Efficient neurotransmitter loading in synaptic vesicles is important as impaired loading leads to a decrease in released neurotransmitter in the synaptic cleft which influences the communication with the postsynaptic neuron. Neurotransmitters are taken up in synaptic vesicles by different vesicular neurotransmitter transporters. Inhibitory neurotransmission is mediated by the neurotransmitters GABA and glycine, which are transported into synaptic vesicles by the vesicular GABA transporter (VGAT). The aim of this project was to characterize the mechanism of VGAT particularly with respect to the role of chloride and ΔμH+ in GABA uptake. At this point in time, two contesting models have been proposed for the VGAT mechanism; VGAT as a proton-GABA exchanger or as a chloride-GABA co-transporter. There has been no definitive answer to which mechanism occurs. To address this issue, liposomes reconstituted with purified VGAT and an ATPase (TFoF1) from *Bacillus thermophilus* were used as a model system, similar to synaptic vesicles, in which all factors could be controlled. After successful reconstitution, acidification at different conditions was studied using two pH sensitive dyes (acridine orange and pHrodo). Based on the data obtained in this study, no evidence was found to support the GABA-chloride co-transporter hypothesis. The data is more readily explained by a VGAT-intrinsic proton leak. Future experiments can include but are not limited to the following: measurements of membrane potential changes, GABA uptake measurements using radiolabelling and measurements of the dissipation of the pH gradient using EDTA.