

Supplemental Material

CBE—Life Sciences Education

Ortega et al.

Classroom Observation Protocol for Undergraduate STEM – COPUS

Smith MK, Jones FHM, Gilbert SL, and Wieman CE. 2013. The Classroom Observation Protocol for Undergraduate STEM (COPUS): a New Instrument to Characterize University STEM Classroom Practices. CBE-Life Sciences Education

Date and time of Observation: _____

1) Background Information

- a) **Observer Name:** _____
- b) **Class No./name/section:** _____
- c) **Observer's location in the class:** _____

2) Classroom and background

- a) Room location and layout (e.g., type of student seating, instructor on podium, etc.).
- b) Note if there is anything unusual about this particular class/lecture (e.g., quiz day, first day of semester, etc) (try to avoid observing classes that are particularly anomalous)
- c) **(Optional, if known)** What goes on out of class? Homework? Pre-readings? Labs? Projects? Other?
Explain briefly.
- d) **(Optional, if know)** How varied are classes for this course? Circle one each, to show balance of **Active Students / Instructor Delivery** ...
 - i) for the **Whole Course**, balance approximates: 0%/100% 20/80 40/60 50/50 60/40 80/20 100%/0%
 - ii) in **Today's Class Only**, balance approximates: 0%/100% 20/80 40/60 50/50 60/40 80/20 100%/0%

3) Narrative Description of Class (also known as field notes) (optional)

Information could include ...

- The structure of the lesson (e.g., how the instructor sequenced material, the narrative arc of the class)
- The range and nature of activities that occurred.
- Dialog/behaviors that illustrate codes you gave, especially for teaching techniques and student engagement.
- Instructor's actions that appear to have affected students' engagement.
- Evidence of variability among students (e.g., if small groups, to what extent did groups behave and engage similarly?)

Observation codes

1. Students are Doing

- L** Listening to instructor/taking notes, etc.
- Ind** Individual thinking/problem solving. Only mark when an instructor explicitly asks students to think about a clicker question or another question/problem on their own.
- CG** Discuss clicker question in groups of 2 or more students
- WG** Working in groups on worksheet activity
- OG** Other assigned group activity, such as responding to instructor question
- AnQ** Student answering a question posed by the instructor with rest of class listening
- SQ** Student asks question
- WC** Engaged in whole class discussion by offering explanations, opinion, judgment, etc. to whole class, often facilitated by instructor
- Prd** Making a prediction about the outcome of demo or experiment
- SP** Presentation by student(s)
- TQ** Test or quiz
- W** Waiting (instructor late, working on fixing AV problems, instructor otherwise occupied, etc.)
- O** Other – explain in comments

2. Instructor is Doing

- Lec** Lecturing (presenting content, deriving mathematical results, presenting a problem solution, etc.)
- RtW** Real-time writing on board, doc. projector, etc. (often checked off along with Lec)
- FUp** Follow-up/feedback on clicker question or activity to entire class
- PQ** Posing non-clicker question to students (non-rhetorical)
- CQ** Asking a clicker question (mark the entire time the instructor is using a clicker question, not just when first asked)
- AnQ** Listening to and answering student questions with entire class listening
- MG** Moving through class guiding ongoing student work during active learning task
- 1o1** One-on-one extended discussion with one or a few individuals, not paying attention to the rest of the class (can be along with MG or AnQ)
- D/V** Showing or conducting a demo, experiment, simulation, video, or animation
- Adm** Administration (assign homework, return tests, etc.)
- W** Waiting when there is an opportunity for an instructor to be interacting with or observing/listening to student or group activities and the instructor is not doing so
- O** Other – explain in comments

3. Student Engagement (optional)

- L** Small fraction (10-20%) obviously engaged.
- M** Substantial fractions both clearly engaged and clearly not engaged.
- H** Large fraction of students (80+%) clearly engaged in class activity or listening to instructor.

Student engagement alternatives:

- (1) Just mark when engagement is obviously high or obviously low.*
- (2) Count "N" students near you (~10) and assess how many appear engaged at every 2 minute interval. Enter value for all engaged instead of L/M/H. NOTE what your value of N was.*

Suggestions regarding codes and comments:

- Clarify code choices with comments.
- Consider indicating your confidence regarding coding, especially when you aren't sure about choice of codes.

HOW TO USE OBSERVATION MATRIX: Put a check under all codes that happen anytime in each 2 minute time period (check multiple codes where appropriate). If no codes fit, choose "O" (other) and explain in comments. Put in comments when you feel something extra should be noted or explained.

Date: _____ Class: _____ Instructor: _____ No. students _____ Arranged how? _____

1. **L**-Listening; **Ind**-Individual thinking; **CG**-Clicker Q discussion; **WG**-Worksheet group work; **OG**-Other group work; **AnQ**-Answer Q; **SQ**-Student Q; **WC**-Whole class discuss; **Prd**-Predicting; **SP**-Student present; **TQ**-Test/quiz; **W**-Waiting; **O**-Other

2. **Lec**-Lecturing; **RtW**-Writing; **FUp**-Follow-up; **PQ**-Pose Q; **CQ**-Clicker Q; **AnQ**-Answer Q; **MG**-Moving/Guiding; **1o1**-One-on-one; **D/V**-Demo+; **Adm**-Admin; **W**-Waiting; **O**-Other
 For each 2 minute interval, check columns to show what's happening in each category (or draw vertical line to indicate continuation of activity). OK to check multiple columns.

