

## Reference

[1] Paller KA, Creery JD, Florczak SM, Weintraub S, Mesulam M-M, Reber PJ, et al. Benefits of mindfulness training for patients with progressive cognitive decline and their caregivers. *Am J Alzheimer's Dis Other Dementias* [Internet] 2014 Aug 25 [cited 2014 Nov 6].

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## EW0193

### Economic recession and mental health distress: Does age matter?

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**Introduction** The association between economic crises and mental health problems can be attributed to a number of factors. Among these, age seems to be an important determinant.

**Objectives** The aim of this study was to assess whether mental health of the Portuguese population following the onset of the 2008 recession, differs by age groups.

**Methods** A follow-up study (2015) on the population aged 18 to >65 years old, using the National Mental Health Survey ( $n=911$ ). The age-group prevalence of mental health distress assessed by the ten-item Kessler's Psychological Distress Scale (K10) was calculated using  $\chi^2$  statistics and mental distress as a categorical variable ( $P<0.05$ ).

**Results** Mean mental distress score differed significantly according to age group,  $\chi^2(3)=10.684$ ,  $P<0.05$ . The results showed that the older groups (50–64 and 65+ years old) were more frequently under mental distress (17–19%) compared to younger people (18–49 years old), which were less likely to report being distressed (8–12%).

**Conclusions** Age seems to be an important determinant of distress levels during the economic crisis in Portugal. Older adults reported to be more distressed compared to younger individuals. There are several hypotheses for a differential expression of psychological distress between age groups such as working status and retirement, which can express differential access to coping resources under such contextual negative pressure of economic recession. Further research on age groups is thus needed to better understand how recession generates adverse effects on mental well-being.

**Keywords** Distress; Age; Mental health; Recession; Older adults  
**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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## EW0194

### The effect of apolipoprotein E $\epsilon 4$ (APOE E4) on visuospatial working memory in healthy elders and amnesic mild cognitive impairment patients: An event-related potentials study

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**Introduction** Previous studies provided inconsistent evidences for the effect of apolipoprotein E  $\epsilon 4$  (APOE  $\epsilon 4$ ) status on the visuospatial working memory (VSWM). Our study was the first investigation with event-related potential (ERP) to explore the effect of APOE  $\epsilon 4$  on VSWM in healthy elders and aMCI patients.

**Objective** The aim was to investigate the effect of APOE  $\epsilon 4$  on VSWM with event-related potential (ERP) study in healthy elders and aMCI patients.

**Methods** Thirty-nine aMCI patients (27 APOE  $\epsilon 4$  non-carriers and 12 APOE  $\epsilon 4$  carriers) and 43 their matched control (25 APOE  $\epsilon 4$  non-carriers and 18 APOE  $\epsilon 4$  carriers) performed an N-back task, a VSWM paradigm that manipulated the number of items to be stored in memory.

**Results** Our study detected reduced accuracy and delayed mean correct response time in aMCI patients than healthy elders. P300 was elicited by VSWM and its amplitude was lower in aMCI patients at the central-parietal and parietal electrodes than healthy controls. In healthy elders, P300 amplitude declined prior to task performance change in APOE  $\epsilon 4$  carriers than non-carriers. Regarding aMCI patients, P300 amplitude result revealed exacerbated VSWM deficits in APOE  $\epsilon 4$  carriers than APOE  $\epsilon 4$  non-carriers. Additionally, standardized low-resolution brain electromagnetic tomography analysis (s-LORETA) result showed enhanced brain activation in right parahippocampal gyrus during P300 time range in APOE  $\epsilon 4$  carriers than non-carriers in aMCI patients (Fig. 1, Tables 1 and 2).

**Conclusions** It demonstrated that P300 amplitude might serve as a biomarker for recognizing aMCI patients and contribute to early detection of worse VSWM in APOE  $\epsilon 4$  carriers than non-carriers.

