Eye-blink conditioning is a well-studied form of classical conditioning that relies on a discrete brainstem-cerebellar circuit. Eye-blink conditioning involved trials in which a tone conditioned stimulus (CS) precedes an eye stimulation unconditioned stimulus (US). The conditioned response (CR) is an eye-blink to the tone. The well times CRs observed in eye-blink conditioning are due to the precisely-timed regulation decrease in Purkinje cell inhibition of the deep cerebellar nuclei (DCN), the output nuclei of the cerebellum. The connection between basket cell axon terminal and Purkinje cell dendrites, may have an important role in this network. The axon terminals of the basket cells have the highest concentration in the brain of the potassium gated ion channel, Kv 1.2. Trafficking of Kv 1.2 out of the presynaptic membrane increases basket cell inhibition of Purkinje cells, which should in turn decrease inhibition of the DCN and produce eye-blink conditioning. Male Wistar rats were trained on a CS (tone) – US (eyelid stimulation) 750ms delay conditioning paradigm. Prior to the first five sessions of conditioning, rats were given intracranial infusions of either the Kv 1.2 blocker tityustoxin (TsTx; 1μg/μl) or vehicle. No infusions were made prior to sessions 6-9 of conditioning and a final infusion was made prior to session 10 of conditioning. Preliminary results show that rats infused with TsTx showed higher percentage of CRs in sessions 3-5 of conditioning than vehicle-treated rats. This facilitation persisted for several days after infusions and did not return after the infusion prior to session 10. These preliminary data show support for the role of Kv 1.2 in eye-blink conditioning.