

# How to Run a Thesis Committee Meeting

Professionalization Seminar

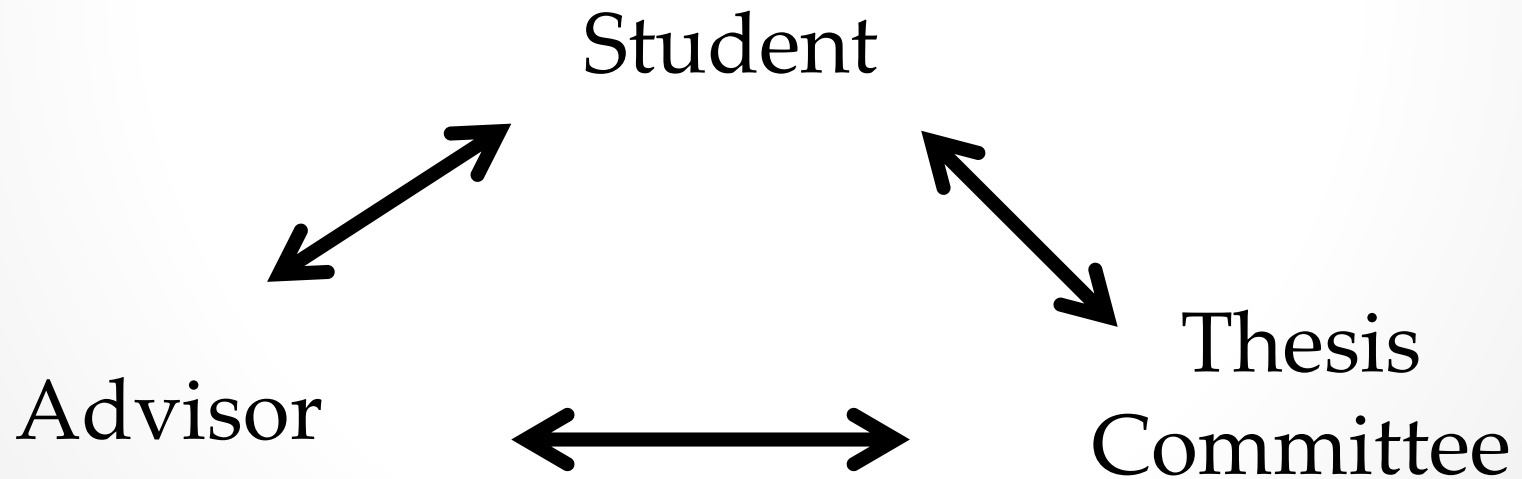
Oct 26, 2012

# BCDB's Definition of a Thesis Committee

## F. Student Advisory (Thesis) Committees

### *1. Function of the Committee*

The duties of the committee include assisting the student in creating and executing an original, publishable research project, assisting in the preparation of an acceptable dissertation, and administration of the final oral examination (*aka*: dissertation defense).



# Format of a Committee Meeting...

## **3. Format of Committee Meetings**

A student scheduled to meet with their committee should prepare a brief written summary of items to cover during the meeting and distribute it to their committee at least one week prior to the meeting. *Rather than re-stating the entire project, this document should focus mainly on the outcome of experimentation conducted since the previous committee meeting.* This will help the committee to determine the extent of interim progress made, allow time for feedback prior to the meeting and help focus the content of the meeting. In addition to the written update, students should also provide their committee members with an updated NIH style biosketch including any publications, abstracts, meeting presentations, and awards. Students experiencing significant difficulty in scheduling a committee meeting should contact the DGS for advice.

# What isn't a thesis committee meeting...

- An exam
- Look how hard I've worked...

# What is a thesis committee meeting?

- To the program: A way to monitor progress made by each student on their thesis
- To the student:
  - Scientific discussion among colleagues
    - Interpretation of data
    - Input on problems
  - Time to set realistic goals
  - Manage student/mentor relationships

# What is a thesis committee meeting supposed to be?

- To the program: A way to monitor progress made by each student on their thesis
- To the student:
  - Scientific discussion among colleagues
    - Interpretation of data
    - Input on problems
  - Time to set realistic goals
  - Manage student/mentor relationships

# What potentially makes up a Brief Summary?

- Personal Accomplishments
  - Publications/presentations, awards/honors received, grants written, conferences attended
- Background: necessary amount for member to understand why you did the experiment
- Scientific Progress- highlights of the accomplishments since your last meeting
- Questions and Discussion-focus of conversation
- Paper Figures or Figure descriptions
- Goals/Timelines

# What other things might you send to your committee?

- NIH Biosketch –mandatory
- Agenda
- **Questions that you want input on**
- Papers written or presentations given