COPUS																												
min	1. Students doing														2. instructor doing										3. Engagement			Comments: EG: explain difficult coding choices, flag key points for feedback for the instructor, identify good analogies, etc.
	L	Ind	CG	WG	OG	AnQ	SQ	WC	Prd	SP	T/Q	W	O	Lec	RtW	Fup	PQ	CQ	AnQ	MG	1o1	D/V	Adm	W	O	L	M	
0 - 2																												
2																												
4																												
6																												
8 - 10																												
	L	Ind	CG	WG	OG	AnQ	SQ	WC	Prd	SP	T/Q	W	O	Lec	RtW	Fup	PQ	CQ	AnQ	MG	1o1	D/V	Adm	W	O	L	M	H
10 - 12																												
12																												
14																												
16																												
18 - 20																												
	L	Ind	CG	WG	OG	AnQ	SQ	WC	Prd	SP	T/Q	W	O	Lec	RtW	Fup	PQ	CQ	AnQ	MG	1o1	D/V	Adm	W	O	L	M	H
20 - 22																												
22																												
24																												
26																												
28 - 30																												

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For each 2 minute interval, check columns to show what's happening in each category (or draw vertical line to indicate continuation of activity). OK to check multiple columns.

page 2

min	1. Students doing													2. instructor doing											3. Engagement			Comments: EG: explain difficult coding choices, flag key points for feedback for the instructor, identify good analogies, etc.								
	L	Ind	CG	WG	OG	AnQ	SQ	WC	Prd	SP	T/Q	W	O	Lec	RtW	Fup	PQ	CQ	AnQ	MG	1o1	D/V	Adm	W	O	L	M		H							
30 - 32																																				
32																																				
34																																				
36																																				
38 - 40																																				
	L	Ind	CG	WG	OG	AnQ	SQ	WC	Prd	SP	T/Q	W	O	Lec	RtW	Fup	PQ	CQ	AnQ	MG	1o1	D/V	Adm	W	O	L	M	H								
40 - 42																																				
42																																				
44																																				
46																																				
48 - 50																																				

Further comments:

If you would like to have a protocol sheet that extends beyond 50 minutes, please check the following website: www.cwsei.ubc.ca/resources/COPUS.htm or contact the corresponding author (michelle.k.smith@maine.edu) for a modifiable spreadsheet.

COPUS Training Guide

1. 10 mins. Introductions and brief rationale for exercise and overall goals.
2. 15 mins. Hand out paper copies of protocol and code explanations. Allow participants to read them over. Project the code explanations. Discuss the codes as a group and answer any questions.
3. 5–10 mins. Show two minutes of a video that is straightforward to code (mostly lecture, administrative announcements). Observers individually mark their paper copy of the protocol. Stop after two minutes and have a group discussion about the codes they selected. Which codes chosen for students? For instructor? How many for each?
4. 8 mins. Now group the observers in pairs and have the two observers sit near each other. Play a video for ~8 minutes and have observers record what is going on in 2-minute segments on the paper copy of the protocol. In order to keep all observers in sync, use either a shared two-minute sand timer or a stopwatch counting up (this feature is often found on cell phones).
5. 10 mins. Have the observer pairs first compare notes with each other for the 8 minute segment and then have a discussion with the larger group. For the group discussion, observers take turns volunteering what they coded for the students and the instructors every two minutes for the 8-minute clip. Discuss any codes that were unclear. For example, observers often want to clarify when to mark the student code “OG Other group activity” and how that differs from having students discuss a clicker question or work on a worksheet. It is also recommended to discuss the instructor code “FUp Follow up” and the importance of marking “PQ Posing non-clicker question to students” if the instructor follows up by posing questions to students. Observers may also talk about the relationship between some student and instructor codes. For example, if observers mark “CG students discussing a clicker question,” they will also likely mark the instructor code “CQ Asking a clicker question.”
6. 15 mins. Have observer pairs code two minutes of a video segment that shows students and instructors showing multiple behaviors such as asking and answering questions, small group activities, and/or discussing clicker questions. After two-minutes have the pairs compare codes and discuss the results with the larger group. Then have observers code the next 6 minutes (8 minutes total of this segment of the class). Again have pairs compare answers and discuss the answers as a whole group volunteering what they coded for the students and the instructors every two minutes for the 8-minute clip.
7. 10 mins. Organize pairs and select classes to observe. Plan a way to collect data from observers (collect paper copies, fill in the information on an on line form). If possible, meet with observers after they have collected data to share aggregate results and talk through any codes that were causing difficulties.

8. If you have two observers in a classroom and would like to calculate inter-rater reliability (IRR), for all 25 codes add up all the total number of times: 1) both observers put a check in the same box, 2) neither observer put a check in the same box, 3) observer 1 put a check in a box when observer 2 did not, and 4) observer 2 put a check in a box when observer 1 did not. With this information, you can use a statistical package such as SPSS (IBM Inc.) to calculate the Kappa values. #

Notice for potential participants in Synthesis Map research project
Cynthia Brame, Principal Investigator
Ryan Ortega, co-investigator

Dear [],

I am writing to request permission to use materials that you generated in BSCI 245: Biology of Cancer during Spring 2013 to investigate the usefulness of synthesis maps as a pedagogical tool. The purpose of the study is to evaluate the effectiveness of the synthesis maps as a tool to help you generate a holistic view of carcinogenesis.

