Alzheimer's disease (AD) is most commonly due to the retention of amyloid β-peptide (Aβ) in the cerebral cortex. We showed that the multidrug resistance P-glycoprotein (Pgp) participates in removing Aβ from the parenchyma into the circulation. Other Pgp substrates have been shown to have either competitive or cooperative impacts on one another's removal, apparently due to binding, respectively, at the same or a distinct active site on Pgp. We aim to identify drugs that impede or promote clearance of Aβ in this way. First, we will mine clinical health data for correlations between AD diagnosis and prescription for drugs which are substrates of Pgp. Second, these substrates will be tested for effects on Aβ transport *in vitro*. Third, molecular modeling will identify the binding sites of these molecules. A clearer understanding of Aβ interactions with Pgp and its other substrates may identify compounds that either promote or impede Aβ clearance.