# How do I run the meeting?

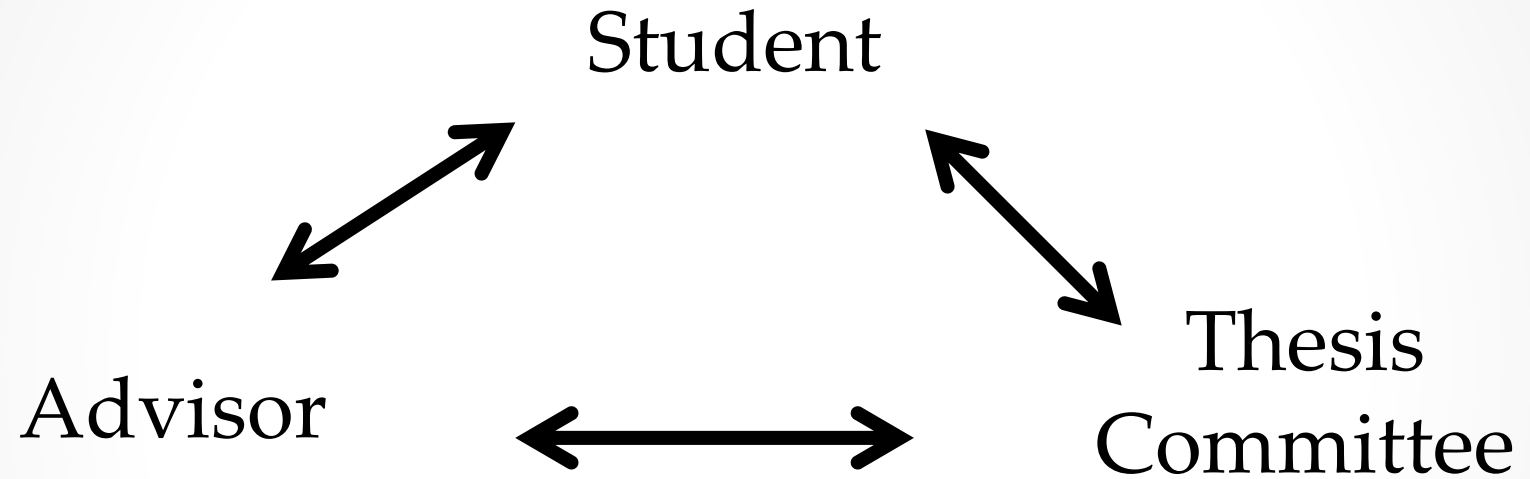
## Key: You're in charge

- Summarize key personal accomplishments
- Powerpoint of data or figures
- Discussion



# How does the meeting change as I progress?

1. Plan and Approach- some data
2. Data Interpretation- how does this make a publishable story
3. Wrapping up- publishing papers, final pieces of data, writing thesis



# Documentation of the meeting

## BCDB THESIS COMMITTEE MEETING AND PROGRESS REPORT FORM

The purpose of this form is to provide documentation and a summary of the outcome of the student's thesis committee meeting. It should be completed at the end of each thesis committee meeting. To receive credit for your committee meeting, please return completed forms promptly to the BCDB Program Office, Suite 300A Dental Building.

_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

**Approximate date of next meeting** \_\_\_\_\_

Note: maximum interval between committee meetings is six months for students up to and including year 5, then four months in year 6 and beyond. The committee may require a student to hold a meeting earlier than these guidelines.

**Approval to prepare written dissertation without holding a further committee meeting** ☐

Note: no more than six/four months (if the student is year five or earlier/if the student is in year six or above, respectively) permitted between receiving approval to prepare a thesis and the defense date.

**Anticipated written dissertation approval and defense date:** \_\_\_\_\_

**Specific Comments; indicate if back or additional page used** ☐

(e.g., progress in preparing results for publication, experiments required to fulfill degree requirements, expected timetable for meeting these goals, etc.)

# Brief Summaries

## RNA Polymerase II elongation regulation and in the *C. elegans* germline

### Background:

The *C. elegans* first germline cells, called primordial germline cells, or PGCs, are descended from cells called the P lineage in the embryo. In the P lineage, RNA Polymerase II (Pol II) transcriptional elongation is globally inhibited by the PIE-1 protein. PIE-1 was thought to inhibit elongation by sequestering cyclin T of the Pol II elongation kinase complex P-TEFb, preventing it from targeting Pol II. When the *C. elegans* PGCs are born from the P lineage, PIE-1 is degraded and the P-TEFb target, serine 2 of the RNA Polymerase II (Pol II) C-terminal domain, becomes phosphorylated (Ser2-P). Importantly, Ser2-P in the PGCs is uniquely independent of the P-TEFb complex.