If you give permission, I and my co-investigator Ryan Ortega will examine your synthesis map for organizational clarity and for the utility of the spatial features of Prezi for this assignment. We will also compare the quality of your synthesis map to your performance on the exams and the final paper. If you agree to participate, Ryan will be able to associate your name with your synthesis map because this information is embedded in the link you provided to your map, but he will have no access to your grades.

We will report any conclusions that we draw about synthesis maps in aggregated form, such that your work is anonymous. Your synthesis map will not be shared without your specific written permission. We believe that there is very minimal or no potential risk for you, the student. Allowing us to use your work in this study will help us to develop this novel assessment method that may then be utilized by other teachers desiring to assess higher level synthetic learning in their students.

Please follow the link to the single-question survey to indicate whether you **do or do not grant permission** for your materials to be used in this study.

If you have any questions or concerns about the study, you may contact me at Cynthia.brame@Vanderbilt.edu. You may also contact the Vanderbilt Institutional Review Board. For additional information about giving consent or your rights as a participant in this study, contact the Vanderbilt Institutional Review Board Office at [\(615\) 322-2918](tel:6153222918) or toll free at [\(866\) 224-8273](tel:8662248273).

Thank you!

Sincerely,

Cynthia J. Brame

Notice for potential participants in Synthesis Map research project—sharing example
Cynthia Brame, Principal Investigator
Ryan Ortega, co-investigator

Dear [],

I am writing to request permission to use materials that you generated in BSCI 245: Biology of Cancer during Spring 2013 to investigate the usefulness of synthesis maps as a pedagogical tool. This is a specific request to **share your synthesis map as an example of the genre** and is in addition to a general request to be able to study your synthesis map as part of the larger research project.

I appreciate your previous permission to share your synthesis map with attribution, and ask you to give that more formally by signing this form and returning it to me by email, campus mail, or traditional mail to

PMB 183
230 Appleton Place
Nashville, TN 37203-5721

If you have any questions or concerns about the study, you may contact me at Cynthia.brame@Vanderbilt.edu. You may also contact the Vanderbilt Institutional Review Board. For additional information about giving consent or your rights as a participant in this study, contact the Vanderbilt Institutional Review Board Office at [\(615\) 322-2918](tel:6153222918) or toll free at [\(866\) 224-8273](tel:8662248273).

Thank you!

Sincerely,

Cynthia J. Brame

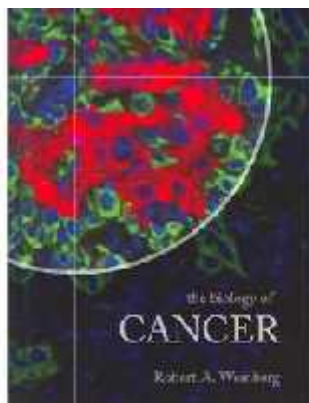
BSCI 245: Biology of Cancer
Spring 2013
MWF 1:10-2:00
SC 2212

Dr. Cynthia J. Brame

Office: Center for Teaching, room 307
1114 19th Avenue South, 3rd floor (ROTC building)
Phone: 615-322-7290 (office); 615-943-3208 (cell; use judiciously)
Email: Cynthia.brame@vanderbilt.edu
Office hours: M 2-3 (in MRBIII coffee shop); R 2-4 (in CB office); other times by appointment

Texts: *The Biology of Cancer*
Robert Weinberg
Garland Science 2007

Various research articles
(listed below and provided on OAK)



Course description and objectives:

Cancer is a complex disease—more accurately, a set of complex diseases with some common underlying causes. Experiments in the last thirty-five years have led to a wealth of information (>1.5 million research articles!) about the causes of cancer and the genes and proteins involved in its development. This deluge of data can be daunting, particularly for students beginning their study of the field. This class is designed to provide an introduction to the underlying principles of cancer development that are emerging from the vast and growing collection of facts about this disease.

We are going to examine these principles through the lens of two overarching questions:

Are cancers newly evolved species?
How does the evolutionary nature of cancer impact treatment?

In examining the principles of cancer development and trying to answer these questions, you will come to understand the following:

- Each cancer is an independently evolved disease that has circumvented multiple limits in order to exist.
- Basic biological barriers are breached in cancer development. Specifically:
 - The expression and activity of cell cycle regulators is tightly controlled in normal cells, and this regulation is derailed in cancers.
 - The normal responses to excessive proliferation, including apoptosis and senescence, are abrogated in cancers.
 - Evolution of a cancer often requires an abnormal shift in differentiation status and abnormal induction of angiogenesis.
 - The immune system normally eliminates abnormal cells, and cancer cells evolve mechanisms to become invisible to this surveillance.
- We have identified common (but not necessarily ubiquitous) mechanisms by which cancers circumvent these barriers.
- Development of treatments is an ongoing challenge.

We'll talk about what it means to "understand" and how you demonstrate understanding throughout the semester.

Course activities:

To achieve these goals, we are going to engage in three major types of activities.

