Protein folding and maturation is an error prone process. Mammalian cells have evolved quality control machinery that evaluates proteins and assists in repairing those with aberrant structures, while targeting proteins that fail to reach their native state for degradation. Proteins that traverse the secretory pathway frequently receive N-linked glycans that are processed by deglucosylation and reglucosylation. Trimming of the first two glucoses generates proteins with monoglucosylated glycans that are recognized by the carbohydrate binding chaperones calnexin and calreticulin. Trimming of the last glucose releases proteins from the lectin chaperones. UGT1 is an early gatekeeper involved in quality control by reglucosylating proteins, thus supporting additional rounds of binding to calnexin and calreticulin. However, how UGT1 functions in its native environment and which types of substrates are recognized by UGT1 is poorly understood. We found that UGT1 reglucosylates wild type substrates that will eventually fold and that trapping these substrates in the reglucosylated state delays their trafficking through the secretory pathway. Interestingly, UGT1 is also able to efficiently recognize terminally misfolded substrates yet trapping these substrates in the reglucosylated state did not affect their degradation rate, indicating that they were actively extracted from the UGT1 cycle and targeted to degradation independent of their glucosylation status as shown in the model above. These findings make UGT1 an interesting target for designing therapies to diseases caused by strict ER retention of substrates with minor imperfections that are otherwise functional, without affecting the clearance of misfolded nonfunctional substrates.