Long and short term effects of glutamate on spider mechanosensory neurons during random stimulation

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Mechanosensory neurons innervating the VS-3 slit sensilla in the spider (*Cupiennius salei*) patella receive extensive efferent innervation. The efferent fibers contain GABA, glutamate, octopamine and acetylcholine, and the mechanosensory neurons have receptors to these transmitters. Activation of the structurally related ionotropic GABA and glutamate receptors opens Cl^- channels. However, while GABA depolarizes VS-3 neurons, glutamate does not have a similar effect.

When GABA was applied to VS-3 neurons while they were stimulated with pseudorandom noise signals, triphasic responses were observed, consisting of brief initial excitation, followed by short duration inhibition and ending in prolonged excitation. Here, we applied glutamate during similar random stimulation. We observed a reduction in firing rate that returned to control level within ~100 s. During this inhibitory period the neurons' sensitivity was reduced and their information capacity decreased. Contrary to the GABA effects, glutamate did not cause significant long term changes in neuronal excitability. These results suggest that in addition to Cl^- channels, glutamate may also activate K^+ channels and the resulting K^+ efflux may constrain depolarization and prevent excitation.