# The role of cargo protein

The experiment shown in figure (3) was done to characterize the role of cargo protein binding domain of sec24p in protein sorting. The vesicles were generated with the Sec24L616W mutated from microsomal membranes. The number of cargo proteins were estimated by immunoblotting or by autoradiography which were then quantified by using secondary antibody (labelled with radioactive molecules).

The results are immune blots and showed the effect of Sec24L616W on packaging of cargo proteins. The findings of the experiment are this that the sec61p and Kar2p which are residents of ER were not found in vesicle fraction. They were missing from the vesicle when compared to wild type. there were group of proteins such as chitin synthase, profactor-a, were found in the mutant vesicles.

In this experiment they learned that there are multiple sites organizing the cargo protein signals that binds at their own and confirmed the presence of additional unidentified domains which also interacts with distinct types of cargo proteins. If the amino acid signal in protein Gap1p was mutated to some random amino acid signal, this will lead to affected condition.

If we can tell DID is replaced by DXE signal and If amino acid signal LxxLE in Bet1p was mutated to the amino acids DID which was signal of Gap1p, the following different result would be expected: There are more chances that there will be two bands on immunoblot in 2nd and 3rd lane, which is normal and signal will not be disturbed for cargo protein, as the result would be similar to Gap1p.

Bet1p is important site and mutation at this site causes defects in cargo packaging. If this is mutated with gap1p signal, the results may resemble to Sed5p, Erp1p or Bos1p, that are affected cases. Due to this mutation there are more chances to disrupt cargo packaging.