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'Below average' Self-Assessed School Performance and Alzheimer's disease in the Aging, Demographics and Memory Study

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Abstract

Background: Low formal education level is becoming accepted as a risk factor for Alzheimer's disease (AD). Though increasing attention has been paid to educational quality differences, no prior studies have addressed participants' own characterization of their overall performance in school. We examined whether self-assessed school performance is associated with AD beyond the effects of educational level alone.

Methods: Participants were drawn from the population-representative Aging, Demographics and Memory Study (ADAMS), 2000-2002. ADAMS participants were asked about their performance in school; possible response options were 'above average,' 'average,' or 'below average'. ADAMS

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participants also had a full neuropsychological battery and were given a research diagnosis of possible/probable AD.

Results: The 725 participants (mean age 81.8 years, 59% female, and 16% African-American) varied in their educational performance: 29% reported ‘above average’; 64% ‘average’; and 7% reported ‘below average’ school performance. Participants with lower self-assessed school performance had higher proportions of AD: eleven percent of participants with above average self-assessed performance had AD; 12 percent of participants with ‘average’ performance and 26% of participants with ‘below average’ performance ($p<0.001$). After controlling for subjects' years in school, literacy test score(W-RAT), age, sex, race/ethnicity, and ApoE-ε4 status, socioeconomic status and self-reported comorbidity, respondents with ‘below average’ self-assessed school performance were 4 times more likely to have AD compared to those who had average performance. (OR 4.0; 95% CI 1.2-14) Above average' and ‘average’ self-assessed school performance did not increase or decrease the odds of AD.(OR 0.9; 95% CI 0.5-1.7)

Conclusion: We suggest an association between ‘below average’ self-assessed school performance and AD beyond the known association with formal education. Efforts to increase cognitive reserve through better school performance in addition to increasing the number of years of formal education in early life may be important to reduce vulnerability throughout the life course.

Keywords

Alzheimer's Disease; Education; cognition; race; ethnicity

1. Introduction

Lower formal education is a consistent risk factor for Alzheimer's disease (AD), as summarized by a recent meta-analysis. [1] As early as the 1990s, Zhang and colleagues reported that poorly educated Chinese subjects were more likely to have dementia than those who had higher levels of education. [2] The brain reserve and cognitive reserve hypotheses followed as conceptual models of the mechanism of action. [3-5] Brain reserve refers to an individual's brain structures, including neurons in key areas of the brain related to learning and memory. A common metaphor used for brain reserve is that it is akin to a computer's hardware. Similarly, following this metaphor, cognitive reserve is the computer's software, wherein the reserve is developed over early life. Education and other early-life experiences may influence the reserve built for each individual. Those who have higher reserve may be able to function longer without reaching a threshold of cognitive impairment compared to people with lower reserve. This is an explanation for why clinical symptoms may appear earlier for people with low formal education and other different contextual experiences (e.g., low early linguistic ability, early IQ), compared to people with higher formal education. Despite this vast array of research, evaluating education using years of formal education may not be sufficient.

Less is known about educational quality differences and risk for AD. For example, in one study, cognitive function test performance for non-demented African Americans was significantly lower compared to whites, with matched educational level. [6] Further work by this group has suggested that quality of education may be a better measure of cognitive reserve than education alone. [7] Literacy, another potential measure of educational quality/reserve, is linearly associated with cognitive function [8] and potentially mediates race/ethnic differences in cognition. [9]

Educational quality has previously been defined broadly as having desirable characteristics of learners (healthy, motivated students), processes (competent teachers using active pedagogies), content (relevant curricula) and systems (good governance and equitable resource allocation). [10] A potential indicator of a healthy, motivated learner is perhaps the individual's self-rating

of performance while in school. Despite this, no studies to our knowledge have examined the relationship between an individual's assessment of how he or she performed in school (self-assessed school performance) and cognitive impairment or AD. Prior studies on performance have not addressed whether self-assessed school performance is essentially different from level of formal education received, level of literacy, or low socioeconomic status (SES) in conferring risk of Alzheimer's disease. This study addresses this gap by examining data from the Aging, Demographics and Memory Study, a detailed study of dementia. We examined whether self-assessed school performance is an independent risk factor for Alzheimer's disease.

