The deterioration in a person’s memory, social and/or language abilities to the extent when normal life becomes impacted is what characterizes the debilitating group of diseases commonly referred to as ‘dementia’. Of the various clinical dementias recognized, Alzheimer’s disease (AD) is the most common and is currently estimated to affect more than 5 million people in the United States alone.

AD is thought to be a ‘proteinopathy’, which means the accumulation of the pathologic proteins in the brain. These proteins deposit in structures called plaques and tangles (Hyman et al. 2012). The accumulation of beta-amyloid protein into plaques occurs as early as 15-20 years prior to the onset of clinical dementia (Bateman et al. 2012) and research has shown that therapies that intervene as early as possible in the disease course more effectively modify the disease (Sperling et al. 2011). However, research has also shown that people other than those with AD deposit beta-amyloid protein in their brain, including patients with other forms of dementia. In particular, we now know that as many as a third of patients with frontotemporal dementia (FTD) have such deposits (Duyckaerts 2011; Chare et al. 2014) and may benefit from any treatment targeting this protein deposition. Given that two thirds of FTD patients deposit different pathologic proteins in their brain (Josephs et al. 2011), accurate identification of people with dementia syndromes caused by different protein deposits is crucial, particularly as treatments targeting these specific proteins become available.

With the recent advent of diagnostic tools that can detect AD amyloid in patients prior to the onset of clinical symptoms, the future of early therapeutic intervention appears promising (Klein and Kaye 2013). Identifying the offending pathologic protein responsible for disease is the first crucial step towards effectively developing molecular treatments that can target and/or inhibit the accumulation of these pathogenic proteins (Sperling et al. 2011). The next challenge for dementia research is to establish the clinically relevant pathological threshold/s and severity indices for amyloid deposition that will inform and improve the diagnostic accuracy of current molecular imaging tools, and enable their application across dementia syndromes.

References:

