Alzheimer’s Disease (AD) is among the most prevalent and debilitating of over 40 different incurable human diseases associated with misfolding of a specific protein or peptide. Despite the large volume of research that has focused on understanding AD mechanisms and identifying possible treatments, the field remains divided on what causes the disease, and therefore, what aspect(s) of disease pathology might be targeted to cure the disease or even alleviate symptoms. One avenue of research hypothesizes that dyshomeostasis of brain metals such as copper, zinc, and iron is a key step in disease progression. To this end, metal chelation therapy has been explored as a possible AD treatment. Promising results with 8-hydroxyquinoline (8HQ) metal chelators, in both animals and humans, has shown improvements to cognition and memory. Although these chelators have been postulated to restore metal ion homeostasis, specifically within the brain, their mechanism of action remains largely unknown. To better determine the mechanism of action of 8HQ chelators, we investigated the solution structure of 8HQ Cu(II) complexes, as well as the Cu(II)-binding site in aggregated Aβ (one of the major components of plaques observed in AD brains), using synchrotron X-ray absorption spectroscopy (XAS). We have also investigated metal ion distributions in mammalian brain tissue treated with 8HQs using synchrotron X-ray fluorescence imaging (XFI). Results from synchrotron techniques, and other more conventional techniques suggest that 8HQs may interact directly with Aβ, which may have important implications for the mechanism of action of these drugs and for the development of novel AD treatments.