2. Methods

2.1 Setting

The Health & Retirement Study (HRS) is a cohort study of US adults 50 years and above that has collected data from 1992 to the present. As the primary purpose of this study is to examine the dynamics of older adults' income and wealth, it included detailed measures of socioeconomic status and self-reported health conditions. The HRS sample is derived from a multi-stage, clustered area probability frame of over 60,000 housing units (for adults aged 50-64) and Medicare enrollment lists (for adults 65 years and above); there were 11,454 respondents in the 2000 wave of HRS and 10,321 respondents in the 2002 wave of HRS age 70 and above.

A complementary study, the Aging, Demographics and Memory Study (ADAMS), was added to the parent HRS to examine dementia. [11] The ADAMS study identified 7,000 respondents aged 70 and above from the 2000 and 2002 HRS waves eligible for the ADAMS study. [12, 13] A stratified sample of 1,770 individuals were selected for eligibility in the ADAMS study based on low to high cognitive function performance. [14] Of 1,770 selected individuals, 856 completed initial assessment,[11,15,16] 227 died before the initial assessment could be completed and 687 subjects (39%) refused or did not participate.

2.2 Participants

A nurse and a neuropsychology technician conducted an in-person 3.5 hour evaluation with each of the 856 participants and with an informant familiar with each participant's medical history. A multidisciplinary conference (geropsychiatrist, neurologist, neuropsychologist and cognitive neuroscientist) reviewed information and assigned a research diagnosis. For dementia diagnoses, the criteria were based on the DSM-III-R, DSM-IV and NINCDS-ADRDA. Of the 856 ADAMS participants, 131 participants had non-Dementia neurological diagnoses and were thus excluded from the current study (e.g., Parkinson's, Huntington's, etc.). For the current study, we primarily focus on the remaining 725 individuals broken down into four relevant groups: 229 with Alzheimer's disease; 71 cases of non-AD dementia (primarily vascular dementia); 118 with cognitive impairment (mild cognitive impairment, prodromal Alzheimer's, not meeting criteria for AD, and cognitive impairment with cause unknown); and 307 who were considered to have normal cognitive functioning). Our primary objective was to contrast the Alzheimer's disease group(229) to those with normal cognitive functioning (307). Other contrasts were made as sensitivity analyses. The current study was approved by the institutional review board at the University of California, San Francisco and the San Francisco VA Medical Center.

2.3 Measures derived from the ADAMS study

Information on education and educational performance was derived from the ADAMS study from 2001-2003, including highest level of formal education received. In addition, our central measure was the question “How well did you perform in school?” with the response choices ‘average’, ‘above average’, ‘below average’. We focused on this question because it is a general

measure, and we felt that asking for more detailed recollections of past performance, such as asking for their exact Grade Point Average (GPA), would make too many demands on memory, while asking too little would not provide the variance necessary to have a good predictor variable. We also queried whether the respondent had ever failed a subject, and whether the respondent was ever held back, given special instruction, or tutored. Literacy was assessed using the Wide Range Achievement Test (WRAT-3), a measure of reading/ literacy in the elderly, which has good test-retest reliability.[17] Respondents with the genetic risk factor Apolipoprotein E-ε4 (either 1 or 2 alleles) were compared to those without the allele.

2.4 Other Measures

The following measures were obtained from the Health and Retirement Study. Race was based on self-report and was categorized into the following groups: White; African American; or Other race. Ethnicity was categorized into Hispanic and Non-Hispanic. For the purposes of this study, race/ethnicity was divided into three mutually exclusive groups: White, not Hispanic (White), African American, not Hispanic (African American), and Hispanic of any race (Latino). Current socioeconomic status was measured by total household income, and total net worth or current value of all assets including individual retirement accounts, stocks, or mutual funds, checking and savings, and real estate less all debt in 2000. Separate indicator variables were calculated for each chronic condition if the participant had a self-reported medical history of high blood pressure, diabetes mellitus, heart disease and stroke in 2000. Early childhood socioeconomic status (SES) was evaluated using four indicators, [18] parent with highest level of education (<8 years, 8-11 years or 12 or more years), self-reported perception of childhood family financial status as “poor”(family poverty), having to move during childhood because of financial difficulties (family hardship) or self-report that the respondent never lived with their father or the father was not alive during their childhood (father absent). Depressive symptoms were assessed in 2000 using a modified version of the Centers for Epidemiologic Study Depression Scale. [19]

2.5 Statistical analyses

Characteristics were compared for participants with ‘below average’, ‘average’ and ‘above average’ self-assessed school performance using chi-squared tests and t-tests where appropriate. The association between self assessed school performance and AD was calculated using age-adjusted logistic regression models. We evaluated the outcome in three ways: 1) contrasting AD patients (229) with the reference group of participants with normal cognitive status (307); 2) contrasting all cause dementia (AD participants and non-AD dementia participants combined 300) with participants with normal cognitive function (307); and 3) calculating an ordinal logistic regression model with three groups: Alzheimer's disease (229), cognitive impairment (118) and normal cognitive status (307).

