There is now consensus for the existence of three major subsets of ILC that can be distinguished on basis of transcription factors important for development and function and the cytokines they produce. Group 1 ILC which include ILC1 and NK cells producing IFN-\(\gamma\) and depend on Tbet and Eomes for function and development, Group 2 ILC that depend on GATA3 produce type 2 cytokines, IL-5 and IL-13 and group 3 ILC that depend on ROR\(\gamma\) and produce IL-22.

Emerging data suggest that each subset can exist immature, naïve and primed stages. I will discuss the different features of these stages. We observed that primed ILC are present in the peripheral blood of individuals with severe inflammation of the airways which might suggest that inflammatory ILC might be able to circulate.

Each subset has the capacity to change phenotype and function dependent on the nature of the signals they encounter in the tissues. Previously we demonstrated that ILC2 can transdifferentiate into ILC1 and vice versa. Here I will present examples of plasticity of ILC2 that enables them changing into producers of IL-17. The possible impact of ILC2 plasticity for pathology in inflammatory diseases of the airways and skin will be discussed.

**ABSTRACT**

**DEVELOPMENT AND PLASTICITY OF HUMAN ILC**

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