### Results:

Ser2-P in the *C. elegans* germline is independent of P-TEFb: I have extended previous findings to show that Ser2-P is independent of P-TEFb in the germline throughout development. Instead, all Ser2-P requires the newly discovered Ser2 kinase complex, CDK12/Cyclin K (CDK-12 is CDTL-7 in *C. elegans*).

Relationship of CDK-12/Cyclin K to the germline transcriptional repressor, PIE-1: I previously showed that PIE-1 likely inhibits transcription in the P lineage by inhibiting the CDK-12/Cyclin K complex. I am currently making progress in showing a physical interaction between PIE-1 and Cyclin K in the P lineage either by Co-IP or by tethering studies. Specifically, I am constructing a transgenic line that expresses PIE-1 tagged by an inner nuclear membrane (INM)-associated protein, BAF-1. I have preliminary data to show that a transgenic line expressing this construct does mis-localize PIE-1 to the INM in Z2/Z3. I plan to test if Cyclin K and or Cyclin T co-localized with this mis-targeted PIE-1.



# How Summaries Change...Nov2009

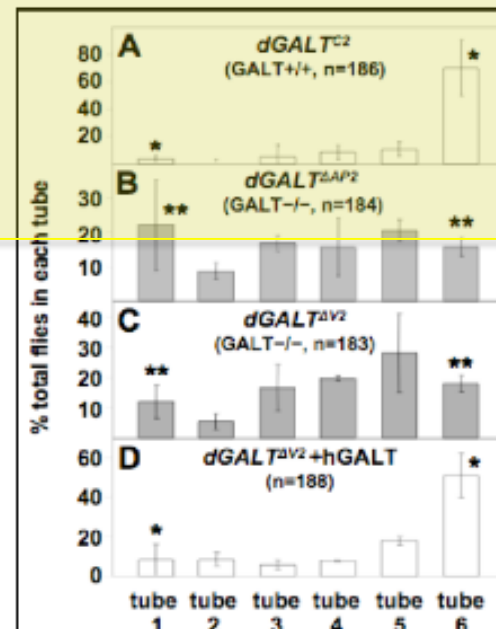
## My main fly project:

**Goal:** To define and characterize possible long-term (sub-lethal) complications in *GALT*-deficient *D. melanogaster*. Specifically, I am interested in exploring possible fly correlates of the long-term complications that are commonly seen in patients with classic galactosemia.

Thus far, I have shown that *GALT*-deficient flies have a geotaxic defect (see figure below). Cohorts of 60 male flies less than 4 days old are run through a tube maze and the flies that get to the last (6<sup>th</sup>) tube are much faster at responding to stimuli and faster more coordinated climbers than those that remain in the first tube. In short, cohorts of adult flies homozygous for either *dGALT* <sup>$\Delta$ 1AP2</sup> or *dGALT* <sup>$\Delta$ 1V2</sup>, reared on molasses food, are much slower than cohorts of the precise excision control flies, *dGALT*<sup>C2</sup>. Transgenic rescue with *hGALT* restores the wild-type distribution. I have also shown that *dGALT* <sup>$\Delta$ 1AP2</sup> and *dGALT* <sup>$\Delta$ 1V2</sup> homozygous females have a reproductive deficiency when compared to controls, although this result is still preliminary.

For both the neuromuscular and reproductive outcomes I plan to characterize the phenotype as follows:

- What are the *GALT* specific abnormalities in these flies and do they mimic what has been reported for patients?
  - Define the of level galactose metabolites in these animals and are the more affected flies different?
  - Determine the affect of galactose exposure
  - How much *GALT* enzyme activity is needed for "normal" outcome?
- What are the anatomical, cellular or biochemical reasons for these specific phenotypes?
  - Are there any gross structural deficiencies in these flies in regards to these phenotypes?
  - What are the glycosylation deficiencies seen in larvae? (data is being done by the CCC at UGA) And could they contribute to these phenotypes
  - Are there alleles of other genes that cause similar phenotypes and do they genetically interact with any



# How Summaries Change...June 2011

1. GALT-deficient flies demonstrate a complex movement phenotype despite dietary restriction of galactose.

- a. I have tested the GALT-deficient allele in flies from both W1118 and OregonR backgrounds
- b. I have tested the GALT-deficient allele over a deficiency
- c. I have tested the GALT-deficient allele over wild-type to confirm that the movement phenotype is a recessive trait
  - i. In the process of doing this work I determined that GALT activity is important very early in development -- prior to zygotic gene expression
- d. I have tested transgenic hGALT rescue via the Actin5C-GAL4 driver and I have tested the GAL4 driver alone in an hGALT-null background.

