



ELSEVIER

Contents lists available at ScienceDirect

Data in Brief

journal homepage: www.elsevier.com/locate/dib

Data Article

Data from a cross-sectional study on Apolipoprotein E (*APOE-ε4*) and snoring/sleep apnea in non-demented older adults



Angeliki Tsapanou^{a,d,*}, Nikolaos Scarmeas^{a,c,d}, Yian Gu^{a,b},
Jennifer Manly^{a,b,c}, Nicole Schupf^{a,b,c}, Yaakov Stern^{a,b,c},
Sandra Barral^b

^a Cognitive Neuroscience Division, Columbia University Medical Center, New York, USA

^b The Gertrude H. Sergievsky Center, Columbia University Medical Center, New York, USA

^c The Department of Neurology, College of Physicians and Surgeons, Columbia University Medical Center, New York, USA

^d National and Kapodistrian University of Athens Medical School, Greece

ARTICLE INFO

Article history:

Received 8 September 2015

Received in revised form

16 September 2015

Accepted 16 September 2015

Available online 30 September 2015

Keywords:

Apolipoprotein E

Snoring

Sleep apnea

Elderly

ABSTRACT

In the present data, we provide the details of the cross-sectional study, from the Washington Heights-Inwood Community Aging Project (WHICAP) that examined the association between Apolipoprotein E (*APOE-ε4*) and snoring/sleep apnea. A total of 1944 non-demented older adults constituted our sample. Sleep dysfunction was measured using sleep categories derived from the Medical Outcomes Study Sleep Scale. Stratified analyses were conducted in order to examine the association between *APOE-ε4* and sleep variables by ethnic group. For further analyses and enhanced discussion, see “Examining the association between Apolipoprotein E (*APOE*) and self-reported sleep disturbances in non-demented older adults” by Tsapanou et al. (2015) [1].

© 2015 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

DOI of original article: <http://dx.doi.org/10.1016/j.neulet.2015.08.037>

* Correspondence to: Cognitive Neuroscience Division, Department of Neurology, Columbia University College of Physicians and Surgeons, PH18-326622, West 168th St, New York, NY 10032, USA.

E-mail address: at2859@cumc.columbia.edu (A. Tsapanou).

<http://dx.doi.org/10.1016/j.dib.2015.09.014>

2352-3409/© 2015 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

Specifications Table

Subject area	Neuropsychology, Neurology, Genetics
More specific subject area	Neurogenetics
Type of data	Questionnaire, Tables
How data was acquired	Quantitative data of 1944 non-demented elderly, using a self-reported sleep questionnaire and their <i>APOE-ε4</i> status
Data format	Raw data, Analyzed
Experimental factors	<i>APOE-ε4</i> and sleep questions were the main variables used. Age, sex, ethnicity, years of education, and body mass index were used as covariates.
Experimental features	Characterization of the <i>APOE-ε4</i> status
Data source location	New York, New York, USA
Data accessibility	Data is in this article

Value of the data

- Identifying the sleep measures associated with *APOE-ε4*.
- Providing novel information about the differences among different ethnic groups.
- Providing the frequencies of the *APOE* alleles in a large sample of older adults.

1. Data, experimental design, materials and methods

In the present data, we present the details of the cross-sectional study that examined the association between Apolipoprotein E (*APOE-ε4*) and snoring/sleep apnea in a large group of older adults. We provide the demographic characteristics of our sample, the frequencies of the *APOE* alleles in our population, the sleep scale we used, and the results of the association analyses between *APOE-ε4* and sleep variables stratified by ethnic group [1].

1.1. Sample collection

Participants were drawn from the Washington Heights-Inwood Community Aging Project (WHICAP) at Columbia University Medical Center [2,3]. WHICAP is a community based research study designed to identify risk factors and biomarkers for aging and Alzheimer's disease in a multi-ethnic cohort that includes Whites, African-Americans, Caribbean-Hispanics, and Other [4]. All participants were over 65 years old, and non-demented at the time of the evaluation. For details, see supplementary Table 1.

