Feeding behavior mutations of interest

- M4 (isthmus peristalsis), M3 and MC (pumping) are sufficient for nearly normal feeding behavior
- *eat-18* - necessary for EAT-2 function
- Pumping still occurs without EAT-2 and MC
- Δ*cca-1* - decreases potential of MC to trigger action potential –> reduced intake of food
- Δ*egl-19*
    1. myotonic group - muscle action potentials are prolonged and relaxation delayed
    2. flaccid group - slow muscle depolarization and feeble contraction
    3. lethal group - lacks all muscle contraction and dies as an embryo
- *eat-4* and *avr-15* are necessary for M3 –> muscle transmission
- Can uncouple TB and corpus
- Δ*ser-7* - MC not stimulated by serotonin
- Δ*gbp-2* - muscarin signalling pathway becomes hyperactive
- Δ*gar-3* - GAR-3 –> MPK-1 pathway is blocked - lower pumping during starvation
- Δ*eat-2/eat-5* - high probability of leaving DA837, but 200 fold preference for DA837 over much larger *B. megaterium*
- AIY interneuron - worms become strongly biased towards dwelling
- Δ*act-5* - absorption mutant
- Δ*egl-21* - don't produce most peptide signals - defective of satiety quiescence
- Δ*pkg-1* - loss of function shows no SQ - necessary for SQ along with DAF-11 - *tax-4* promoter rescues this SQ defect
- Δ*tax-2* - no quiescence
- ASI - potential lifespan extension via diet restriction
- Δ*daf-2/daf-7* - no SQ - higher than normal fat storage