We used sequential adjustment of potential confounders according to adjustment blocks approximating timing of risk factors over the life course. First, demographics and genetics (gender, race and presence of ApoE-ε4) were added to the age-adjusted model. To assess whether level of formal education mediated the relationship between educational performance and AD, this factor was added to the model adjusting for age, demographics and genetics. Then, socioeconomic markers (either childhood or current) were added to the previous model; they were parents' educational level in years completed, family poverty experienced in childhood (yes/no), family hardship experienced in childhood (yes/no) and whether the father was absent, respondent's current income, and total current value of assets including individual retirement accounts, stocks, or mutual funds, checking and savings, and real estate. An additional adjustment was for self-reported comorbidities: high blood pressure, diabetes, heart disease and stroke. The last adjustments were for literacy score as measured by the WRAT, and, separately, an adjustment for depressive symptoms as measured by the CES-D scale.

We tested potential interactions between key risk factors and self-assessed school performance, such as: 1) race/ethnicity; 2) gender; 3) median educational level; and 4) ApoE-ε4 allele presence. Each interaction was tested in a model additionally adjusted for age. All analyses were calculated in two ways, unweighted and utilizing study weights. The weighted analyses utilized survey procedures in SAS 9.1 in order to account for the ADAMS and HRS studies' complex survey design. We applied three weighting variables using SAS survey procedures: the stratum variable (sestrat) was used to account for the sampling stratum, a cluster variable (seclust) was used to account for the clustering of the data, and a sample weight (aasampwt) was used to account for differing sampling probabilities due to oversampling of some groups. As 36 participants used a proxy informant to answer the question regarding self-assessed school performance, we performed all analyses again excluding these 36 participants.

3. Results

The 725 participants in the ADAMS study were on average 81.8 years old and were more often female (59%). Sixteen percent of the older adult participants were African American, 8% were Latino and the rest were White. Twenty-nine percent had one or two Apolipoprotein E-ε4 alleles. Subjects with ‘below average’ self-assessed school performance were younger and were made up of a higher proportion of men and non-White race/ethnicity groups. In terms of comorbidities and socioeconomic markers, those with ‘below average’ self-assessed school performance had similar comorbidities and had more socioeconomic burden compared to participants with average and above average self-assessed school performance.(Table 1)

The median level of education was 12.0 years. Overall, 7% of subjects reported that they had ‘below average’ school performance, 64% reported ‘average’ school performance and 29% reported ‘above average’ performance. Of the participants who reported that they had failed a subject in school 11% reported ‘above average’ 21% reported ‘average’ and 52% reported ‘below average’ school performance, $p<0.001$. Of the participants who reported they had been held back, given special instruction, or tutored, 10% percent reported they had ‘above average’ school performance, 16% reported ‘average’ school performance, and 26% reported ‘below average’ school performance ($p<0.001$).

Participants with lower self-assessed school performance had higher proportions of AD: eleven percent of participants with ‘above average’ self-assessed performance had AD; 12 percent of participants with ‘average’ performance and 26% of participants with ‘below average’ performance ($p<0.001$). A report of ‘below average’ school performance was associated with Alzheimer's disease, adjusted for age.(Age-adjusted Odds Ratio=OR 5.9; 95% Confidence Interval(CI) 2.0-18) This relationship remained after controlling for demographic characteristics and presence of the genetic risk factor ApoE-ε4.(OR 6.7; 95%CI 2.2-21) This result was slightly attenuated after additional adjustment for years of formal education attained (OR 4.8; 95% CI 1.5-16) and after additional adjustment for current and childhood socioeconomic indicators and comorbidity.(OR 4.2; 95% CI 1.2-15) (Table 2) We additionally adjusted for the W-RAT, a measure of literacy. After addition of W-RAT, the results were further slightly attenuated.(OR 4.0; 95% CI 1.2-14) In the fully adjusted model, formal education attained was also associated with AD, with each year in school associated with a 15% reduced odds of Alzheimer's disease.(OR 0.84; 95% CI 0.76-0.92). However, when we additionally added premorbid depression score as measured by a modified version of the CES-D in 2000, results were significantly attenuated for ‘below average’ self-assessed school performance, results not in table (OR 3.7; 95% CI 0.8-17).

