Iron is an essential nutrient for many critical functions in the human body and the deficiency or overload of iron in the cells contributes to the cause of many disorders. This is why it is carefully regulated in order to maintain iron homeostasis. DMT1 is the major iron importer across the enterocytes, while Ferroportin is the only known iron exporter [1]. DMT1 has 4 major isoforms which differ in where transcription starts and whether it has an Iron Responsive Element [1-3]. Research has shown that Ferroportin degrades thus losing its function when hepcidin is bound to it. Recent reports have shown that hepcidin reduces the activity of DMT1 in enterocytes before Ferroportin degrades [7, 8]. We are interested in finding out if hepcidin binds DMT1 as observed for Ferroportin. Our results have shown that hepcidin does not directly bind to DMT1, which led us to query how hepcidin signals to DMT1.

**Abstract**

Hepcidin may be able to inhibit DMT1 [HEK293 1A+IRE] in enterocytes by binding to the ferrous iron exporter Ferroportin (Fpn) [1] and thus reducing its activity. This study was performed to determine whether or not this was the case. The activity of DMT1 in HEK293 1B cells was measured by varying the incubation time (30min, 1hr, 2hr).

**Results**

**RhoG-hpcp may bind to another protein than Fpn in HEK2937 Fpn-GFP**

We wanted to see if RhoG-hpcp binds to Fpn on a western blot and since we have more data points for western blots for DMT1, we decided to start the experiment on both 1A+IRE and 1B-IRE cell lines.

**References**