Jian Liu, Med Chem 2014, 4:12
http://dx.doi.org/10.4172/2161-0444.S1.011

OMICS Group Conferences
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3rd International Conference on
Medicinal Chemistry & Computer Aided Drug Designing
December 08-10, 2014 DoubleTree by Hilton Hotel San Francisco Airport, USA

Investigation of human CA1-induced toxicity in the *Drosophila* model of motor neuron degeneration

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Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, is one of the neurodegenerative diseases that affect motor neuron function leading to paralysis and fatal death. Mechanisms of ALS pathogenesis remain elusive. Carbonic anhydrase I (CA1) was recently discovered to be altered in ALS patients. Whether and how CA1 might be involved in ALS pathology is completely unknown. Our study demonstrated for the first time that CA1 was expressed in spinal cord motor neurons. We further established *Drosophila* model of CA1-induced motor neuron degeneration and investigated molecular mechanisms of pathology. Our results showed: 1) Expression of human CA1 in *Drosophila* motor neurons caused locomotion defect in both larval and adult flies without affecting lifespan; 2) Impairment of larval locomotion is correlated with reduced numbers of boutons and branches at NMJs, while preliminary data also indicated that the number of adult motor neurons was decreased by CA1 expression. 3) CA inhibitor acetazolamide ameliorated larval locomotion defect in *Drosophila* suggesting that carbonic anhydrase activity was required for the toxicity. Results from our studies provide potential new drug targets and design for therapeutic treatment of ALS.

Biography

Jian Liu has completed her PhD in Neuroscience from Washington University and postdoctoral studies from University of California, San Diego.

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