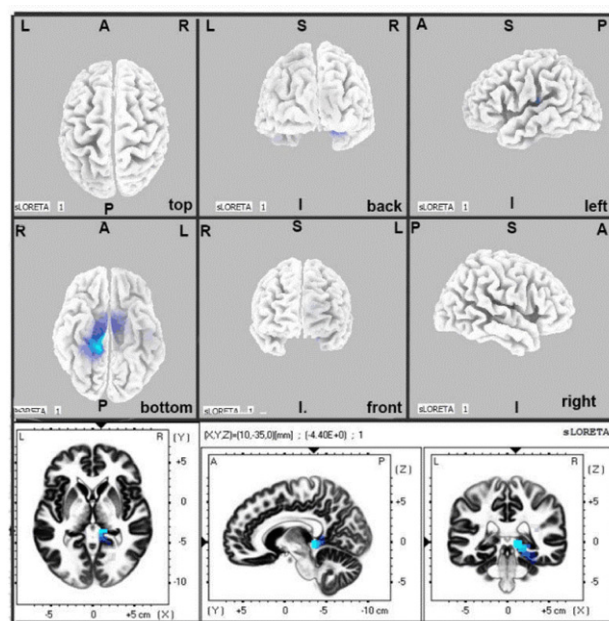


Fig. 1 The sLORETA images showing statistical differences between aMCI- APOE  $\epsilon 4$ - and aMCI- APOE  $\epsilon 4$ + group (3D-view and slice-view) in the P300 time-range. The three slice-view images below located the maximal difference between aMCI- APOE  $\epsilon 4$ - and aMCI- APOE  $\epsilon 4$ + group (MNI coordinates  $x, y, z=10, -35, 0$ ). Negative difference was in blue color with reference of aMCI- APOE  $\epsilon 4$ + group. Abbreviations: aMCI: amnesic mild cognitive impairment; APOE: apolipoprotein E; MNI: Montreal Neurological Institute; sLoreta: standardized low-resolution brain electromagnetic tomography analysis.

**Table 1** Behavioral data (accuracy and response time) for healthy controls and a MCI patients with different APOE ε4 status.

Condition	Stimulus	HC		aMCI	
		APOE ε4 - (n = 25)	APOE ε4 + (n = 18)	APOE ε4 - (n = 27)	APOE ε4 + (n = 12)
<b>Accuracy</b>					
0-back	Non-Target	0.95 (0.04)	0.91 (0.07)	0.90 (0.08) <sup>a</sup>	0.86 (0.13)
	Target	0.88 (0.11)	0.83 (0.08)	0.78 (0.18)	0.72 (0.14) <sup>a</sup>
1-back	Non-Target	0.82 (0.10)	0.77 (0.09)	0.74 (0.14) <sup>a</sup>	0.70 (0.20)
	Target	0.83 (0.10)	0.78 (0.09)	0.74 (0.18)	0.60 (0.18) <sup>a</sup>
<b>Response time</b>					
0-back	Non-Target	640.96 (117.98)	645.22 (58.44)	663.57 (119.89)	768.61 (206.29) <sup>b</sup>
	Target	682.02 (118.39)	699.71 (93.11)	713.53 (92.59)	787.60 (172.46) <sup>b</sup>
1-back	Non-Target	643.33 (122.62)	665.64 (62.34)	787.74 (169.42)	838.15 (197.73) <sup>b</sup>
	Target	759.40 (158.11)	817.06 (107.08)	941.52 (187.56)	988.89 (180.22) <sup>b</sup>

Data are presented as mean ± standard deviation (SD). aMCI: amnesic mild cognitive impairment; APOE: apolipoprotein E; HC: healthy controls.

<sup>a</sup>Post-hoc tests by Bonferroni's analysis further revealed the source of ANCOVA difference ( $P < 0.05$ , HC-APOE ε4– vs. aMCI-APOE ε4–).

<sup>b</sup>Post-hoc tests by Bonferroni's analysis further revealed the source of ANCOVA difference ( $P < 0.05$ , HC-APOE ε4+ vs. aMCI-APOE ε4+).

**Table 2** ERP data (P300 amplitude) for healthy controls and aMCI patients with different APOE ε4 status.

Task	site	HC		aMCI	
		APOE ε4 - (n = 25)	APOE ε4 + (n = 18)	APOE ε4 - (n = 27)	APOE ε4 + (n = 12)
0-back	CP1	3.69 (2.07)	3.23 (2.42)	3.16 (3.00)	2.44 (1.62)
	CPz	4.11 (1.63)	3.17 (0.68)	3.03 (1.82) <sup>f</sup>	2.45 (1.61)
	CP2	3.23 (1.69)	3.16 (0.87)	2.97 (1.64)	2.35 (1.66)
	P1	3.84 (2.37)	3.54 (1.01)	3.22 (1.80)	2.03 (1.78) <sup>b,d</sup>
	Pz	4.42 (2.25)	3.50 (0.91)	3.31 (1.77)	2.59 (2.56)
	P2	4.89 (2.02)	3.11 (1.00) <sup>a</sup>	3.04 (2.10) <sup>f</sup>	2.34 (1.96)
1-back	CP1	3.61 (2.14)	3.34 (0.65)	2.98 (3.38)	2.42 (1.59)
	CPz	4.63 (2.90)	3.21 (1.21) <sup>a</sup>	2.62 (1.80) <sup>f</sup>	2.53 (1.78)
	CP2	3.93 (1.92)	3.60 (1.12)	3.34 (2.07)	2.31 (1.56) <sup>b,d</sup>
	P1	4.49 (2.58)	3.24 (1.07)	3.00 (1.93) <sup>f</sup>	2.49 (2.10)
	Pz	5.11 (2.34)	3.43 (0.93)	3.23 (1.89)	2.54 (1.39)
	P2	4.52 (2.34)	3.71 (1.26)	3.53 (2.28) <sup>f</sup>	2.54 (1.74) <sup>f</sup>

