In the present thesis is investigated the interaction of domain D of *S. aureus* Protein A (SpaD) with domain A1 of von Willebrand factor onto biofunctionalized scaffolds - experimentally as well as with molecular dynamic techniques. SpaD was cloned in pAN5 plasmid, expressed, in vivo biotinylated by AVB101 *E.coli* strain and immobilized onto cellulose acetate scaffolds by using biotin-streptavidin strategies. The effectiveness of the functionalization of the scaffolds was tested with the immobilization of biotinylated green fluorescent protein (GFP) and its direct visualization with confocal fluorescence microscopy. The scaffolds were subsequently incubated with *S. aureus* culture with or without the presence of A1 domain of vWF. The non-adherent properties of the resulting scaffolds were examined with scanning electron microscopy (SEM). The exact configuration of interaction of A1 and SpaD was further elucidated by using molecular dynamics and docking techniques. Six putative configurations were modeled and equilibrated for 10 ns. The interaction energies and inter-protein distances were measured in order to identify the possible configuration of interaction. The interaction site of A1 consists of two antiparallel beta sheets (b2 and b3) and part of an α-helix, resembling the interaction interface of SpaD with IgM. In addition mutants of SpaD exhibiting decreased binding to A1, as determined experimentally, were modeled based on the putative complex of SpaD-A1 interaction. The resulting mutant manifests considerable higher interaction energy with A1 and significant translation and rotation in comparison to the wild type. Furthermore, the denaturing effect of urea and high temperature on A1, A2 and A3 domain of von Willebrand factor was investigated in order to elucidate the mechanism of lethal hemorrhages occurring in case of renal failure. The extent of denaturation was estimated by structural parameters as helical/beta sheet content, native contacts and RMSD. A1 manifests significant resistance to the denaturing effect of both heat and urea, whereas A2 and A3 exhibit considerable denaturation. The cleavage site of A2 domain for ADAMTS13 is exposed after 16 ns of simulation time. The intermediate states of the pathway of denaturation were determined making use of the principal component analysis technique. Urea induces an additional loss of secondary structure and a considerable increase of total protein energy in comparison to heat alone. The study of urea induced denaturation and solubility of proteins in aqueous urea solutions was based on the investigation of the behaviour of the N-terminal 1-57 peptide of HPNAP protein of *Helicobacter pylori* in urea solutions of variable potency. A considerable amount of the simulations has been carried out on the Southeastern European Grid making use of a parallel running version of NAMD molecular simulation package. A detail protocol concerning the workflow of preparation, submission and collection of simulation data has been developed to this end.