1. We will read and discuss much of Robert Weinberg’s excellent text *The Biology of Cancer*, which lays out a concise and data-driven history of our understanding of cancer. I will lead these discussions.
2. We will read, discuss, and analyze about twelve primary research articles that extend our understanding of ideas in *The Biology of Cancer*. These discussions will also allow you to better understand how the scientific community builds new knowledge about a disease. I will lead the first of these discussions, and groups of two or three students will lead the remainder.
3. We will construct “synthesis maps” as visual representations of a model of carcinogenesis. One of the challenges in understanding a complex process like carcinogenesis is fitting the different components into a coherent whole. By constructing visual representations of our model (which, by definition, changes in response to new knowledge), we will clarify and structure our growing understanding of carcinogenesis. The figure at right is a model of epithelial inflammation and tumor promotion, and provides one example of how to construct a synthesis map.

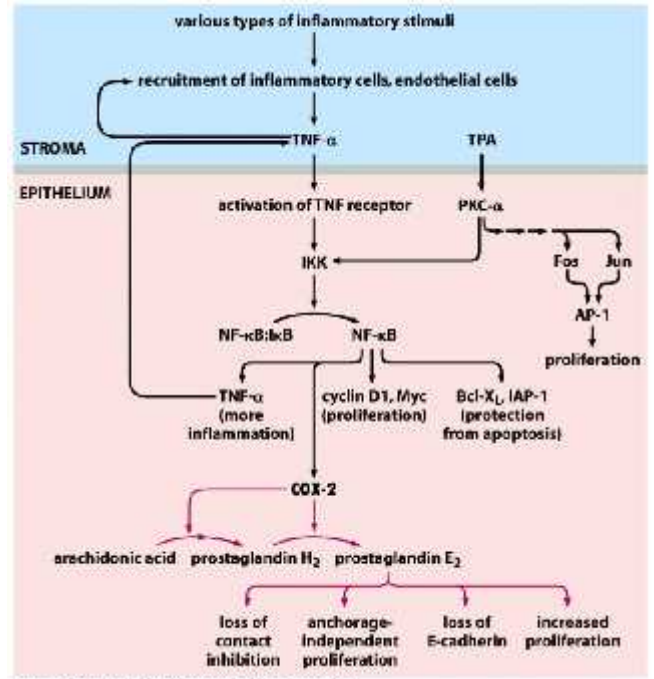


Figure 11-41 The Biology of Cancer (© Garland Science 2007)

Method of assessment

I will assess your growing understanding of cancer via two tests during the semester; your performance as a discussion leader; your participation in discussions; your synthesis maps; and a final paper. Specifically, your grade will be based on the following distribution:

Exam I	100 points
Exam II	100 points
Performance as discussion leader	100 points
Participation	75 points
Final paper	100 points
Synthesis maps (5)	75 points

I will use the following grading scale:

93-100	A	90-92	A-	87-89	B+	83-86	B	80-82	B-
77-79	C+	73-76	C	70-72	C+	67-69	D+	63-66	D
60-62	D-	0-59	F						

Note: The Vanderbilt Honor Code governs all exams, including any make-up exams, on which no assistance may be given or received.

OAK

I will post papers, Ppts, discussion questions, and announcements on OAK. Please check OAK regularly for assignments and updates.

Dropbox

I will send you an invitation to join a shared Dropbox folder on Friday, January 11. The folder will contain a schedule on for our paper discussions. You can sign up for the paper for which you wish to lead discussion. Be sure to save and close immediately.

Reading the text

Reading the textbook is an important way that you'll gain new information in this class. In order to make your reading more productive, and therefore more efficient, it's important to consider *how* you do the reading.

This textbook is well-written and relatively easy to read. Nonetheless, it is very information-dense, and therefore requires attention and memory aids to help you get the most out of it. Here is what I recommend:

1. Preview the chapter: read the introduction (prior to section 1 of the chapter) and the list of key concepts at the end of the chapter. If there are key concepts that seem particularly surprising or unclear, make a note.
2. As you read each section, note the main point of that section, and if relevant, evidence that supports that main point (i.e., what experiment tells you the main point is true?). A sketch of the experimental results or a note of the relevant figure can be very helpful.
3. Summarize the content of the chapter in one or more figures *that you draw*. The human brain comprehends, stores, and accesses visual images much more efficiently than it does words, so this summary provides a concise study guide that you can use later. In addition, the process of converting textual information to visual information allows you to make connections that help you to remember and help you to be able to transfer this knowledge to new situations.
4. It's helpful to go another step and integrate the main points/notes of evidence you generated in #2 with the visual images you created in #3.
5. Ask yourself what you understand, what is unclear, and which processes helped the most with your understanding. Bring your questions to class and/or office hours.