‘Above average’ educational performance was associated with similar risk of Alzheimer's disease as compared to those with ‘average’ performance, after adjustment.(OR 0.7; 95% CI 0.4-1.2) Similar results were seen after additional adjustment for level of formal education

attained and socioeconomic indicators. If a participant reported that they had failed a subject in school, reported being held back, were given special instruction, or tutored in school, they were not at increased odds of Alzheimer's disease either alone or after adjustment.(results not shown) As few participants had non-AD dementia and cognitive impairment, we undertook two analyses using combined outcomes. Participants reporting ‘below average’ self-assessed school performance had a similar increased odds of all-cause dementia.(AD and non-AD dementia combined).(OR 4.3; 95% CI 1.4-13) Participants reporting ‘below average’ self-assessed school performance had increased odds of cognitive impairment, not meeting the criteria for dementia. However, there was limited power for this analysis (OR 2.6 95% CI 1.3-4.9). (Table 3) Model fit was adequate for all models; the c-statistic ranged from 0.85-0.89.

In order to understand whether the noted results differed for particular subgroups, we calculated four possible interactions: sex (men/women), apoE-ε4 allele (present/absent), formal educational level (12 years or more/less than 12 years), and race/ethnicity (non-Hispanic White compared to African American and Latino). No significant interaction was present for sex and self-assessed school performance (p=0.51), ApoE-ε4 presence and self-assessed school performance (p=0.28), formal educational level and self-assessed school performance (p=0.07) and race/ethnicity and self-assessed school performance.(p=0.10)

As 36 of the respondents had proxy informants, all of the above analyses were calculated excluding these 36 participants. The conclusion of all analyses remained the same with the exclusion of these participants, thus we report all models including these 36 participants.

4. Discussion

Our result extends the well-established inverse relationship between formal education and Alzheimer's disease. This study suggests that ‘below average’ self-assessed school performance is associated with a four-fold increased odds of Alzheimer's disease after accounting for the known effects of formal education and literacy. In addition, these findings remained after adjustment for several key factors, including Apolipoprotein E-ε4 presence, childhood and current socioeconomic indicators, and self-reported comorbidities. Our results also show that the relationship between ‘below average’ self-assessed school performance and Alzheimer's disease is similar for vulnerable groups including participants with low levels of formal education and who are of non-White race/ethnicity.

Several important studies have linked educational attainment in young adulthood and late life risk of Alzheimer's disease, supporting the brain reserve and cognitive reserve hypotheses reserve hypotheses. [2-5] Brain reserve is a concept that individuals begin with brain structures including neurons in key areas of the brain related to learning and memory. A common metaphor used for brain reserve is that it is akin to a computer's hardware. Similarly, following this metaphor, cognitive reserve is the computer's software, wherein the reserve is developed over early life, and education and other early-life experiences may influence the reserve built for each individual. Those who have higher reserve may be able to function longer without reaching a threshold of cognitive impairment compared to people with lower reserve. Educational level has been used to approximate brain and cognitive reserve as it modified the association between neuritic plaques and cognitive function in the Religious Orders Study. [20] Further work suggests that literacy, another potential measure of educational quality/ reserve [21] is linearly associated with cognitive function [8] and potentially mediates race/ ethnic differences in cognition. [9] Though our data do not directly speak to this issue, we believe that self-assessed school performance may have its impact on increasing cognitive reserve (i.e., it will provide strategies to help delay the onset of clinical symptoms of AD without impacting the neuro-pathological course of AD).

Few studies have examined metrics of educational performance. In childhood, educational performance is linked to poor health. [22] To our knowledge, there have been no studies reported in the medical literature that correlate self-reported school performance in old age with actual performance in younger ages. In older age, overall self-assessed school performance has not been studied per se, but related constructs have been studied. For example, idea density at age 20 was associated with decreased risk of Alzheimer's disease in older adulthood; as these subjects were part of a religious order (nuns), these findings may not be generalizable. [23] In another study, higher Public school IQ scores in 1944-46 were associated with 50% decreased odds of a combined outcome of MCI and dementia 60 years later [24]; a similar study in Scotland suggested that the mean mental ability scores at age 11 were lower in patients who developed dementia compared to age-matched controls. [25] The latter two studies were limited because they were restricted to performance on one test in early life and were limited to defined geography. Our findings extend this prior work, suggesting that a more general metric, that is, report of 'below average' educational performance, may be yet another risk factor for AD beyond the effects of educational performance, mental ability/IQ, or linguistic ability – but all are probably related to or are markers for cognitive reserve in early life.

