ANSWERING REVIEWERS

Long-term potentiation in autonomic ganglia: Potential role in cardiovascular disorders

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Answers to reviewers' comments:

Reviewer 1:
All corrections are done.

Reviewer 2:
About effect of serotonin in clinical settings. This is beyond the scope of this mini review as it is complicated and need a review all to it self.
The concentrations we used are comparable to doses used in clinical settings. This now added to text.

Are there any explanations why some PTSD patients may recover after a few years while others will not?

It is not clear why some PTSD may have lifetime symptoms whereas the majority recovers after a few months or years. This is, probably, related to a number of factors including types of traumatic event, previous traumatic exposure (e.g. being in two or more natural disasters at different times in their lives) and comorbidity (Kessler, R.C et al., (1995) Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 52:1048-1060).

Are there any ideas how to pharmacologically reverse already existing gLTP in a clinical setting (not preventing gLTP but reversing gLTP)?

Basically, termination of the condition that causes induction of gLTP can reverse it, e.g. preventing seizures with anti-epileptic drugs. In our experimental setting in rats with chronic stress, blood pressure returned to normal in 5-7 days after termination of stress.

The author should shortly comment on other hypothesis concerning causes for SUDEP.

The exact mechanism of SUDEP is not known, but a variety of factors, including cardio/autonomic, have been extensively reviewed in a chapter by Lathers et al., in "Sudden Death in Epilepsy-Forensic and Clinical Issues".

Are there any studies / data concerning the assumed increased risk of patients with epilepsy who smoke (page 11)?

The exact roles tobacco use plays in seizures or epilepsy have not been well studied. Seizure risks are higher in acute secondhand smokers, chronic active smokers, and babies whose mothers smoke (New reference added: Rong et al., 2014).

Are there any data concerning a caffeine-consumption-dependent-risk for cardiac events in smokers, i.e. does caffeine consumption increase the risk in smokers independent of other factors?

Smoking is a known risk factor for cardiovascular disease and has been implicated in sudden cardiac death. Heavy caffeine consumption is confounding factor in smokers.

All corrections and other suggestions are done.