Reading papers

Reading primary research articles is another important way you'll gain new information in this class. Again, considering *how* you do the reading can help you be more productive and efficient. In general, when you are reading a paper, you should have two foci: What do we know from this paper? How do we know it? As you read the paper, you will want to take notes that will help you answer these questions. Here are my specific suggestions:

1. Read the introduction. Note the experimental question the authors are asking as well as any key pieces of data informing that question.
2. Read the results section. As you read the text of the results, refer to the figures. For each figure, ask yourself, "How did the authors do this experiment? What can I conclude from these results?" To answer the first of these questions, you may need to refer to the Materials and Methods section, which describes experimental procedures. Be sure to jot down notes that will help you remember the key information in each figure, as well as interpretations of unclear abbreviations. If you notice any discrepancies or points of concern, note those as well.
3. When you finish the results section, ask yourself: What do I know from the paper at this point?
4. Read the Discussion section. Do the authors' conclusions match your own? Do they introduce any interesting insights that didn't occur to you?
5. If you are the discussion leader, generate 6-10 discussion questions to distribute to the class. If you are not the discussion leader for this paper, read and jot down answers to the discussion questions provided by the leaders.

Leading discussion

As the discussion leader, your job is to help the class as a whole digest the paper and to put it into the larger context of carcinogenesis. You will be working in a team of two or three to lead discussion. Your goals while leading discussion are 1) to provide background to help put the paper and the experimental question asked into context; 2) to identify the key experiment(s) in the paper to focus discussion; 3) to facilitate discussion/interpretation of the paper by multiple (if not all) class members; 4) to provide a summary that helps the class put the results into the larger context of carcinogenesis; 5) to provide visual aids for the discussion during the class, usually in the form of a Powerpoint presentation with key experimental results shown.

To help you do these things, you will need to do a couple of things. **First, your group will need to meet with me at least one week prior to the class session in which you will lead discussion.** The purpose of this meeting is to allow you to clear up any questions you have about the paper and to get some formative feedback about your interpretation of the paper. **Second, you should submit a list of 6-10 discussion questions to me at least four days prior to the class session in which you will lead discussion.** The goal of these discussion questions is to focus class members' attention on the most important aspects of the paper, which should enrich the discussion.

I will use the following rubric to evaluate your discussion leadership:

Pre-discussion meeting	9-10 points Attended pre-discussion meeting; was prepared, participatory, and responsive.	5-8 points Attended pre-discussion meeting, but was ill-prepared or non-participatory.	1-4 points Attended pre-discussion meeting but was obviously unprepared	0 points Did not attend meeting
Discussion questions	9-10 points Provided 6-10 discussion questions that focus attention on key elements of paper; met deadline of four days prior to discussion. Revised questions if requested.	5-8 points Provided discussion questions, but a) did not focus on key elements; b) were provided fewer than 4 days prior to discussion; c) provided fewer than 6 questions; or d) failed to revise questions if requested.	1-4 points Provided discussion questions but failed to do several of the elements noted at left.	0 points Did not provide discussion questions.
Discussion: Providing background and context for question	9-10 points Provided clear and useful background and context	5-8 points Provided some background, but reason for question somewhat unclear	1-4 points Background unclear and confusing	0 points Did not attempt to provide background for paper
Discussion: Engaging students in interpretation of key experiments	14-15 points Engaged multiple students in interpreting experiments. Asked questions to extend discussion (e.g., are there different interpretations possible? What were the key controls in this experiment?)	9-13 points Provided most interpretation rather than promoting discussion and/or allowed only a few students to interpret experiments and/or did not ask follow-up questions.	3-8 points Provided all interpretation, only asking students follow-up questions.	0-2 points Did not engage students in interpretation; was dismissive of students' interpretations

Discussion: Providing summary and context for results	9-10 points Provided clear and useful summary and context for new results	5-8 points Summary or context unclear	1-4 points Both summary and context unclear	0 points No attempt to provide summary or context for results
Discussion: Visual aids	9-10 points Clear visual aids with useful introductory and summary elements and key elements of figures highlighted	5-8 points Clear visual aids, but lacking introductory or summary elements or highlights of key elements in figures	1-4 points Visual aids unclear or lacking several of the elements noted at left	0 points No visual aids provided
Discussion: Managed time effectively	9-10 points Effectively managed time during discussion, allotting time for key figures and saving time for summary	0-8 points Less effective time management (e.g., no time for summary; key figure skipped for time reasons; etc.)		
Assessment by group members	0-25 points Each group member assesses the others on a 1-25 scale, citing the contributions that each group member made as well as any deficits in preparation or participation. Each group member also assesses herself, documenting her activities, contributions, and any deficits in preparation or participation. These assessments will be submitted to me via email no later than two days after the paper discussion. Scores are averaged for final score.			

Participation

Participation in this class is vital, particularly during paper discussion and during synthesis activities. Effective participation can take the form of active contribution and active listening. To help ensure fair assessment of your participation, we will design the rubric for grading participation together in class.

Synthesis maps

We are going to use Prezi to construct our synthesis maps, which you will submit for comments five times during the semester. After each of the first four submissions, you will receive comments from me and from two of your colleagues (and will therefore be responsible for providing comments to two of your colleagues), but no grade. The synthesis map will be graded after the final submission. Each submission may be one class day late without penalty; each additional day it is late results in a loss of 2 points from the final synthesis map grade. Although the first four submissions are not graded, please note that each submission is required, and each failure to submit the map will result in a loss of 15 points from the final synthesis map grade.

Final paper

Your final paper will address some aspect of the big questions we are tackling in this class: Are cancers newly evolved species? How does the evolutionary nature of cancer impact treatment choices? We will design the exact nature of the assignment together.

Schedule

The schedule on the next page is a guide for our activities this semester but may be modified as needed.

