Open Access Rheumatology: Research and Reviews

Dovepress
open access to scientific and medical research

Open Access Full Text Article

LETTER

Advances in diagnostic and treatment options in patients with fibromyalgia syndrome

This article was published in the following Dove Press journal:
Open Access Rheumatology: Research and Reviews
15 June 2010
Number of times this article has been viewed

Jill M Recla[1,2]

[1]IGERT Program in Functional Genomics, Graduate School of Biomedical Sciences, The University of Maine, Orono, ME, USA; [2]The Jackson Laboratory, Bar Harbor, ME, USA

Dear Dr Liu,

I have read with great interest the article entitled "Advances in diagnostic and treatment options in patients with fibromyalgia syndrome" by Gur and Oktayoglu, which has been published online in *Open Access Rheumatology: Research and Reviews*.[1] The authors aimed to evaluate new diagnostic tools and new therapeutic treatment approaches for fibromyalgia (FM). I would like to comment on the article by referring to my recently published work in *Medical Hypotheses*,[2] which the authors reference in their section on the N-methyl D-aspartate receptor (NMDAR) antagonist memantine. My article describes the hypothesis that a combined therapeutic approach of the pharmaceuticals pregabalin and memantine may provide analgesic and neuroprotective benefits to patients with FM. The authors' statement, which directly references my work – "Memantine may also suppress neuronal excitability and confers neuroprotection in a manner similar to pregabalin" – was either misinterpreted or misquoted from my article, in which I make a similar statement (using the word "confer" rather than "confers"). Although the syntactical difference is slight, the ramifications are profound, and I thusly feel that this statement requires further clarification.

There are two primary points that need to be addressed. First is the assertion of neuroprotection. Memantine has been proven to confer neuroprotective benefits in human clinical trials and is currently approved by the United States Food and Drug Administration for the treatment of moderate to severe Alzheimer's disease. Conversely, while pregabalin has demonstrated neuroprotective capabilities in rats,[3] the question of its clinical utility as a neuroprotective agent has yet to be adequately addressed in humans. Given the assumption that both drugs confer neuroprotection, the second concern is the molecular mechanism of this protection implied by the authors' statement. Without the context of the biochemical effects of each drug that illustrate their ability to suppress neurodegenerative excitotoxicity, the reader may assume that both drugs confer their neuroprotective benefits via the same mechanism of action. This is technically inaccurate, as memantine is thought to target NMDARs, while pregabalin is thought to block the alpha-2-delta subunit of the voltage-gated calcium ion channel. Thus, while both memantine and pregabalin may confer neuroprotective benefits by suppressing neuronal excitability, I wish to clarify that 1) this capability has yet to be proven conclusively in humans for pregabalin, and 2) that the mechanisms by which these drugs suppress neuronal excitability differ at the molecular level.

Correspondence: Jill M Recla
600 Main Street, Bar Harbor, ME 04609, USA
Tel +1 207 282 8033
Email jill.recla@jax.org

Disclosure

This work was supported by the National Science Foundation, IGERT training grant # 0221625. The author declares no conflicts of interest.

References

1. Gur A, Oktayoglu P. Advances in diagnostic and treatment options in patients with fibromyalgia syndrome. *Open Access Rheumatology Research and Reviews*. 2009;1:193–209.
2. Recla JM, Sarantopoulos CD. Combined use of pregabalin and memantine in fibromyalgia syndrome treatment: a novel analgesic and neuroprotective strategy? *Med Hypotheses*. 2009;73(2):177–183.
3. Ha KY, Kim YH, Rhyu KW, Kwon SE. Pregabalin as a neuroprotector after spinal cord injury in rats. *Eur Spine J*. 2008;17(6):864–872.