human is reduced and this may be a possible reason that GlcN did not get absorbed and consequently did not increase PRN BA as in *in vivo* results **(Figure 3.19)**. However, a recent study in rats has shown a linear relation between the oral administered dose and the average AUC. Moreover, everted rat colon *in vitro* data also demonstrated a linear relationship between GlcN concentration and the accumulation rate from mucosal to serosal fluid indicating that GlcN intestinal absorption is linear and not capacity limited (Ibrahim *et al.* 2012). Transporters involved in GlcN absorption have been revealed to be (GLUT 1, 2 and 4), which also facilitate glucose absorption. In particular, GLUT2 has demonstrated a 20-fold greater affinity for GlcN than that for glucose. Transporter-regulated GlcN absorption increases the possibility of a capacity-limited intestinal absorption for the compound (Uldry *et al.* 2002), which was also addressed by Persiani *et al*. (Persiani *et al.* 2005). Nonetheless, PRN is much less influenced by cell transporter since it is a class I drug (Custodio *et al.* 2008), which most likely excludes the transporters as the potential reason of reduced PRN BA.