



SDI FINAL EVALUATION FORM 1.1

Vision requirePART 1:

Journal Name:	Journal of Advances in Medical and Pharmaceutical Sciences
Manuscript Number:	Ms_JAMPS_24441
Title of the Manuscript:	MOLECULAR INSIGHTS INTO THE ROLE OF INFLAMMATION AND OXIDATIVE STRESS IN EPILEPSY.
Type of Article	REVIEW ARTICLE

PART 2:

FINAL EVALUATOR'S comments on revised paper (if any)	Authors' response to final evaluator's comments
The authors improved the quality of the paper but there are still some mistakes in English and also in the use of abbreviations. The authors are also kindly encouraged to add a list of all the non common abbreviations used in the paper. For example: Pg 1 "Epilepsy represents a socioeconomic", Pg 2 "persist regional as well as cellular"; reactive oxygen species (ROS); please eliminate the text from "such as cytokines" to "severe inflammation" as it is a repetition; "Toll like receptors and cells"; "via many transcriptional factors such as nuclear factor κB"; "Blood brain barrier is made up of", Pg 3 "inflammatory response which leads to a decrease in seizure"; "of blood-brain barrier function (Rivest, 2010)."; "Disruption of the blood–brain barrier",..... Pg 4 "Toll-like receptors."; (Ye and Sontheimer, 1996); "TNF-α is another inflammatory factor implicated in epilepsy, in fact its expression",.... Pg 5 status epilepticus; "Oxidative stress is defined as an imbalance between generation and elimination of ROS"; "In OS there are alterations in ROS"; ... Pg 6"The increased activity of glutaminergic systems induces status epilepticus and there is a decrease in ATP levels due to change in redox potential, which leads to collapse in brain energy production and supply (Wasterlain et al., 1993). Seizures le ad to an increase in glutamate release and activation of NMDA receptor with a decrease in extracellular Ca²⁺ and an increase in cytosolic Ca²⁺ concentration (White et al, 2007). The effects mediated by Ca²⁺ during excessive glutamate receptor activation (excitotoxicity) lead to neuronal degeneration and overload of mitochondria with Ca²⁺, so free radicals are generated. This overload of Ca²⁺ leads to OS"; which generates superoxide (O₂⁻),.. Pg 7 "activation of N-methyl-D-aspartate receptor, influx of cytosolic"; "Increase in mitochondrial H₂O₂ production, lipid peroxidation"... Pg 8 "The literature clearly indicates that Inflammation and OS are mediators of acute"; "Disruption of the blood–brain barrier, the COX-2 signaling pathway"; "as well as Toll-like receptors are various"...	All the suggestions of distinguished reviewer have been incorporated in the final revised manuscript, and highlighted in red.