**Background:** Identification of causal relationships between modifiable targets and risk of dementia is central to the development of evidence-based prevention strategies. Blood lipid levels have been implicated in the aetiology of dementia by genetic linkage and functional cell biology studies, but current epidemiological evidence has yet to reach a consensus on their role in dementia risk. This thesis sought to triangulate the causal effects of lipid levels (total cholesterol, LDL-c, HDL-c, and triglycerides) on risk of incident dementia (all-cause dementia, Alzheimer’s disease, and vascular dementia) using existing and new evidence.

**Methods:** Four distinct analyses were conducted. Firstly, a systematic review of 81 published and preprinted studies was used to summarise the existing evidence base. Preprints were searched using a new research tool created as part of this thesis. The existing evidence was then supplemented via two primary studies: i) a cohort study of the association of lipid regulating agents and dementia incidence in the Clinical Practice Research Datalink (CPRD); and ii) an individual patient data meta-analysis of the association of blood lipids with dementia incidence in previously unanalysed cohorts accessed through the Dementia Platform UK. Finally, a novel quantitative triangulation framework was proposed, building on recent developments in risk-of-bias assessment and bias-/indirectness-adjusted meta-analyses. This framework was then used to quantitatively triangulate the evidence identified and produced by this thesis.

**Results:** The systematic review did not identify a consistent effect of blood lipids on any dementia outcome across study designs, through there was some suggestion of a protective effect of LDL-c lowering on all-cause dementia and Alzheimer’s disease in observational studies of statin use. The analysis of lipid regulating agents in the CPRD provided weak evidence for a protective effect of statins on all-cause dementia and Alzheimer’s disease but suggested a harmful association with vascular dementia. However, the use of control outcomes illustrated this finding was likely due to confounding by indication related to vascular factors. The individual patient data meta-analysis suffered from a low response rate to data access requests, but in the three cohorts analysed, there was some evidence only for the association of triglycerides and vascular dementia. Finally, the triangulation analysis integrating the results of the previous three studies did not provide strong evidence for the causal effect of blood lipids on dementia outcomes.

**Conclusions:** This thesis provides new evidence concerning the role of blood lipids as a modifiable risk factor for dementia and highlighted the uncertainty that still remains in relation to this causal question. In addition, it has developed new evidence synthesis methods and tools, specifically around the inclusion of preprints in systematic reviews and the quantitative triangulation of evidence sources.