1.2. Sleep measures

Sleep quality was assessed using the Sleep Scale from the RAND Medical Outcomes Study. This scale is a self-report 12-item questionnaire [5,6]. Each of the questions has a possible rating of 0–6, based on the frequency of the sleep problem. Using the sleep questionnaire manual [6], we used the five clustered sleep categories to define our analyses phenotype: 1. Sleep disturbance, 2. Snoring, 3. Sleep short of breath/awaking with a headache, 4. Sleep adequacy, and 5. Daytime somnolence. Additionally, categories 2 and 3 were combined into a single variable 'sleep apnea' (see Supplementary Fig. 1).

1.3. APOE genotyping

WHICAP participants were APOE genotyped as previously described [7]. APOE genotypes were transformed into a dichotomous trait based on the number of APOE- ϵ 4 alleles: 0 if the individual does not carry any copy of the ϵ 4 allele (non- ϵ 4 carriers) or 1 if the individual carries 1 or 2 copies of the ϵ 4 allele (ϵ 4 carriers). Carriers of ϵ 2 ϵ 4 alleles were not included in the initial sample due to the opposite effect of these two alleles [8–10]. More details about the APOE genotyping can be found in previous studies [7]. For the frequencies of the APOE alleles, see supplementary Table 2.

1.4. Analysis

All statistical analyses were performed using SPSS 22 (SPSS, Chicago, Illinois). Nominally significant p values were defined as $p < 0.05$. Unadjusted Linear Regression Analyses: We used linear regression models with APOE- ϵ 4 at baseline evaluation as the predictor and the previously described sleep score as the independent variable. Adjusted Linear Regression Analyses: Secondary analyses were performed adjusting for: age, sex, ethnicity, education, and body mass index (BMI). Ethnicity was ascertained based on self-report using the format of the 1990 census [11]. Participants were then assigned to one of the three groups: White, African-American, and Caribbean-Hispanic. To further examine any possible differences among the ethnic groups, we stratified the sample and perform analyses within each ethnic group independently (Whites $n = 431$, African Americans $n = 465$, and Caribbean-Hispanics $n = 1048$) (see Supplementary Table 3).

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:<http://dx.doi.org/10.1016/j.dib.2015.09.014>.

References

- [1] A. Tsapanou, et al., Examining the association between Apolipoprotein E (APOE) and self-reported sleep disturbances in non-demented older adults, *Neurosci. Lett.* 606 (2015) 72–76.
- [2] J.J. Manly, et al., Implementing diagnostic criteria and estimating frequency of mild cognitive impairment in an urban community, *Arch. Neurol.* 62 (11) (2005) 1739–1746.
- [3] M.X. Tang, et al., Incidence of AD in African-Americans, Caribbean Hispanics, and Caucasians in northern Manhattan, *Neurology* 56 (1) (2001) 49–56.
- [4] M.X. Tang, et al., The APOE-epsilon4 allele and the risk of Alzheimer disease among African Americans, whites, and Hispanics, *JAMA* 279 (10) (1998) 751–755.
- [5] R.D. Hays, et al., Psychometric properties of the medical outcomes study sleep measure, *Sleep Med.* 6 (1) (2005) 41–44.
- [6] K.H. Spritzer, R.D. Hays, *MOS Sleep Scale: A Manual for Use and Scoring, Version 1.0*, Los Angeles, CA, 2003.
- [7] R. Mayeux, et al., Synergistic effects of traumatic head injury and Apolipoprotein-epsilon4 in patients with Alzheimer's disease, *Neurology* 45 (3 Pt. 1) (1995) 555–557.
- [8] G. Berge, et al., Apolipoprotein E ϵ 2 genotype delays onset of dementia with Lewy bodies in a Norwegian cohort, *J. Neurol. Neurosurg. Psychiatry* 85 (11) (2014) 1227–1231.
- [9] R. Cacabelos, The application of functional genomics to Alzheimer's disease, *Pharmacogenomics* 4 (5) (2003) 597–621.
- [10] E.H. Corder, et al., Gene dose of Apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families, *Science* 261 (5123) (1993) 921–923.
- [11] N.L. Barclay, et al., The heritability of insomnia progression during childhood/adolescence: results from a longitudinal twin study, *Sleep* 38 (1) (2015) 109–118.