Data, data, data

2. Maternal loading from a GALT+ mother impacts severity of the long-term outcome in her offspring
  - a. Flies that are heterozygous for GALT activity (GALT normal females crossed to GALT deficient males) that come from wild-type mothers and mutant fathers do not have a defect in the countercurrent
  - b. Flies that are heterozygous for GALT activity (GALT deficient females crossed to GALT normal males) that come from mutant mothers and wild-type fathers have a slight defect in the countercurrent.
  - c. I have some preliminary evidence that flies that are themselves GALT deficient but came from mothers who were heterozygous for the mutant allele may not be as severely affected as GALT deficient flies that came from GALT deficient mothers. I should have these data when we meet

have since gone back to my counter current apparatus and tested the controls you all mentioned in this behavior assay. Below you will find my updated outline and my goal is to complete these experiments in the next 3 weeks and have a paper ready for submission in early August. I will be in Bar Harbor Maine for the Jackson Labs Short Course on Medical and Experimental Mammalian Genetics from July 17 to July 29.

# How Summaries Change

March 22, 2012

Oct 24, 2012

## Goal 1: Determine the basal physiology of pharyngeal satellite cells.

*A. Do pharyngeal muscle fibers contain more satellite cells than limb muscle fibers?*

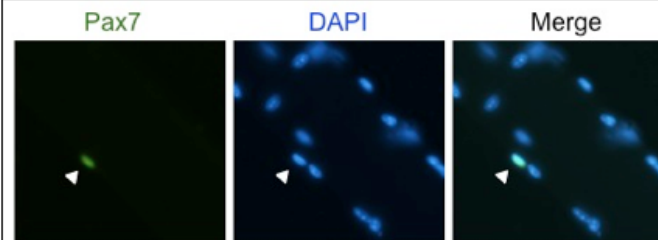
### 1. Isolating single myofibers from pharyngeal muscles and staining for Pax7-expressing satellite cells:

**Purpose:** To directly quantify and compare the number of satellite cells per single myofibers isolated from the pharynx or the limb muscle, extensor digitorum longus (EDL) using an endogenous transcription factor, Pax7.

**Approach:** Single myofibers were isolated from EDL muscles and then stained with various Pax7 antibodies and immunostaining reagents to determine optimal immunostaining conditions.

**Results:** After several experiments and reagent optimizations, Pax7 immunostaining of satellite cells was obtained on isolated EDL myofibers (Fig. 2).

**Conclusions and Plan:** We finally have a Pax7 immunostaining protocol working for single isolated myofibers. Additionally, we have recently worked out the conditions necessary for isolating single pharyngeal myofibers. Therefore, pharyngeal and EDL fiber preps will be collected and immunostained for Pax7 from 2-3 month old mice to quantify and determine satellite cell number per length of fiber. We hypothesize that the pharyngeal myofibers will have an increased number of satellite cells compared to the limb as suggested by our previous X-gal stainings of muscle sections from Myf5LacZ mice.



**Figure 2. Pax7 immunostaining of satellite cells on isolated EDL myofibers.** Single myofibers were isolated from 10-week-old C57/BL6 mice, formalin fixed, and stained for the presence of the myogenic transcription factor Pax7 (green). Arrowhead indicates a Pax7<sup>+</sup> satellite cell. DNA was labeled using DAPI (blue). EDL=extensor digitorum longus.

## Paper #1: Pharyngeal Satellite Cells: A Novel Stem Cell with Enhanced Myogenic Properties

Figure 1: Pharyngeal muscles of mice.

- A. Schematic of tissue dissection/region analyzed in current studies.
- B. Histology demonstrating and identifying each region of the pharynx.

Figure 2: Pharyngeal muscles possess a larger number of satellite cells than limb

- A. Demonstration and quantification of increased satellite cell numbers being present in pharyngeal versus limb muscle using a LacZ transgenic mouse which expresses LacZ under the promoter of the myogenic transcription factor, Myf5.
  - a. Done.
- B. Independent genetic mouse model used to assess satellite cell numbers in limb and various regions of pharyngeal muscle. Muscle sections from tdTomato/Pax7CreER mice were used to identify satellite cells which co-stained with laminin.
  - a. Two tdTomato/Pax7Cre mice, induced with either vehicle or tamoxifen 2 months prior, were euthanized and muscles collected for sectioning.
    - i. Result/Assessment: Most fibers in pharynx and limb muscle had some tdTomato fluorescence in both vehicle and TM induced animals, suggesting moderate expression of Pax7CreER protein at some point during development resulting in recombination events. However, bright red cell-shaped puncta are visualized along the periphery of myofibers in all tissues examined. Both laminin and DAPI staining co-localized with the tdTomato fluorescence, confirming these cells as satellite cells.
  - ii. Plan: Perform laminin staining on pharyngeal sections of induced tdTomato/Pax7Cre mice and compare SC numbers to previous data from the Myf5/nLacZ mice.