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Authors

Ringman, JM

Diaz-Olavarrieta, C

Murrell, J

et al.

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Presenilin-1 Mutation Carriers Attain Less Education Than Their Nonmutation Carrying Kin

John M. Ringman, Irvine, CA, Claudia Diaz-Olavarrieta, Mexico City, Mexico, Jill Murrell, Indianapolis, IN, Yaneth Rodriguez, Mexico City, Mexico, Bernardino Ghetti, Indianapolis, IN, Francisco Paz, Mireya Chavez, Mexico City, Mexico, Claudia Kawas, Irvine, CA

OBJECTIVE: To determine if genetic status in persons at-risk for inheriting a Presenilin-1 (PS1) mutation influences level of educational attainment.

BACKGROUND: There is convergent evidence suggesting that an increased level of intellectual achievement protects one from manifesting the cognitive decline of Alzheimer's disease (AD). These data however, being largely retrospective, are subject to many biases. Considering the well-described genetic contributions to the development of AD, another possible explanation for these associations is that people predisposed to develop AD have a life-long cognitive disadvantage that limits their educational attainment. We studied persons at-risk for the development of autosomal dominant AD to test this hypothesis.

DESIGN/METHODS: Twenty-five persons representing 4 distinct Mexican families at-risk for inheriting a PS-1 mutation were enrolled in a study measuring demographic variables, risk factors, and scores on neurocognitive tests. Subjects were not excluded if they had subjective memory complaints or cognitive decline had been noted by an informant but were excluded if they had experienced functional decline. Mutation status was determined in 16 of these subjects. To reduce cohort effects only subjects between the ages of 20 and 45 were considered. No subject was currently enrolled in school or had plans to continue schooling. Mutation carriers and non-mutation carriers were compared using an independent samples T-tests with regard to age, years of education, self-reported average grades, Mini-Mental State Examination (MMSE) scores, and Beck Depression Inventory (BDI) scores.

RESULTS: Five carriers (3 female, 2 male) of the A431E mutation in PS1 were compared to 6 related non-carriers (all female). Subjects represented 3 superficially distinct families carrying this same mutation. Mean ages were 32 (range, 22–44) for carriers and 36 (range, 23–45) for non-carriers (non-significant, ns). Non-carriers had significantly more years of education than did carriers (16.3 vs. 11.4, $p<.05$) and had slightly better mean self-reported grades (8.5 vs. 7.6 on a scale from 1–10, ns). Mean MMSE scores did not differ between the groups (28.3 in non-carriers, 28.6 in carriers). Mean self-rated depression as measured on the Beck Depression Inventory was higher for the mutation carriers (18.8) than for the non-carriers (5.5) though this difference was not statistically significant ($p=.06$).

CONCLUSIONS: Despite having grossly comparable cognitive function, carriers of a PS1 mutation causing autosomal dominant AD did not achieve as high a level of education as did their non-mutation carrying kin. This is consistent with the hypothesis that persons predisposed to develop AD may have subtle, possibly life-long, cognitive deficits which limit their intellectual achievement. Further study of this group with more rigorous control of other psychological, psychiatric, and socioeconomic variables should help to evaluate the contribution of other factors influencing educational attainment in this population.

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