What role does octopamine play in behavioral control in *Drosophila*?

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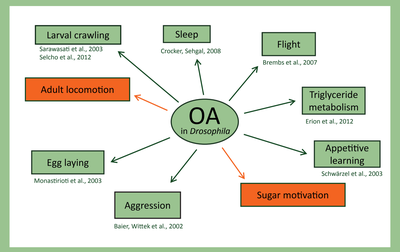
Biogenic amines in insects

coordination of physiological processes and behaviour

Introduction

Octopamine acts as a neurohormone, a neuromodulator and a neurotransmitter, contributing to the control of the animal physiology and behavior.



What cellular processes are at play in order to coordinate those different behaviors?

Flies mutant for the enzyme tyramine-beta-hydroxylase (*tßh*) have no octopamine (OA) and a 10-fold increased tyramine (TA) level.

Octopamine synthesis

Wild type:

TYR —TDC→ TA —TßH→ OA

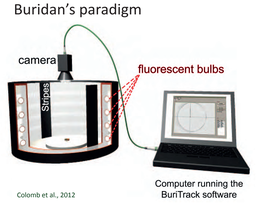
Mutant:

TYR —TDC→ TA↑ —TßH→ ~~OA~~

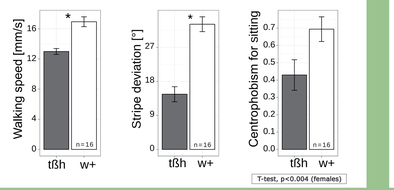
We investigate walking behavior in Buridan's paradigm and study the proboscis extension response (PER) as a locomotion-independent test for sucrose responsiveness after starvation.
We hypothesize that different subpopulations of octopaminergic neurons are involved in the two different behaviors.

Locomotion behavior is different in *tßh* mutants

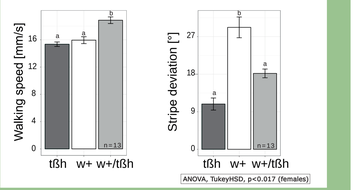
Buridan's paradigm



tßh mutants:
Decreased speed and increased fixation

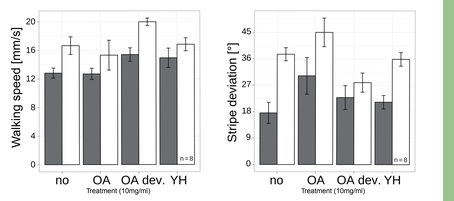


tßh mutation is semi-dominant



Pharmacological approach

preliminary



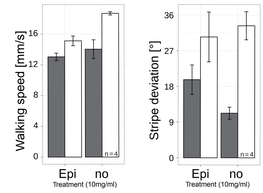
tßh
w+

OA - Octopamine, fed for 3h until test

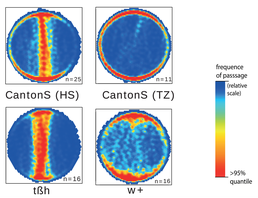
OA dev. - Octopamine, fed in food during development and for 3h until test

YH - Yohimbine (tyramine receptor blocker), fed for 3h until test

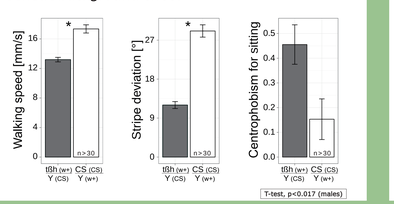
Epi - Epinastine (octopamine receptor blocker), fed for 3h until test



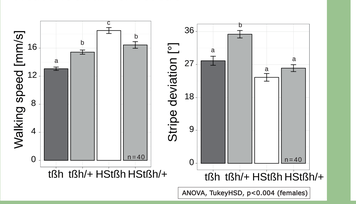
Transition plots



Genetic background effect

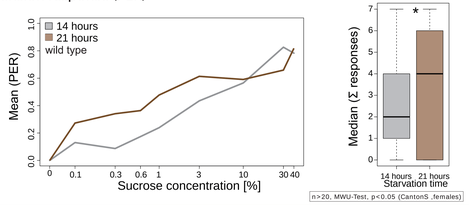


Heat shock driven rescue

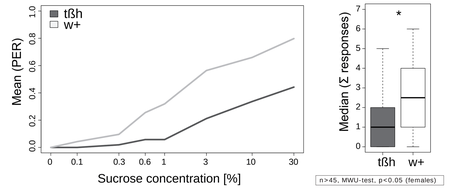


Sugar motivation is lower in *tßh* mutants

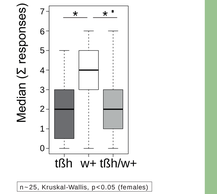
Paradigm to test proboscis extension response (PER)



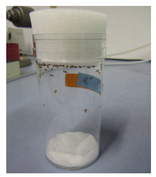
tßh mutants are less responsive to sugar after 20h starvation

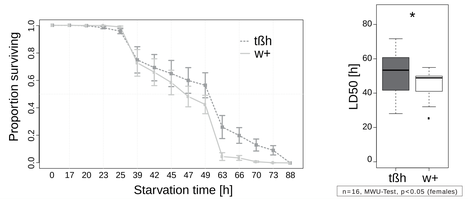


tßh mutation is dominant

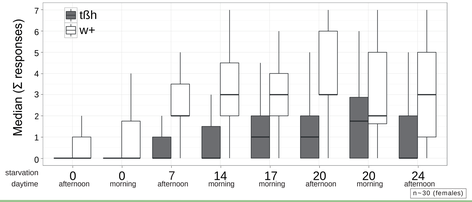


tßh mutants survive longer when starved to death



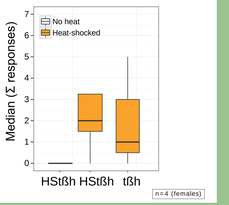


Starvation dependence of sugar response



Heat shock driven rescue

preliminary



Conclusions

tßh mutants have a deficit in locomotion and fixation behavior.
Walking speed may be controlled by a balance between both, tyramine and octopamine since the mutant phenotype can be rescued by giving a tyramine receptor blocker (yohimbine), and induced by an octopamine receptor blocker (epinastine).
Fixation behavior seems to be controlled dosage-dependently by octopamine.

tßh mutants show lower starvation-dependent sucrose responsiveness and prolonged survival while starved to death. Both phenotypes may be explained by an action of the amines on the metabolic rate.

Interestingly, the *tßh* loss of function mutation shows dominant effects for sugar motivation, while having semi-dominant effect on locomotion control.

Outlook

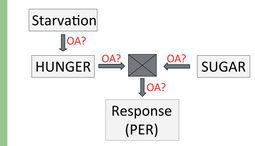
1. Trehalose measurement to calibrate starvation effect

2. Neuron-specific rescue of *tßh* mutation (cell cluster)

3. Activation and silencing of aminergic neurons

4. Test octopamine and tyramine receptor mutants

Possible roles of OA in the sugar response process



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