have important roles in antiviral and antitumor immunity. Based on the transcriptional regulation, organ distribution, and cell function, NK cells have recently been further divided into cytotoxic conventional NK cells (cNK) and noncytotoxic helper - like group 1 innate lymphoid cells (ILC1s). It is well known that chronic alcohol consumption decreases peripheral NK cell number and cytolytic activity; however, the underlying mechanism remains to be elucidated. How chronic alcohol consumption affects ILC1s is, to our knowledge, completely unexplored. Herein, we used a well - established mouse model of chronic alcohol consumption to study the effects of alcohol on transcription factor expression, maturation, and cytokine production of cNK cells and ILC1s in various organs. We found that alcohol consumption significantly decreased Eomes - expressing cNK cells in all the examined organs, except BM, but did not significantly affect ILC1s. Alcohol consumption compromised cNK cell development and maturation. Exogenous IL - 15/IL - 15R α treatment caused full recovery of Eomes - expressing cNK cell number and maturation. Taken together, our data indicated that chronic alcohol consumption decreases cNK cell number and cytolytic activity by arresting cNK cell development at the CD27⁺CD11b⁺ stage. This developmental arrest of NK cells results from a lack of IL - 15 availability in the microenvironment. IL - 15/IL - 15R α treatment can recover alcohol consumption - induced developmental defect in NK cells.

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