Histidine-rich glycoprotein (HRG) is a plasma adaptor protein that regulates a number of biological processes in the blood, including immune function, angiogenesis and coagulation. This heavily glycosylated protein contains two proline-rich regions which flank a distinctive disordered histidine-rich region (comprised of a repeating GHHPH motif). This domain associates with Zn$^{2+}$ ions to stimulate HRG-complex formation. Under normal conditions the majority of Zn$^{2+}$ in the blood associates with human serum albumin (HSA), but we have recently demonstrated that high levels of fatty acid (within the physiological range) allosterically disrupt the major Zn$^{2+}$-binding site on HSA. Increased levels of circulatory fatty acids are therefore likely to increase the proportion of plasma Zn$^{2+}$ associated with HRG. HRG regulates coagulation via association with heparin (which in turn inhibits antithrombin III activity) in a Zn$^{2+}$-dependent manner. We speculate that fatty acid-binding to HSA increases HRG-complex formation via this mechanism. Clinically, elevated levels of fatty acids such as those observed in patients with cancer, obesity and diabetes are associated with an increased risk of thrombosis. In this study we investigated Zn$^{2+}$-binding to purified HRG and HSA in the presence of different concentrations of lauric acid by competitive equilibrium dialysis. The formation of the HRG-heparin complex in the presence of different Zn$^{2+}$ concentrations was also characterised using Isothermal Titration Calorimetry (ITC).