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