The occurrence of Alzheimer’s disease (AD), together with the major health and social care burden associated with it, is predicted to increase considerably with our ageing population. Current drug treatments at best slow the progression of the disease and only in some patients with AD. It is understood now that inflammatory processes play a major role in the development and progression of AD. Tumour necrosis factor alpha (TNF-α), one of the key chemical messengers mediating this inflammatory response, appears to accelerate both the changes in brain structure and the memory problems that comprise AD. Preliminary clinical data has raised the possibility that drugs which block the action of TNF-α may actually result in clinical improvement in AD. However the effects of these drugs, which are already used in the treatment of rheumatoid arthritis (hence their designation as biological disease-modifying anti-rheumatic drugs or DMARDs), now need to be studied in larger samples. The one large UK study that we know of identified patients with AD from death certificates, which we believe significantly underestimates the prevalence of the disease. Our proposed study uses treatment with anti-dementia drugs as a more sensitive and specific marker of AD diagnosis, with the aim of linking this data to registers of patients receiving biological and older, synthetic DMARDs. The primary research question is whether treatment with biological DMARDs, i.e. inhibition of TNF-α, is associated with a reduction in the occurrence of AD, compared to synthetic DMARDs which do not block TNF-α.