

Letter to the editor – inconsistency on frequencies of the human apolipoprotein E Gene expression

Letter to the Editor

Ivan Sosa*¹, Ines Strenja-Linic²

1 Department of Forensic Medicine and Criminalistics; Rijeka University School of Medicine; Rijeka; Croatia

2 Neurosonology Laboratory; Rijeka University Hospital; Rijeka; Croatia

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Abstract: In this letter, we share our concerns regarding fluctuations of homozygosity for *ApoE/ε4* allele. It appeared that over time, expression of this gene raised the felled as reported by various researchers. Considering the diversity of phenotypic characteristics assigned to expression of *ApoE/ε4*, seems to be a potentially useful to direct practitioner on concrete figures related to the genetic propensity of many conditions, from circulatory to mental disorders.

Keywords: *Alzheimer's disease • ApoE/ε4 • Chronic traumatic encephalopathy*

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Sir,

We find the following issue so peculiar that we need to bring surface our cocerns. This matter rose out upon an attempt to pursue a comment on a review paper on allele frequencies of the apolipoprotein E (*ApoE*) $\epsilon 4$ predisposing Alzheimer's disease (AD) triggered by chronic traumatic encephalopathy (CTE).

This allele is associated with increased cholesterol levels and inheritance of the early-onset AD. We have been confused by some conclusions claimed in, otherwise gladly cited, review [3]. Besides of 48 CTE cases elaborated in the literature review, authors of that same article report three own cases. That sufficed to conclude that 15% of the general population is carriers of the *ApoE/ε4* allele [3]. Homozygosity in the general population was estimated to be 3%, contrasting 15% of the homozygosity among patients with AD.

Eight years before, Yoshida et al. evaluated findings upon autopsies [4] attaching frequency of this allele to nearly 30% of the general population, and 45% to 60% of patients with AD. Even Hollingworth et al. [2] put heterozygosity for this gene in the general population to be

this high. Consequently, it seems the general population closely resembles the population in paper subject. What, finally, supports [1] the number providently suggested years ago [3]. Masking such an important hypothesis in a review article written years before some other sources with similar epidemiology seems a bit doubtful.

When we authored this paper, it was not our intention to criticize, but to follow up in a constructive manner and produce a creative debate on the subject. Whereas McKee et al. [3] might not feel that the $\epsilon 4$ prevalence rate in AD is relevant to their findings on CTE; they astonish by referring to Eisenberg et al., though the late paper is newer than paper subject is [1].

Regardless of the sympathy and our preference towards review article of McKee et al, [3] epidemiological data used by Eisenberg et al. [1] should be set forth as a reference, even not verbatim.

Indicating a discrepancy in data about the prevalence of *ApoE/ε4*, rather than presenting any data, extenuates our contribution to the science, but not to the literature as relevance of our paper is to anyone trying to connect *ApoE/ε4*, CTE, and AD. Thus, this letter could be accepted as a "cry for accurate information" from a confused practitioner.

* E-mail: ivan.sosa@medri.uniri.ht

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