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CD103 regulation in human dendritic cells using retinoic acid in the gastric microenvironment

CD103 (α E integrin) is an important marker for dendritic cells (DCs) in the human mucosa. Iliiev et. al (2016) showed that CD103+ DCs display tolerogenic behavior in the human gut and induce Treg cell development. However, not much is known about the regulation of its expression, though it is widely used as a delineator of DC populations. Previous work in my group shows that retinoic acid (RA) and toll-like receptor agonists contribute to the regulation of CD103 expression in human DCs (Roe et al, 2016). We postulate that CD103 functions to initiate DC binding to gastric epithelium, possibly to E-cadherin, in order to allow for antigen sampling through tight junctions by DC dendrites. Additionally, previous research in this lab has concerned the identification of gastric stromal factors using gastric stroma- conditioned media (SCM), which is a model for the gastric microenvironment. I have shown that gastric stromal factors are responsible for suppressing dendritic cell maturation in *Helicobacter pylori* infection. We have thus confirmed SCM-derived immunoregulatory factors as a suitable model for generating dendritic cells with a tolerogenic mucosal phenotype. Here, we've confirmed that (RA) induces CD103 expression in peripheral-blood monocyte derived dendritic cells (MoDCs). Additionally, we show that the addition of SCM increases the extracellular expression of CD103 in both RA and non RA treated conditions. Lastly, I show that intracellular concentrations of CD103 in the presence of SCM are lower than extracellular concentrations, whereas CD103 is predominantly located in the cytoplasm in the absence of SCM, indicating that SCM may be a catalyst for initiating redistribution of CD103 to the cell membrane.

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