### Background and Introduction

Our hypotheses that decreased membrane plasmalogen capacity and APOE allele status contributed to increased risk of conversion from NCI to MCI and MCI to AD. This means that we must develop tools that enable the precise and accurate determination of a person’s causative factors in AD. This is a critical step in the development of effective interventions for AD.

### Clinical trial design

- **Purpose:** To assess the effect of plasmalogen on cognition and AD incidence.
- **Participants:** 98 NCI, 159 MCI, and 149 AD cases were enrolled in the longitudinal study. Non-cases were matched to cases on demographic, medical, and lifestyle variables.

### Measuring the Effect of Peroxisomal Function and Output on Cognition and AD Incidence

- **Dependent variables:** Global cognitive function and AD incidence were measured using the Alzheimer Res 2012, 9(6):646 scale.
- **Independent variables:** Peroxisomal function and output were measured using mass spectrometric analyses of serum ethanolamine phospholipids.

### Plasmalogen Metabotype Interactions with Age, Gender and Metabolic Syndrome Biomarkers

- **Plasmalogen metabotypes:** High-risk, medium-risk, and low-risk metabotypes were identified based on their association with AD incidence and cognitive function.
- **Age and gender:** Interactions between plasmalogen metabotypes and age, gender, and metabolic syndrome biomarkers were examined using statistical models.

### Conclusions and Future Work

- **Key findings:** Decreased plasmalogen capacity and increased metabolic syndrome biomarkers were associated with increased risk of AD and decreased cognitive function.
- **Implications:** Developing interventions to increase plasmalogen capacity and reduce metabolic syndrome biomarkers may be a critical step in the prevention and treatment of AD.