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How does blood coagulation/neutrophils shape innate immunity and uncontrolled inflammation to autoimmune disease?

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Continuous challenges of microbes, non-living entities or occasional trauma/injuries shape host defense systems at various hierarchical levels. The primary defense of the host is anatomical and biochemical barriers that continuously work to protect the host from common and pathogenic microbes. This specific defense barrier consists of various cell-types expressing molecular sensors that sense numerous pathogen-specific molecular signatures and activate complex signaling pathways for the synthesis of inflammatory cytokines and subsequently facilitate the elimination of invading microbes. Dysregulation of signaling pathways may lead to the heightened production of inflammatory cytokines that culminate to cause autoimmune diseases. This issue of *International Reviews of Immunology* focuses on innate immune defenses triggered through blood clotting and blood cells known as neutrophils. This issue also describes an autoimmune disease and its impact on various physiological systems such as connective tissue and blood vascular system (Figure 1).

The wound or injury to the host not only causes pain and loss of blood, but it also opens the gates for microbes to establish themselves, colonize and cause disease. To overcome such multidimensional threats, the mammalian host evolved a unique, biochemically complex phenomenon known as blood clotting. Blood clotting is rapidly initiated through sequential activation of serine proteases at the wound site to convert fibrinogen (a soluble protein) to fibrin (insoluble network) and is further stablized by several other blood factors to stop blood loss and make a physical barrier against the invading microbes. The first review article in this issue by Minasyan et al. discusses the importance of blood clotting in innate immune defense, particularly how blood clots develop anti-bacterial responses by various mechanisms (Figure 1).

The neutrophils are predominant cell-type in white blood cells and the first responders during infection, injuries or any homeostatic changes taking place within the host. Neutrophils are ephemeral and depend on physiological changes (due to infection, cancer or autoimmune diseases); the death signals to neutrophils are unique. The second review article in this issue, by Browska et al., focuses on different signaling events converging to the neutrophils for neutrophils death under various extrinsic and intrinsic stimuli. The authors also discuss the readout of these death pathways that may help in the development of diagnostics for different diseases. Additionally, manipulation of death signaling pathways may also be helpful in altering the disease condition. Since the field is in its infancy, the review article may be useful to basic and translational researchers working on interfaces of innate immunobiology and applying the knowledge to the development of innate immune parameter-based diagnostic or therapeutics (Figure 1).

Psoriasis is primarily a skin autoimmune disease, and the severity of this disease is influenced by genetics and environment factors. This disease not only affects skin, but it also targets joints and the cardiovascular system, causing psoriatic arthritis and cardiovascular disease (CVD). The third review article in this issue by Manolis et al. discusses the beneficial effects of biomolecules used in therapy of psoriasis to psoriatic CVD. However, the authors suggest conducting large number patient-based studies before concluding its beneficial effects to CVD in psoriasis patients. This article is useful to broad readers working in clinics and conducting research in fundamental immunology, autoimmunity and associated fields (Figure 1).

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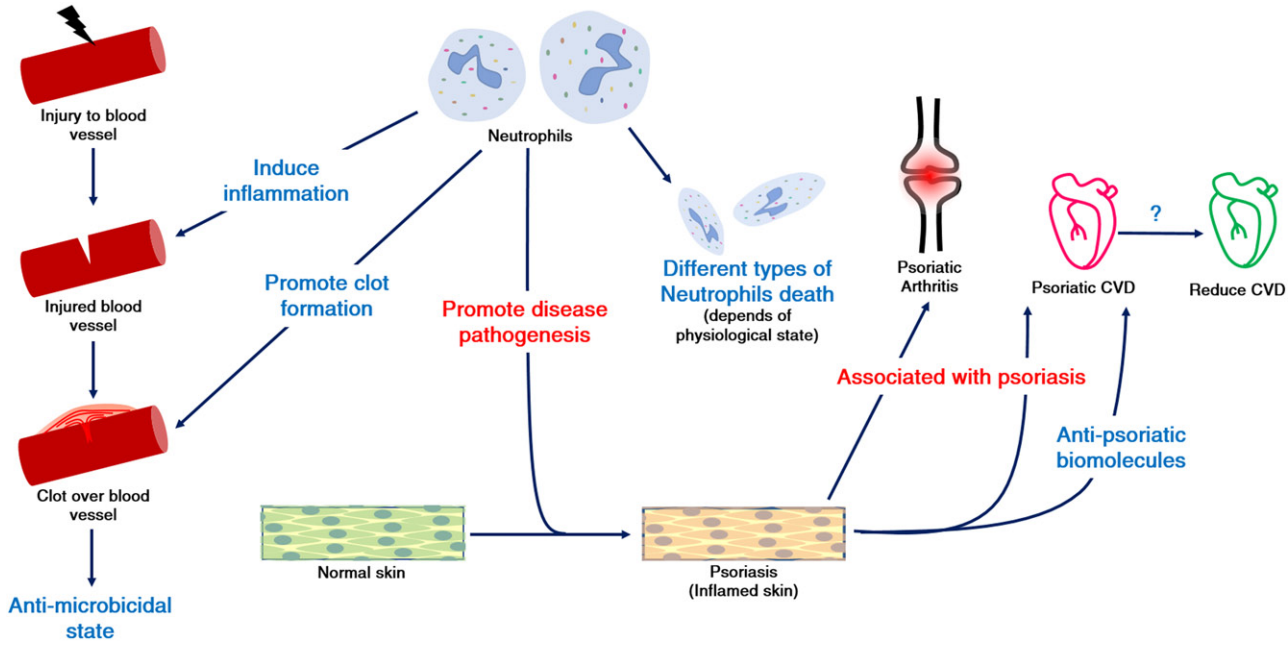

Figure 1. Immunity and auto-immune disease.

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