



## Genetic Basis of Fibrinogen Disorders

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### Message from the Guest Editor

Dear Colleagues,

Congenital fibrinogen disorders, including a-, hypo-, and dys-fibrinogenemia, are estimated to represent ~8% of rare inherited coagulopathies. In addition to the most obvious consequences of fibrinogen disorders (ie, hemorrhagic/thrombotic manifestations), point mutations can also lead to organ damage; in the liver, due to endoplasmic accumulation of mutant fibrinogens, and in the kidney as a result of an increased susceptibility to proteolysis of aggregation-prone fibrinogen peptides, leading to systemic amyloidosis.

In this Special Issue of the *International Journal of Molecular Sciences*, the focus will be the “Genetic Basis of Fibrinogen Disorders”, including insights into epidemiologic data, mutational spectra, and molecular pathogenesis. Studying fibrinogen spontaneous mutants in the population can represent a useful tool to inspect critical residues for fibrinogen assembly, secretion, function, and interaction with other proteins, as well as to elucidate mechanisms underlying fibrinogen-chain mRNA processing.

Assoc. Prof. Dr. Rosanna Asselta  
*Guest Editor*





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