Data are presented as mean ± standard deviation (SD); aMCI: amnesic mild cognitive impairment; APOE: apolipoprotein E; HC: healthy controls.

<sup>a</sup>Post-hoc tests by Bonferroni's analysis further revealed the source of ANCOVA difference ( $P < 0.05$ , HC-APOE ε4– vs. HC-APOE ε4+).

<sup>b</sup> Post-hoc tests by Bonferroni's analysis further revealed the source of ANCOVA difference ( $P < 0.05$ , aMCI-APOE ε4– vs. aMCI-APOE ε4+).

<sup>c</sup>Post-hoc tests by Bonferroni's analysis further revealed the source of ANCOVA difference ( $P < 0.05$ , aMCI-APOE ε4– vs. HC-APOE ε4–).

<sup>d</sup>Post-hoc tests by Bonferroni's analysis further revealed the source of ANCOVA difference ( $P < 0.05$ , aMCI-APOE ε4+ vs. HC-APOE ε4+).

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## EW0195

### Charles Bonnet Syndrome (CBS): Successful treatment of visual hallucinations due to vision loss with agomelatine in three cases

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**Background** CBS becomes more prevalent as the population ages. CBS is characterized by the triad of impairment of vision, complex visual hallucinations with insight, mentally normal people. Although visual hallucinations in the elderly are often associated with dementia with Lewy body, Alzheimer's disease and delirium, they are excluded from the diagnosis of typical CBS. Here, we describe three typical CBS patients whose visual hallucinations developed after bilateral severe visual impairment due to diabetic retinopathy. The effectiveness of agomelatine adds to evidence implicating serotonergic and melatonergic pathways in the pathogenesis of visual hallucinations.

**Case report** The average age of these three patients (2 males and 1 female) is 71. Except for the visual hallucinations, all patients showed no psychiatric symptoms or cognitive decline or neurological focal signs. They were frequently upset by the fact of hallucinating, fearing that they are losing their minds. They lived in fear of impending insanity, guilty feeling, unhappy mood, insomnia. The frequency of visual hallucinations stopped with agomelatine 25 mg/day for 3 weeks in these cases.

**Discussion** To our knowledge, this is the first report describing the effectiveness of agomelatine in treating typical CBS patients and indicates that agomelatine is a safer option for the treatment of CBS, especially in the elderly, diabetic population. Therapeutic options for CBS still remain poor and of uncertain benefit for the individual patient. CBS has a high prevalence rate (0.4%–30%) among the visually impaired. Clinicians must ask elderly people with visual impairment whether they have hallucinations. Firm reassurance that the syndrome is not related to mental illness is a major relief to an elderly person burdened already with failing vision.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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## EW0196

### White matter hyperintensities as a new predictor of driving cessation in the elderly

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**Background/aims** Motor, perceptual, and cognitive functions affect driving competence. White matter hyperintensities (WMH) changes on brain MRI are associated structural brain changes along with cognitive and motor performance. The aim of this study was to investigate the association between WMH and driving ability in the elderly.

**Methods** Participants ( $n = 540$ ) were drawn from a nationwide, multicenter, hospital-based, longitudinal cohort study. Each participant underwent clinical evaluations, neuropsychological tests, and interview for caregiver including driving capacity, which was categorized as 'now driving', and 'driving cessation (driving before, not now)'. A total 540 participants were divided into three groups (389 mild, 116 moderate, and 35 severe) depending on the degree of WMH. The same evaluations of them were followed after each year. The statistical analyses were performed using  $\chi^2$  test, an analysis of variance (ANOVA), structured equation model (SEM), and generalized estimating equation (GEE).

**Results** In a SEM, greater baseline degree of WMH was directly associated with driving cessation regardless of cognitive and motor dysfunction ( $\beta = -0.110$ ,  $P < 0.001$ ). In GEE models controlling for age, sex, education, cognitive, and motor dysfunction, the more severe changes of the degree of WMH was associated with the