

A systems model for immune cell interactions unravels the mechanism of inflammation in human skin

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Abstract

Inflammation is characterized by altered cytokine levels produced by cell populations in a highly interdependent manner. To elucidate the mechanism of an inflammatory reaction, we have developed a mathematical model for immune cell interactions via the specific, dose-dependent cytokine production rates of cell populations. The model describes the criteria required for normal and pathological immune system responses and suggests that alterations in the cytokine production rates can lead to various stable levels which manifest themselves in different disease phenotypes. The model predicts that pairs of interacting immune cell populations can maintain homeostatic and elevated extracellular cytokine concentration levels, enabling them to operate as an immune system switch. The concept described here is developed in the context of psoriasis, an immune-mediated disease, but it can also offer mechanistic insights into other inflammatory pathologies as it explains how interactions between immune cell populations can lead to disease phenotypes. © 2010 Valeyev et al.

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