study has shown that the energy-dependent efflux P-gp pump located on the apical plasma membrane of rabbit conjunctival epithelial cells plays a role in restricting PRN transport (Yang *et al.* 2000). Hirakawa *et al.* have shown that GlcN induced the expression of Multidrug Exporter Genes (*mdtEF*) in *Escherichia coli* that is stimulated through catabolite control. This result indicated that GlcN may be responsible for the increased multidrug resistance in the bacteria (Hirakawa *et al.* 2005). However, there are limited studies of the effect of GlcN on P-gp in rats, therefore, further studies are still warranted. The current study did not investigate the effect of P-gp efflux transporter on PRN-GlcN combination; however, it cannot be excluded that this could be potentially responsible for the reduction in the *in vivo* levels of PRN.

GlcN PK studies in rats have shown that GlcN undergoes extensive hepatic first pass metabolism resulting in low BA (about 19- 21%) (Adebowale *et al.* 2002; Persiani *et al.* 2005; Thakral *et al.* 2007). PK and BA of GlcN have shown that C_{max} and AUC of GlcN in human were linear only within dose range of 750-1500 mg that was preceded by a pre-saturated 750 mg phase. GlcN C_{max} and AUC at 3000 mg dose were significantly lower than the corresponding values calculated at the dose of 750 mg. This suggests the involvement of two processes; a process that is capacity limited which is saturated with low doses, and a process that is linear for higher doses (Jackson *et al.* 2010; Persiani *et al.* 2005). If GlcN doses (100 and 200 mg/kg) used in this study were converted to human adults (7000 and 14000 mg/kg, respectively), which both are higher than 3000 mg/kg, wherein GlcN absorption in