Day	Activity	Reading
Jan 7	Introduction to class Review: What is a species? How does evolution work? What would be evidence that a cancer is a new species?	
Jan 9	Assessment of background knowledge Review of assignments	
Jan 11	Overview of cancer	Ch. 2, sections 1-5
Jan 14	Causes of cancer and the mutation theory	Ch. 2, sect. 6-10; ch. 12, sect. 1-3, 7
Jan 16	Mutation theory and the role of DNA repair in cancer	Ch. 12, sections 9-12
Jan 18	Cellular oncogenes	Ch. 4
Jan 21	MLK Day; no class	
Jan 23	Cellular oncogenes (Paper discussion)	Cancer Research (2007) 67: 2800-2808
Jan 25	Growth factor receptors and cancer	Ch. 5
Jan 28	Catch up and synthesis	
Jan 30	Synthesis map due Growth factor receptors (paper discussion)	Cancer Research (2011) 71: 7587-7596
Feb 1	Cytoplasmic signaling networks and cancer	Ch. 6
Feb 4	Cytoplasmic signaling networks and cancer	Ch. 6
Feb 6	Cytoplasmic signaling	J. Neurosci (2012) 32: 15849-15858
Feb 8	Synthesis map due Tumor suppressor genes	Ch. 7
Feb 11	Rb	Ch. 8
Feb 13	Rb (paper discussion)	Cancer Research (2012) 72: 5418-5427
Feb 15	p53 and apoptosis	Ch. 9, sections 4-11, 13, 14
Feb 18	p53	PNAS doi: 10.1073/pnas.1212047110
Feb 20	Catch up and synthesis	
Feb 22	Synthesis map due Cell immortalization (not on Test 1)	Ch. 10
Feb 25	Test 1	
Feb 27	Dr. Joyce Johnson, pathology of cancer	
Mar 1	Test review and mid-semester feedback	
Mar 11	hTERT and cancer	Oncogene (2012) 1-11
Mar 13	Multistep tumorigenesis and the role of inflammation	Ch. 11
Mar 15	Role of inflammation in carcinogenesis (paper discussion) Drop and pass/fail deadline	PLOS One (2012) 7: e44658
Mar 18	Heterotypic interactions and cancer development	Ch. 13
Mar 20	Metastasis	Ch. 14
Mar 22	Catch up and synthesis	
Mar 25	Synthesis map due Heterotypic interactions and cell reprogramming (paper disc.)	Cancer Research (2010) 70: 6945-6956
Mar 27	Heterotypic interactions and cell reprogramming (paper disc.)	Cancer Research (2012) 72: 5130-5140
Mar 29	Heterotypic interactions and cell reprogramming (paper disc.)	PNAS (2011) 108: 4852-4857
Apr 1	Tumor immunology	Ch. 15
Apr 3	Tumor immunology	Ch. 15
Apr 5	Catch up and synthesis	Ch. 15
Apr 8	Dr. Jill Gilbert: Novel therapeutics Synthesis map due	
Apr 10	Test 2	
Apr 12	Test review and discussion of final	
Apr 15	Novel therapeutics: inhibition of angiogenesis (paper disc.)	Cancer Cell (2012) 21: 212-226
Apr 17	Vicki Viar: Novel therapeutics	
Apr 19	Cancer treatments (Dead week)	
Apr 22	Cancer treatments (Dead week)	

Rubric for grading synthesis maps
Worth 75 points total

Organization—Worth up to 25 points

Does the synthesis map organize information into a coherent model of cancer? Does it tell a coherent story that articulates a comprehensive understanding of elements that contribute to cancer?

Two elements: Relationships among concepts and articulation of relationships between a concept, related examples and supporting data

Relationships among major concepts:

Excellent: Relationships among major concepts are clear. 11-12 points

Good: Relationships among most concepts are clear, but a few relationships are muddy or unarticulated. 8-10 points

Fair: Relationships among some concepts are clear, but many relationships are muddy or unarticulated. 5-7 points

Poor: Synthesis map demonstrates little synthesis; relationships among most concepts are unclear. 0-4 points

Relationships between a concept, related examples, and supporting data

Excellent: The relationships between each concept and its examples and supporting data are clear. 12-13 points

Good: The relationships between most concepts and the examples and supporting data are clear, but there are 1-3 muddy points. 9-11 points

Fair: The relationships between many concepts and the examples and supporting data are clear, but there are 4-7 muddy points. 5-8 points

Poor: There are many relationships between concepts and supporting information that are muddy. 0-4 points

Accuracy—Worth up to 25 points

Is the synthesis map accurate?

Excellent: All information on the synthesis map is accurate (as far as we know based on representation in the text or current papers). 24-25 points

Good: Most information is accurate but there are a few inaccuracies. 20-23 points

Fair: There are more than three inaccuracies. 15-19 points

Poor: There are many inaccuracies. 0-14 points

Completeness—Worth up to 25 points

Is the synthesis map complete?

