multi-linear regression adjusted for age, sex, educational level and apolipoprotein E-e4 genotype. **Results:** The healthy older participants involved in this study had good nutritional and vitamin A statutes. Strong positive correlations between RXRa and between RARa or PPARg and negative correlations between RARg and RARa or RXRa have been highlighted. In fully adjusted models, RARa and RXRa expressions were significantly inversely associated with MMSE, IST and FCSRT performances (Table 1). The PPARg expression was only inversely associated with IST performances. No association between RARg or RXRb and any of the four cognitive tests was evidenced. **Conclusions:** We observed that particular retinoid and fatty acid nuclear receptor expression patterns are associated with lower cognitive performances in humans while the originality of our work prevents us from comparing our study with previous published research. Several lines of evidence have already suggested in animals mechanisms by which retinoid and fatty acids participate in modulation of neurobiological processes that underlie mnemonic abilities while further research is needed to confirm this hypothesis in humans.

P1-231 ROLE OF OLR1 AND ITS REGULATING HSA-MIR369-3P IN ALZHEIMER'S DISEASE: GENETIC AND EXPRESSION ANALYSIS

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Background: LDL Receptor 1 (*OLR1*) gene has been shown to act as risk factor for Alzheimer's disease (AD). **Aims:** 1) To carry out an association analysis of *OLR1* in a population of 443 patients with AD, compared with 391 age-matched controls. 2) To perform an expression analysis of *OLR1* and its regulatory hsa-miR-369-3p in Peripheral Mononuclear Blood Cells (PBMC) from patients and controls. **Methods:** The genetic analysis was performed by allelic discrimination. The expression analysis was done by Real-time PCR. **Results:** A statistically significant increased frequency of *OLR1* rs1050283*C* allele was observed in AD patients compared with controls (46 versus 43%, $P = 0.011$, OR: 1.48, 95% CI: 1.10-2.00). Stratifying according to gender, a statistically increased frequency of *OLR1* rs1050283*C* allele was observed in female AD patients as compared to female controls (51 versus 37%, $P < 0.001$, OR: 2.90, 95% CI: 1.97 - 4.24), but not in males. A significantly decreased relative expression level of *OLR1* in PBMC was observed in AD patients carrying the rs1050283C allele (0.23 ± 0.13 versus 0.92 ± 0.8, $P = 0.04$) as compared with AD patients not carrying the *C* allele. Increased relative expression levels of the hsa-miR-369-3p was observed in AD patients carrying the rs1050283*C* allele, although the significant threshold was not reached (2.23 ± 1.35 versus 1.16 ± 0.31, *POLR1* and hsa-miRNA-369-3p gene expression was found in AD patients carrying rs1050283*C* allele ($r = -0.313$, $P = 0.05$) . **Conclusions:** The *OLR1* rs1050283*C* allele likely acts as a risk factor for sporadic AD; *OLR1* and its transcriptional regulatory factor hsa-miR-369-3p are deregulated in AD patients carrying the rs1050283*C* allele.

P1-232 MATERNAL TRANSMISSION OF ALZHEIMER'S DISEASE

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Background: Some propose maternal Alzheimer's disease (AD) inheritance because patients more frequently have AD, affected mothers than fathers, but this has not been evaluated in controls. The present study compared dementia family histories in AD cases and cognitively normal controls. Because of female longevity, we predicted more mothers than fathers would be affected with AD for both cases and controls. However, if maternal risk was not only due to female longevity, then the maternal/paternal ratio would be significantly higher in AD cases than controls. **Methods:** We matched 196 AD cases to 200 controls by gender and age strata. With family history forms and medical record review, we obtained parent dementia status and age of death for 348 AD and 319 control parents. For the primary analysis we used logistic regression on correlated data. **Results:** Age, gender and parental age at death were not significantly different between groups. ApoE4 occurred in 27% of controls and 64% of cases ($p < 0.001$). 12% of control and 13% of AD case fathers had dementia while 29% of control and 28% of AD case mothers had dementia. Thus, more mothers than fathers had dementia for both AD cases and controls. The statistical significance of the association persisted after adjusting for parent age at death and APOE for controls ($OR = 2.40$, $p = 0.004$) but not cases ($OR = 1.63$, $p = 0.14$), although the results are qualitatively similar. **Conclusions:** Consistent with previous research, more mothers than fathers were affected with AD for both AD cases and controls. However, no difference in the ratio of affected mothers to fathers was noted between the two groups. Mothers of both AD cases and controls outlived fathers. These results do not support the maternal transmission of AD. Rather, the increased number of affected mothers of AD patients is seen as often in controls and likely relates to female longevity.

P1-233 WILLINGNESS TO PAY FOR ALZHEIMER'S DISEASE GENETIC SUSCEPTIBILITY TESTING: RESULTS FROM THE REVEAL STUDY

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Background: Availability of a genetic susceptibility test for Alzheimer's disease (AD) raises questions about what people are willing to pay for this test. We studied willingness to pay for genetic susceptibility testing for AD in the REVEAL Study, a clinical trial of AD risk assessment, including *APOE* disclosure, among first-degree relatives of persons with AD. In secondary analyses, we examined interest in genetic testing if testing were covered by health insurance versus self-pay. **Methods:** We analyzed 276 subjects who participated in *APOE* testing and disclosure for risk of AD. We conducted univariate analyses and multivariate logistic regression to determine factors associated with willingness to have insurance pay, to self-pay and to pay more than $100 as reported 6 weeks after disclosure of AD risk and *APOE* genotype. Pre-test desire to know future AD status (agreeing with, "I would like to know if I am going to develop AD at some point later in my life") and concern (agreeing with, "I am concerned that I will develop AD someday") were evaluated along with demographic factors and *APOE* status as predictor variables. **Results:** Seventy-one percent of participants reported they would request AD genetic testing if covered by insurance versus 60% if by self-pay. Twenty-nine percent would not have testing even if covered by insurance. Forty-one percent would be willing to pay more than $100 for the test; 54% would pay more than $500. Household income above $50,000 (OR 2.97; 95% CI 1.37, 6.45) and desire to know future AD status (OR 3.22; 95% CI 1.52, 6.86) were associated with greater willingness to pay more than $100 for testing. Desire to know future AD status was also associated with greater willingness to self-pay (OR 3.01; 95% CI 1.60. 5.98) and greater willingness to have insurance pay (OR 2.36; 95% CI 1.21, 4.61). Positive *APOE* e4 carrier status (OR 0.44; 95% CI 0.24, 0.81) and years of education (OR 0.84 per year; 95% CI 0.73, 0.96) were associated with lower willingness to have insurance pay. **Conclusions:** These findings reveal that participants generally see value in genetic susceptibility testing for AD, even though results have no proven clinical utility.