

Antioxidant effect of buspirone in epilepsy – Is the case closed?

Efeito antioxidante de buspirona nas epilepsias. O caso está encerrado?

Comment about “Antioxidant effect of buspirone in epilepsy model induced by pilocarpine¹”

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Received: 11/3/2012 – Accepted: 11/3/2012

Scorza FA / Rev Psiq Clín. 2012;39(6):209

I read with interest a very comprehensive article titled “Antioxidant effect of buspirone in epilepsy model induced by pilocarpine” by Ferreira *et al.*¹ published in the December issue of *Revista de Psiquiatria Clínica*. In brief, the authors demonstrated that the pretreatment with buspirone has an anticonvulsant and antioxidant effect during the acute period of pilocarpine model of temporal lobe epilepsy. Given the dearth of published data about the relation of buspirone and epilepsy, we applaud Dr. Ferreira *et al.* for pursuing this topic, but I also find it necessary to address some purposes regarding their comments.

Initially developed for use in the treatment of generalised anxiety disorder, it appears that buspirone may be useful in various other psychiatric and neurological disorders, such as attenuating side effects of Parkinson's disease therapy, ataxia, depression, social phobia, and behaviour disturbances following brain injury, and those accompanying Alzheimer's disease, dementia and attention deficit disorder². Concerning epilepsy, few studies have accurately assessed the relationship between buspirone, seizures, and chronic epilepsy. Following a chronological sequence, Pranzatelli *et al.* showed in 1993 some inspiring proposals after evaluating the effects of buspirone administration in four individuals with progressive myoclonus epilepsy³. From experimental point of view, it was shown two years later that buspirone, using the low Mg²⁺ induced model epilepsy, was effective in reducing the frequency of occurrence of low magnesium induced field potentials in CA1 and CA3 areas of the hippocampus slice preparation (guinea pigs) in a dose dependent manner⁴. In 2004, Macêdo *et al.* demonstrated that small doses of buspirone protected against cocaine-induced seizures and/or mortality⁵. Recently, the research group led by De Freitas showed that buspirone exerted anticonvulsant effects associated with the inhibition of the development of oxidative stress caused by pilocarpine-induced seizures, suggesting a therapeutic use potential of buspirone in epilepsy treatment⁶.

In sum, the data presented by Ferreira *et al.*¹ open up new avenues of research in epilepsy field. Obviously, many question still

to be answered, such as: What are the effects of buspirone during epileptogenesis process in the pilocarpine model? Buspirone exerts antioxidant action in other brain structures, beyond the hippocampal formation? What is the effect of buspirone in animals with refractory epilepsy?

Finally, while these and other questions have not been answered, I express my congratulations to Ferreira *et al.* for the stimulating article and I am sure that the integration of the advancements in basic science with clinical trials allows us to create highly effective pharmacological strategies against uncontrolled epilepsy.

References

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