A recent review has summarized early life risk factors and Alzheimer's disease and found varying results. [26] The strongest associations between early life socioeconomics and AD were found in people with the ApoE-ε4 allele. [27,28] We did not find a stronger effect of 'below average' self-assessed school performance in ApoE-ε4 allele carriers. Moreover, adjusting for early life or late life socioeconomic indicators did not alter our results appreciably. This suggests that the effects of 'below average' educational performance are independent of poor socioeconomic conditions, and possibly signifies that educational performance captures vulnerability beyond resource dissolution alone.

Prior studies suggest that educational quality may vary by race/ethnicity. Previous studies have suggested that race differences in performance on cognitive function tests exist after matching on educational level. [21,29] Our evaluation of an interaction between race/ethnicity and self-assessed school performance suggests that the effects of 'below average' educational performance are similar for African Americans compared to Whites. This may be due to the fact that African Americans of this generation may have had different educational quality opportunities as compared to White adults of the same generation. [30-35] Considering the strength of this self-report of school performance with AD, we suggest that further studies, particularly in diverse race ethnic groups, evaluate a simple self-report of educational performance as a good surrogate marker for cognitive reserve when other markers of childhood performance are unavailable. One possible explanation for the mechanism by which self-assessed school performance and later dementia may be through self stereotype or stereotype threat. [36] If one believes they are "not academic" they may consequently avoid cognitively demanding tasks (or at least activities generally perceived to be demanding) or possible skill development in the future. Other possible mechanisms may that a related risk factor, such as pre-school cognitive skills or depressive symptoms influence both actual or assessed school performance and later AD risk. [37] We believe that depressive symptoms and assessing school performance may be related concepts and that depressive symptoms is not a simple confounder or mediator in this instance.

Some limitations of our study deserve comment. Perhaps the strongest source for potential bias is the reliance on self-report of educational performance and the limited number of respondents who reported 'below average' educational performance. As a result, the confidence intervals for these estimates are wide. Subjects themselves may not recall their own school performance. However, there is some evidence that long-term memories are preserved in early stages of dementia. For 36 of the respondents in this study, proxies provided information, it is possible

that they inadequately reported school performance or the respondent's level of education. For those who had proxy reports, it is important to include them, so that even older adults with advanced frailty and cognitive impairment might be included in studies [38,39]; in our study, only 36 proxy informants answered the question on school performance. When their responses were excluded, the effect of 'below average' school performance remained the same. Additionally, the question "How well did you perform in school" is subjective in its interpretation; some individuals may focus on one set of skills (verbal ability, math, biology) instead of others. Differential item functioning may occur, whereby the question is interpreted differently in people of different groups – in this case, possibly race/ethnic groups. We were limited by power to examine other types of dementia or cognitive impairment separately. As the follow up of the ADAMS study was not complete, we cannot examine to what extent our findings are affected by ascertainment bias. Lastly, perhaps at play is "cognitive dissonance" wherein the participants who have poor economic outcomes in adulthood attribute it to poor performance in school in early-life. As our measure, self-assessed school performance, has not been extensively validated, future studies should assess the validity of this measure and link it to actual performance in school. Additional future studies could examine self-assessed school performance at the individual level are associated with objective measures of school quality at the level of the school.

In summary, this study suggests that 'below average' self-assessed school performance is associated with increased odds of Alzheimer's disease in late-life. As this is a preliminary study, further study of self-assessed school performance is suggested. Efforts to increase cognitive reserve early in life and throughout the life course are important, not only in themselves, but also as a possible protective mechanism to stave off the onset of Alzheimer's disease in the future. Thus, not only is 'staying in school' important, but increasing school performance is equally important to stave off the potential long-reaching effects of low educational quality.

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Table 1

Characteristics of Participants in the Aging, Demographics and Memory Study (n=725).