Excellent: The synthesis map includes all major concepts related to carcinogenesis covered in the class, the necessary supporting and/or explanatory information, and when appropriate, relevant examples. *(For example, I would identify proto-oncogenes as a major concept that requires supporting information and examples. I would identify the progressive nature of cancer as a major concept that requires explanatory information but not necessarily examples.)* 23-25 points

Good: The synthesis map omits one major concept related to carcinogenesis covered in the class or omits important supporting and/or explanatory information for one major concept or omits examples for a concept that requires them. 19-22 points

Fair: The synthesis map has two omissions of the types described in "Good." 15-18 points

Poor: The synthesis map has more than two omissions of the types described in "Good." 0-14 points

Organization			
Are the relationships between the major concepts clear?	Progressive nature		<p>Excellent: Relationships among major concepts are clear. 11-12 points</p> <p>Good: Relationships among most concepts are clear, but a few relationships are muddy or unarticulated. 8-10 points</p> <p>Fair: Relationships among some concepts are clear, but many relationships are muddy or unarticulated. 5-7 points</p> <p>Poor: Synthesis map demonstrates little synthesis; relationships among most concepts are unclear. 0-4 points</p>
	Accumulation of mutations		
	Protooncogenes		
	TSGs		
	Immortalization		
	Heterotypic interactions		
	Metastasis		
Are the relationships within a concept clear—between the concept, the related examples, and the supporting data?	Progressive nature		<p>Excellent: The relationships between each concept and its examples and supporting data are clear. 12-13 points</p> <p>Good: The relationships between most concepts and the examples and supporting data are clear, but there are 1-3 muddy points. 9-11 points</p> <p>Fair: The relationships between many concepts and the examples and supporting data are clear, but there are 4-7 muddy points. 5-8 points</p> <p>Poor: There are many relationships between concepts and supporting information that are muddy. 0-4 points</p>
	Accumulation of mutations		
	Protooncogenes		
	TSGs		
	Immortalization		
	Heterotypic interactions		
	Metastasis		

Accuracy			
Is the synthesis map accurate?	Progressive nature		<p>Excellent: All information on the synthesis map is accurate (as far as we know based on representation in the text or current papers). 24-25 points</p> <p>Good: Most information is accurate but there are a few inaccuracies. 20-23 points</p> <p>Fair: There are more than three inaccuracies. 15-19 points</p> <p>Poor: There are many inaccuracies. 0-14 points</p>
	Accumulation of mutations		
	Protooncogenes		
	TSGs		
	Immortalization		
	Heterotypic interactions		
	Metastasis		
Completeness			
Is the synthesis map complete?	Progressive nature		<p>Excellent: The synthesis map includes all major concepts related to carcinogenesis covered in the class, the necessary supporting and/or explanatory information, and when appropriate, relevant examples. 23-25 points</p> <p>Good: The synthesis map omits one major concept related to carcinogenesis covered in the class or omits important supporting and/or explanatory information for one major concept or omits examples for a concept that requires them. 19-22 points</p> <p>Fair: The synthesis map has two omissions of the types described in "Good." 15-18 points</p> <p>Poor: The synthesis map has more than two omissions of the types described in "Good." 0-14 points</p>
	Accumulation of mutations		
	Protooncogenes		
	TSGs		
	Immortalization		
	Heterotypic interactions		
	Metastasis		

Figures used in synthesis map from Figure 1.

Most images are from the textbook used for the course:

Weinberg, R.A. (2007). *The Biology of Cancer*. Garland Science: New York. Figures include 2-2, 3-7, 3-19, 4-6, 4-11, 5-12, 5-15, 6-12, 6-14, 6-16, 6-19, 6-29, 7-7, 7-17, 8-4, 8-6, 8-8, 8-10, 8-11, 8-12, 8-13, 8-19, 8-22, 8-24, 10-2, 10-6, 10-7, 10-8, 10-13, 10-16, 10-23, 10-25, 11-41, 12-1, 12-2, 12-20, 12-21, 13-1, 13-10, 13-14, 13-20, 13-21, 13-25, 13-27, 14-4, 14-15, 14-25, and Table 14-2

Other images are from the following sources:

Frame 4:

MicrobiologyBytes. Baculovirus-Host Interactions.

<http://www.microbiologybytes.com/virology/kalmakoff/baculo/baculohostinteract.html>.

Frame 6:

Zielinski, S. (2010). Henrietta Lacks' 'Immortal' Cells. Smithsonian.Com.

<http://www.smithsonianmag.com/science-nature/henrietta-lacks-immortal-cells-6421299/?no-ist>.

Frame 7:

<http://www.fanpop.com/clubs/oncology/images/6590365/title/angiogenesis-photo>.

Frame 8:

Figure 1 from Steeg, P.S. (2003). Metastasis is a complex, multistep process. *Nature Reviews Cancer* 3: 55-63.

Frames 11, 14, and 15:

Slides 4, 7,8 and 28 from the National Cancer Institute. Understanding Cancer Series.

<http://www.cancer.gov/cancertopics/understandingcancer/cancer/AllPages>.

Frame 20:

Box 1 from Martin, A., and Scharff, M.D. (2002). AID and mismatch repair in antibody diversification. *Nature Reviews Immunology* 2: 605-614.

Frame 21:

Figure 1 from Gerson, S.L. (2004). MGMT: its role in cancer aetiology and cancer therapeutics. *Nature Reviews Cancer* 4: 296-307.

Frame 22:

Molecular Biology Web Book, chapter 7. DNA Repair Mechanisms. <http://www.web-books.com/MoBio/Free/Ch7G.htm>.

