Increasing excitability of motor neurons at the *Drosophila* Neuromuscular Junction to examine functional plasticity

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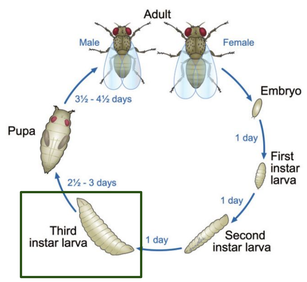
Abstract

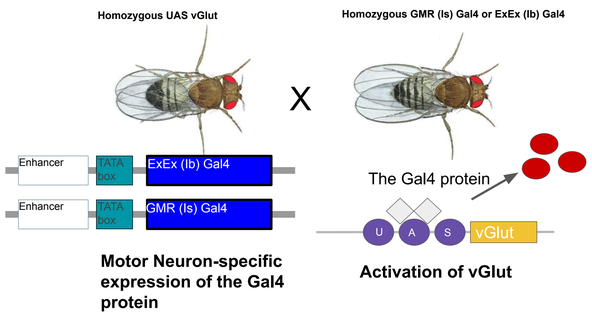
The Drosophila neuromuscular junction (NMJ) consists of two different motor neurons, which can compensate for stressors in the other motor neuron. This phenomenon is known as heterosynaptic plasticity. In this experiment, we overexpressed the glutamate transporter vGlut in individual motor neurons to examine functional plasticity. Overexpression of the gene vGlut increases the amount of glutamate in the synapse. We hypothesized that increasing excitability in one of the motor neurons will result in compensation of the unaffected motor neuron. Flies with vGlut overexpression in 1s neurons had lower amplitude than baseline transmission (wildtype) and 1b vGlut overexpression amplitude was slightly lower than baseline transmission. There is functional plasticity with 1b vGlut overexpression, but the amplitude of 1s > vGlut mimicked those of complete ablation.

Methods

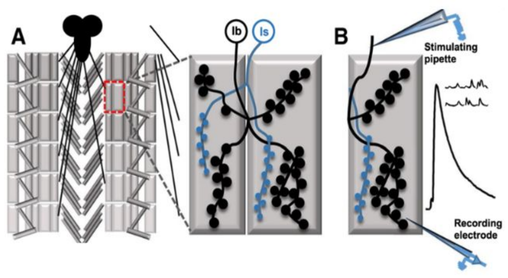
Drosophila is the premier genetic model organism



vGlut Overexpression using Gal4/UAS System



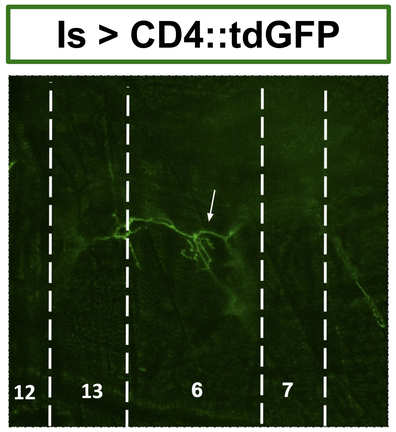
Electrophysiology at the Drosophila NMJ



Results

Using genetic tools to visualize motor neurons in *Drosophila*

Figure 1. ls motor neuron at the *Drosophila* neuromuscular junction (NMJ) expressing a transmembrane green fluorescent protein (GFP).



Comparing vGlut overexpression in ls and lb motor neurons to baseline transmission

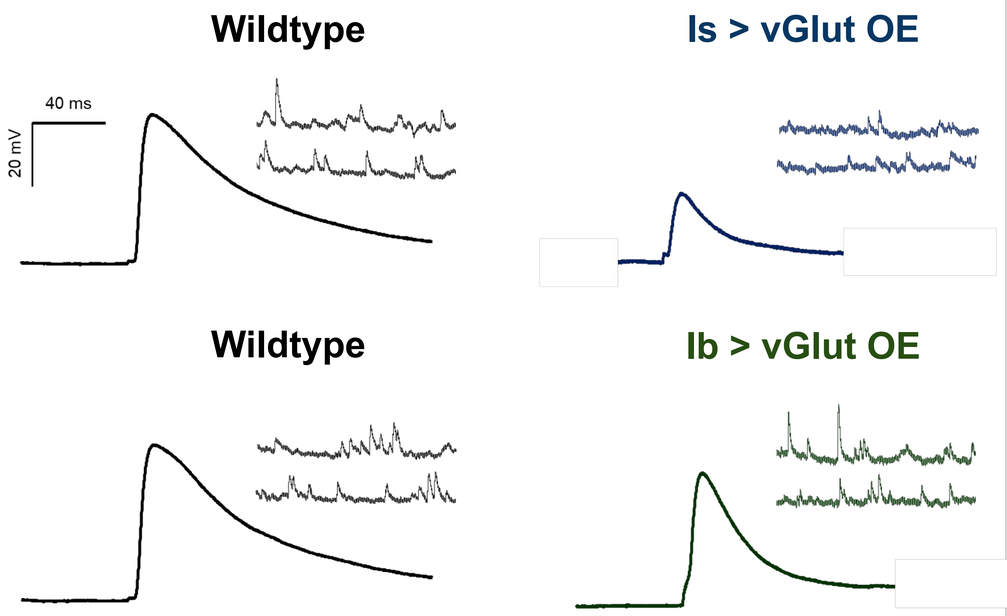


Figure 2. Representative traces of EPSPs and mEPSPs from 1s > vGlut and 1b > vGlut compared to baseline transmission.

Quantification

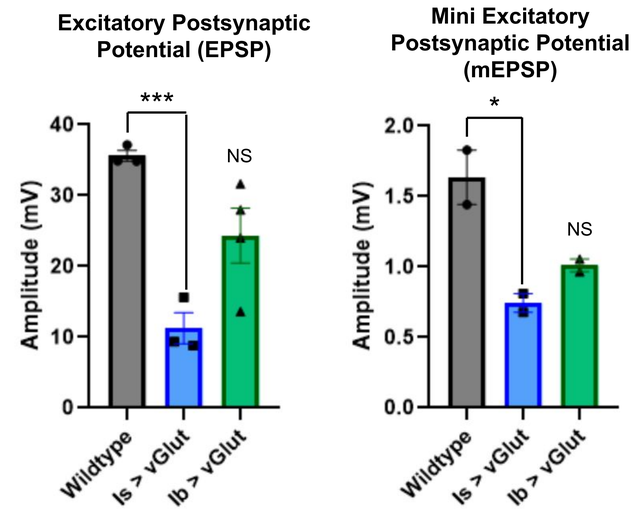


Figure 3. Overexpression of vGlut in ls motor neurons significantly decreased the evoked (p=0.00046, unpaired t-test) and mini amplitude (p = 0.049, unpaired t-test). There was no significance in evoked and mini amplitude in lb motor neurons compared to baseline transmission..

Conclusion

These results show that there is probably some natural plasticity when overexpressing vGlut in 1b motor neurons. However, the amplitude of 1s > vGlut flies was much lower than baseline transmission, mimicking the amplitude of ablation. These results were a blended phenotype, so in the future one would need to silence the other motor neuron.

References

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