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[CANCELLED] Coagulation cascade as a dynamic biological system

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Coagulation cascade is one of the best-known physiological systems, which biological role is regulated by 20 coagulation factors, 7 fibrinolytic factors and cell elements (platelets and endothelium). The number of known factors has been incrementally extended within 50-ties of previous century, giving clinicians more understanding about this extremely complicated biological system, to treat patient conditions and decide about a patient outcome.

Traditionally, for the medical purpose, the coagulation cascade is classified into two pathways: an intrinsic and an extrinsic pathway, both of which meet factor X activation. This classical theory of blood coagulation developed by MacFerlane, Davie and Ratnoff is particularly useful for understanding the *in vitro* coagulation. Coagulations factors activity and concentrations easily became to be used as biological indicators (*in vitro* tests) to predict a physiological or pathological state of a patient.

In fact, coagulation process is more complicated, and this simple classification fails to incorporate the central role of cell-based surfaces in in vivo coagulation process. Developing mathematical models of biochemical networks is a significant facet of systems biology. Bottom-up approach assumes specific molecular properties of coagulation and fibrinolytic factors, and quantified interactions as stoichiometry, kinetics, binding properties, inhibition, diffusion, and others. The resulting models then predict the activity of biochemical pathways, platelets, and vascular tissues, either during physiological hemostasis (coagulation and fibrinolysis) or during pathological thrombosis of bleeding.

In this talk I will discuss a traditional model of coagulation (tissue-factor-triggered models) and compare them with a dynamic fibrin polymerization and platelet signaling.

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