The involvement of the intracellular chaperone system in cellular quality control mechanisms is well established; however, information about the extracellular counterparts have been relatively unknown. Recently, extracellular chaperones have been discovered, with the most well documented one being clusterin. Clusterin has been linked as a risk factor for late onset Alzheimer’s disease (LOAD) in 2009 following 2 independent genetic screens; therefore, understanding the mechanism by which clusterin prevents protein misfolding is of particular interest. In our study, we investigate the interaction of clusterin with amyloid beta (Aβ) oligomers, with specific focus on cellular effects such as binding and uptake of the oligomers into a range of cell types. Clusterin appears to reduce binding of oligomers to a number of cell lines, implying a generic mechanism of action, and also alters the mechanism of binding/uptake when compared to oligomers alone, as seen by confocal microscopy. The biophysical interaction between clusterin and the oligomers has been investigated with a range of techniques to attempt to characterise the effect of the clusterin on the oligomers and to put forward a hypothesis as to how the chaperone is exerting its action.