the *in situ* single pass intestinal perfusion (SPIP) experiments, GlcN increased PRN BA significantly ($p<0.05$) by two-fold at 60 min as compared to cimetidine and rifampin. This was confirmed by everted gut experiment where GlcN enhanced the absorption of PRN at 20, 40, and 60 min. Finally, using isolated hepatocyte cell culture, GlcN at 200 mM decreased PRN metabolism and increased PRN concentration significantly ($p<0.05$). On the other hand, 50 μM of rifampin increased PRN metabolism and decreased PRN concentration, whereas cimetidine at 5 μM increased PRN concentration as expected for such positive controls.

Overall, GlcN decreased PRN BA in a dose-dependent manner by decreasing its *in vivo* intestinal absorption and permeability but increased PRN concentration levels *in situ* and *in vitro*. This might be attributed to factors prior intestinal absorption such as the pH of the stomach, PRN and GlcN pK_a and the efflux transporter P-glycoprotein (P-gp). Furthermore, only the highest tested dose of GlcN (200 mM) was capable of affecting PRN levels when incubated with viable rat hepatocytes. Therefore, it might be necessary to prescribe PRN with GlcN with caution due to the current reported interactions. A dosage regimen adjustment of PRN might be required to achieve the desired therapeutic effect in patients receiving GlcN.