

Link Between Obsessive-Compulsive Disorder and ApoE Gene Polymorphisms[Letter]

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Dear editor

We have read the paper written by Ayse Dondu et al regarding Link Between Obsessive-Compulsive Disorder(OCD) and ApoE Gene Polymorphisms.¹ This study is the first to explore the relationship between ApoE gene polymorphisms and OCD subtypes, thus demonstrating a link between OCD and neurodegeneration/AD. This is a great contribution to the field of psychiatry. We commend the authors for their work and make a number of recommendations.

First, we would like to talk about the inclusion criteria. In this study, the distinction between early-onset OCD and late-onset OCD was made solely on the basis of a single variable, age of onset (split by whether or not the person had reached the age of 15), which is prone to selection bias. Steven Taylor conducted a systematic review and evaluation of OCD subtype programs, including meta-analysis and re-analysis of the original data, and found that age of onset was not a single-peak phenomenon, and that there were differences in variables such as gender and co-morbidities.² And, setting the age of onset threshold at 18 is more in line with the general rule. Thus, a combination of other confounding factors or the selection of appropriate age thresholds for age of onset groupings may provide a more comprehensive assessment.

Secondly, in previous research, the ApoE gene has been linked to autism spectrum disorders (ASDs), and there may be a complex genetic relationship between OCD and ASD.³ Many OCD symptoms are similar to the core features of autism spectrum disorders, and there are certain commonalities between the two, such as similar neurobiological mechanisms and the fact that they are often clinically coexisting or intersecting. The ApoE gene may be evidence of a correlation between the two at the genetic level, and some studies have confirmed that aberrant methylation of ApoE is associated with Alzheimer's disease (AD), which may have overlapping mechanisms with ASD.⁴ Consequently, should this study consider the complex genetic correlation between OCD and ASDs and the mechanism by which aberrant methylation of ApoE provides an overlap between AD and ASDs when considering the association of OCD with AD as well as neurodegenerative disorders based on ApoE gene polymorphisms.

Furthermore, we observe an interesting point. In recent study, mitochondrial autophagy has been implicated in neurodevelopmental as well as neurodegenerative changes, ApoE4 increases glycolytic activity but impairs mitochondrial respiration in astrocytes, and ApoE4-induced cholesterol accumulation impairs lysosome-dependent removal of damaged mitochondria.⁵ Therefore, in future studies, can we explore the more in-depth mechanism of diseases such as OCD and ApoE gene with respect to mitochondrial homeostasis. As a clinician, we expect more basic research to advance the identification and treatment of psychiatric disorders.

In conclusion, this study provides important information on the link between OCD and neurodegenerative disorders, but we recommend that Ayse Dondu and coworkers consider the above points to improve the accuracy and reliability of their findings. By integrating these methods and studies, they can assess the link between ApoE gene polymorphisms and obsessive-compulsive disorder (OCD) in greater depth and advance the cause of human health.

Disclosure

The authors report no conflicts of interest in this communication.

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