Editorial
Volume 12:7, 2021

Journal of Tissue Science and Engineering

ISSN: 2157-7552 Open Access

The Role of Myeloid Cell Differentiation in Natural Killer Cell

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Editorial Note

A number of specialised transcription factors, including PU.1, CCAAT/Enhancer-Binding Proteins (C/EBP) and interferon-regulatory factors, drive myeloid cell development from common myeloid progenitors into discrete subsets. PU.1 works with IRF-8 to drive macrophage commitment from granulocyte/macrophage progenitors, as well as encourage the formation of Dendritic Cells (DCs), whereas C/EBP directs granulocytic differentiation. Myeloid cells are sensitive to microenvironmental signals and can be reprogrammed to support inflammation or immunosuppression, primarily *via* Nuclear Factor kappa B (NF-B) and the Signal Transducer and Activator of Transcription (STAT) family of transcription factors.

Peptidase and inhibitor genes are among the genes that are differently regulated during lineage commitment and myeloid cell differentiation. In DCs and macrophages, IRF-8 worked together with PU.1 to stimulate the expression of genes encoding various lysosomal enzymes, including cathepsins C, L, and S, as well as their inhibitor cystatin C. C/EBP, on the other hand, decreases cystatin F levels during monocytic and even more so during granulocytic development of the promonocytic cell line U937.

As a result, after conversion of U937 cells to macrophages, the activity of cathepsins C, L, and S, which are no longer blocked by cystatin F, increase. Even though cystatin F suppresses cathepsin H in NK cells, it had no effect on cathepsin H activity in U937 cells, showing that the sensitivity of specific cathepsins to inhibition differs between cell types. Cystatin F differs from other cystatins in that, it can be transferred to endolysosomal vesicles following production.

As a result, it's thought to be the primary regulator of cathepsin vesicular activity in immune cells. Immune cells such as CTLs, promonocytic cells, and DCs, on the other hand release cystatin F. Indeed, cystatin F-rich myeloid cells could serve as a reservoir for extracellular cystatin F, which could reduce NK cell cytotoxicity after internalisation into bystander cells.

Cysteine cathepsin expression can be regulated further after lineage commitment. The production and release of cathepsins B, C, H, L, S, and X from murine macrophages is triggered by the synergistic activation of STAT-3 and -6 by the cytokines IL-4 and IL-6 or IL-10. Furthermore, cathepsin expression can be elevated in hypoxic TME, as evidenced by cathepsin B's promoter region, which contains a hypoxia response element. While there is no doubt that signalling pathways begun by differentiating, activating, or inhibiting stimuli have a significant impact on peptidase production and activity in immune cells, cathepsins and their inhibitors can also contribute to signal transduction.

Cathepsins F, H, L, K, S, and V are inhibited by extracellular thyropin from the p41 invariant chain, which is one of the isoforms of the MHC class II chaperone protein involved in antigenic peptide presentation. In lipopolysaccharide-stimulated DC however, it was demonstrated to interfere with nuclear translocation of the NF-B p65 subunit, resulting in decreased IL-12 production. The cellular signalling mediated by cathepsin X, which is mediated by proteolytic processing of the C-terminus of the 2 integrin chain, has also been well documented. When DC is activated, cathepsin X translocates to the plasma membrane, allowing the integrin receptor Mac-1 to convert to its high-affinity form, allowing DC to mature adhesion-dependently. The periplasmic interaction of cathepsin X with 2 integrin receptors produced by activating stimuli is also necessary for adherence and phagocytosis of U937 macrophages.

How to cite this article: Nicholas, Robinson. "The Role of Myeloid Cell Differentiation in Natural Killer Cell ." *J Tiss Sci Eng* 12 (2021) : e011.

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Received: December 16, 2021; **Accepted** : December 30, 2021; **Published**: January 06, 2022