Background:
- The purinergic P2X7 receptors, are ionotropic membrane channels, which can be activated by extracellular ATP.
- ATP is known to be involved cortical spreading depression (CSD), a substrate of migraine [1, 2].
- P2X7R activation promotes IL-1β[3] and TNF-α[4] release and CSD triggers these neuroinflammatory factors release[5].
- Inhibition of the P2X7-Panx1 complex suppresses spreading depolarization in rats and the induction of IL-1β post CSD [6].
- Whether the anti-P2X7R antibody could suppress CSD and the subsequent induction of neuroinflammatory factors is unknown.

Objectives:
- To examine the effects of anti-P2X7R antibody on cortex susceptibility to CSD.
- To address whether P2X7R contributes to the induction of IL-1β and TNF-α induced by CSD

Methods:
Electrophysiological technique was performed to detect the effect of anti-P2X7R antibody on CSD.
Real time PCR was then performed to analyze the gene level of IL-1β and TNF-α post CSD with or without antibody treatment.

Results:
1. Anti-P2X7R antibody suppressed CSD in rats

2. Anti-P2X7R antibody reduced the induction of mRNA of TNF-α, but not IL-1β by CSD

Conclusion and Further Work:
- Blockade of P2X7R by antibody reduced cortex susceptibility to CSD and rapidly reduced CSD induced gene expression of TNF-α in rats, indicating P2X7R as a potential target for migraine prophylaxis and treatment.
- The anti-P2X7R antibody did not reduce the induction of gene expression of IL-1β immediately after CSD. This may be due to: 1) the time points, as a significant down-regulation of IL-1β mRNA level was reported by inhibition of the P2X7/PANX1 pore complex 4 hours post CSD[6]. 2) suppression of spreading depolarization-induced IL-1β mRNA upregulation was due to inhibition of the P2X7/PANX1 pore complex, not the P2X7R cation channel [6].

References:
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