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# On the regimes of blood coagulation

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## Abstract

Reaction-diffusion system of equations describing blood clotting is studied. Different regimes of clot growth are identified in a quiescent plasma and in blood flow depending on the relative strength of initiation, propagation and inhibition of the thrombin production.

**Keywords:** blood coagulation, clot growth regimes, numerical simulations

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## 1. Introduction

Blood coagulation is a defensive mechanism that allows the organism to preserve hemostasis. Among various blood factors, thrombin plays a particularly important role in blood coagulation by converting fibrinogen into fibrin polymer in an enzymatic reaction. The generation of thrombin in blood plasma occurs upon the activation of the factors IX and X by tissue factor (TF) located at the outer surface of endothelial cells. In normal situations it interacts with blood plasma in the case of an injury. There are also various pathological situations where it is involved and initiates blood coagulation (thrombosis, inflammation, cancer, etc.). Once activated, the thrombin production can be continued in a self-sustained manner due to a positive feedback but it can also be inhibited by other factors such as antithrombin and activated protein C (APC).

There exists an extensive literature devoted to modelling of blood coagulation and clot growth. The methods of modelling are based on partial differentiation equations for concentrations of blood factors and for blood flow [1, 2, 3], various particle methods (especially in modelling of blood flows with blood cells), and hybrid methods which represent a combination of these two approaches [4, 5, 6, 8]. In spite of numerous works in this area, peculiarities of clot growth are not yet completely understood due to the complexity of the process and large variability of physiological conditions. In this work we identify different regimes of clot growth depending on the balance between different stages of thrombin production: the initiation, the propagation and the inhibition. These regimes can be characterized as clot initiation without propagation, initiation with propagation, propagation with inhibition by APC and by blood flow. We study different regimes of clot formation in numerical simulations of the complete model (section 2) and in a simplified analytical model (section 3). Platelets and their influence on regimes of clot growth are not considered in this work (cf. [5, 6]) and will be discussed in the subsequent work.

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