Variables	Total Cohort * (n=725)	Above Average (n=189, 29%)	Average (n=424, 64%)	Below Average (n=46, 7%)
Demographics				
Age (mean years, SD)	81.8 (7.2)	81.6 (7.1)	81.5 (7.1)	80.1 (6.8)
% Female	59	60	61	35
Race				
% African American	16	12	16	37
% Latino	8	7	8	11
Genetic Risk Factor				
% with ApoE-ε4	29	32	28	26
Educational Markers				
Education Level (mean years, SD)	10.3 (4.3)	12.2 (4.0)	10.3 (3.7)	7.5 (3.5)
Educational Level (median, range)	12.0 (0-17)	12.0 (0-17)	11.0 (0-17)	7 (0-17)
Wide Range Achievement Test Reading recognition Subtest score (mean, SD)	55 (44)	55 (43)	53 (44)	56 (43)
Failed a subject	20	11	21	52
% Held back or tutored	15	10	16	26
Center for Epidemiologic Depression Scale (mean, SD)	1.9 (2.0)	1.6 (1.9)	1.9 (1.9)	2.7 (2.4)
Comorbidities (self-reported)				
% high blood pressure	53	48	56	49
% diabetes mellitus	17	18	16	27
% heart disease	29	27	29	38
% stroke	17	19	16	13
Childhood SES				
US Region of Birth **				
South	45	39	47	56
West	11	8	12	19
Midwest	27	33	25	16
Northeast	17	20	16	9
Parent with the highest education level (mean, SE)	8.2 (2.5)	8.6 (2.5)	8.3 (2.5)	7.8 (1.8)
(%) Family poor	38	38	37	47
(%) Family hardship	25	30	22	25
(%) Father absent	10	4	11	19
Current SES				
Respondents' income (in dollars, SD)	2102 (17900)	5167 (33876)	1153 (5525)	1176 (4773)
Current assets (in dollars, SD)	268111(56782)	456071 (910460)	223191 (376336)	138049(340903)

* 66 Participants with missing information on educational performance excluded

** 61 Participants were missing information on region of birth

Table 2

Educational Performance and Alzheimer's Disease in the ADAMS Study with a Series of Adjustments (Odds Ratios and 95% Confidence Intervals).

Group Educational Performance	% Alzheimer's Disease	Age-adjusted Odds Ratio (95% CI)	Demographics and ApoE-ε4 allele presence* Odds Ratio (95% CI)	+ Educational level in years † Odds Ratio (95% CI)	+ Childhood SES, current SES, and comorbidities‡ Odds Ratio (95% CI)	+Literacy (W-RAT score)** Odds Ratio (95% CI)
Below average (n=46)	26	5.9 (2.0-18)	6.7 (2.2-21)	4.8 (1.5-16)	4.2 (1.2-15)	4.0 (1.2-14)
Above average (n=189)	11	0.8 (0.5-1.3)	0.7 (0.4-1.2)	0.9 (0.5-1.6)	0.9 (0.5-1.8)	0.9 (0.5-1.7)
Average (n=424)	12	1.0	1.0	1.0	1.0	1.0

* Model adjusted for age, age-squared, sex, race and apolipoprotein E – ε4 presence

† Model additionally adjusted for respondent's level of education

‡ Model additionally adjusted for childhood SES (parents' highest level of education in years, family poverty (yes/no), family hardship (yes/no) or whether the father was absent) ; current SES (respondents' income and total current assets); and comorbidities (self-reported medical history of high blood pressure, diabetes mellitus, heart disease, and stroke).

** Model additionally adjusted for Wide Range Achievement Test (W-RAT) Score

Table 3

Educational Performance and Alzheimer's Disease, All-Cause Dementia and Cognitive Impairment in the ADAMS Study (Odds Ratios and 95% Confidence Intervals).

Models	Predictors	Group	Possible/Probable Alzheimer's Disease (229 PPAD/307 normal) Odds Ratio (95% CI)	All Cause Dementia (300 Dementia/ 307 normal) Odds Ratio (95% CI)	Cognitive Impairment (Ordinal outcome with 118 CI/229 AD/307 normal) Odds Ratio (95% CI)
Model 1	Educational Performance	Below average (n=46)	4.0 (1.2-14)	4.3 (1.4-13)	2.6 (1.3-4.9)
		Above average (n=189)	0.9 (0.5-1.7)	0.9 (0.5-1.6)	1.0 (0.7-1.6)
		Average (n=424)	1.0	1.0	1.0

*Models adjusted for age, age-squared, sex, race, apolipoprotein E – ε4 presence, respondent's level of education, childhood SES (parents' highest level of education in years, family poverty (yes/no), family hardship (yes/no) or whether the father was absent) ; current SES (respondents' income and total current assets); comorbidities (self-reported medical history of high blood pressure, diabetes mellitus, heart disease, and stroke); and literacy as assessed by the Wide Range Achievement Test (W-RAT) Score