North Dakota INBRE
2010 Annual Meeting – Thursday, Oct. 28, 2010
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ABSTRACT SUBMISSION GUIDELINES
Deadline: Monday, Oct. 18, 2010

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GUIDELINES
1. Submission as a word document will allow us to make format changes as necessary. Abstracts will be compiled into a booklet.
2. Send as Microsoft Word document (.doc/.docx)
3. Left and right, top and bottom margins – 1 inch
4. Use single spacing; Font - Times New Roman, 10 point
5. The body of the abstract should include purpose, brief methods, summary of results and conclusion. List support on a separate line.
6. 250 word limit for body of abstract (not including title, authors, affiliations, support)
7. No tables or figures; see sample below.

SAMPLE ABSTRACT

RGS7 Protein Suppression of $G\alpha_o$ Protein-Mediated α_{2A}-Adrenergic Receptor Inhibition of Mouse Hippocampal CA3 Epileptiform Activity

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G-protein coupled α_2 adrenergic receptor (AR) activation by epinephrine (EPI) inhibits epileptiform activity in the mouse hippocampal CA3 region. The mechanism underlying this action is unclear. This study investigated which subtypes of α_2ARs, G-proteins ($G\alpha_o$ or $G\alpha_i$), and RGS proteins were involved in this response using recordings of hippocampal CA3 epileptiform bursts in mouse brain slices. First, we determined that this effect was mediated by the α_{2A}AR subtype as the inhibitory action of EPI on epileptiform burst frequency was abolished in slices from α_{2A}AR, but not α_{2C}AR, knockout mice. Next, using transgenic mice with the G184S Gnai2 allele (knock-ins) which interrupts G-protein α unit binding to regulators of G-protein signaling (RGS), we found that the α_{2A}AR antiepileptic effects of EPI were enhanced in hippocampal slices from mutant $G\alpha_o$ mice but not $G\alpha_{i2}$ mice. Finally, knockout mice for the RGS7 protein family were found to have increased α_{2A}AR-mediated hippocampal antiepileptic actions compared to their littermate controls. These results indicate that the EPI-mediated inhibition of mouse hippocampal CA3 epileptiform burst activity is through an α_{2A}AR/$G\alpha_o$-mediated pathway under strong inhibitory control by proteins of the RGS7 family. This suggests a possible role for selective α_{2A}AR agonists or RGS7 inhibitors as a novel antiepileptic drug therapy.

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