Frame 23:

Smith J., Smith, K., and Mezardi, C. (2001). Atlas of Genetics and Cytogenetics in Oncology and Haematology.

<http://atlasgeneticsoncology.org/Deep/DoubleStrandBreaksID20008.html>.

Frame 27:

<http://staff.jccc.net/pdecell/evolution/mutations/mutation.html>

Frame 31:

Figure 3 from Nossal, G.J.V. (2003). The double helix and immunology. Nature 421: 440-444.

Frame 39:

<https://www.hinsdale86.org/staff/kgabric/Disease10/Cowden%20Syndrome/Index1.htm>

Frame 44:

<http://sunmoonx.blogspot.com/2013/11/med-s-04-pathology-07-neoplasia.html> and <http://en.wikipedia.org/wiki/P53>

Frame 64:

Cell Signaling Technology MAPK/Erk in Growth and Differentiation Signaling Pathway. <http://www.cellsignal.com/common/content/content.jsp?id=pathways-mapk-erk>.

Frame 71:

Figure 15-64 from Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., and Walter, P. (2008). Molecular Biology of the Cell. Garland Science: New York.

Frame 80:

Figure 15-68 from Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., and Walter, P. (2008). Molecular Biology of the Cell. Garland Science: New York.

Frame 81:

Signalway Antibody. Jak/Stat Pathway. <http://www.sabbiotech.com/a-112-Jak-Stat-Pathway.html>.

Frame 84:

Figure 1 from Bray, S.J. (2006). Notch signaling: a simple pathway becomes complex. *Nature Reviews Molecular Cell Biology* 7: 678-689.

Frame 85:

Figure 1 from Pasca di Magliano, M., and Hebrok, M. (2003). Hedgehog signaling in cancer formation and maintenance. *Nature Reviews Cancer* 3: 903-911.

Frame 86:

Figure 1 from Baylin, S.B., and Ohm, J.E. (2006) Epigenetic gene silencing in cancer—a mechanism for early oncogenic pathway addiction? *Nature Reviews Cancer* 6: 107-116.

Frame 87:

Box 1 from Guo, W., and Giancotti, F.G. (2004). Integrin signaling during tumour progression. *Nature Reviews Molecular Cell Biology* 5: 816-826.

Frame 99:

Figure 1 from Bullock, A.N., and Fersht, A.R. (2001). Rescuing the function of mutant p53. *Nature Reviews Cancer* 1: 68-76.

Frame 100:

Figure 1 from Meek, D.W. (2009). Tumour suppression by p53: a role for the DNA damage response? *Nature Reviews Cancer* 9: 714-723.

Frame 101:

Figure 4 from Meek, D.W. (2009). Tumour suppression by p53: a role for the DNA damage response? *Nature Reviews Cancer* 9: 714-723.

Frame 102:

Figure 8 from Knights, C.D., Catania, J., Di Giovanni, S., Muratoglu, S., Perez, R., Swartzbeck, A., Quong, A.A., Zhang, X., Beerman, T., Pestell, R.G., and Avantaggiati, M.L. (2006). Distinct p53 acetylation cassettes differentially influence gene-expression patterns and cell fate *The Journal of Cell Biology* 173: 533-544.

Frame 109:

<http://www.olympusfluoview.com/gallery/cells/hela/helacells.html>

Frame 128:

Figure 6 from Giannoni, E., Bianchini, F., Masieri, L., Serni, S., Torre, E., Calorini, L., and Chiarugi, P. (2010). Reciprocal activation of prostate cancer cells and cancer-associated

fibroblasts stimulates epithelial mesenchymal transition and cancer stemness. *Tumor and Stem Cell Biology* 70: 6945-6956.

Frame 129:

Figure 7 from Fiaschi, T., Marini, A., Giannoni, E., Taddei, M.L., Gandellini, P., De Donatis, A., Lanciotti, M., Serni, S., Cirri, P., and Chiarugi, P. (2012). Reciprocal metabolic reprogramming through lactate shuttle coordinately influences tumor-stroma interplay. *Tumor and Stem Cell Biology* 72: 5130-5140.

Frame 133:

Figure 5 from Antonyak, M.A., Li, B., Boroughs, L.K., Johnson, J.L., Druso, J.E., Bryant, K.L., Holowka, D.A., and Cerione, R.A. (2011). Cancer cell-derived microvesicles induce transformation by transferring tissue transglutaminase and fibronectin to recipient cells. *PNAS* 108: 4852-4857.

Figures used in synthesis map from Figure 2.

Most images are from the textbook used for the course:

Weinberg, R.A. (2007). *The Biology of Cancer*. Garland Science: New York. Figures include 2-23, 3-4, 3-19, 5-1, 5-10, 5-12, 6-12, 6-14, 6-22, 6-24, 6-26, 6-29, 8-8, 8-14, 8-22, 8-24, 8-28, 10-19, 10-23, 11-41, 12-8, 12-20, 12-21, 12-22, 12-32, 14-14, 14-15, 14-25.

Other images are from the following sources:

Frame 20:

Figure 2 from Freidberg, E.C. (2001). How nucleotide excision repair protects against cancer. *Nature Reviews Cancer* 1: 22-33.

Frame 36:

Figure 2 from Kolch, W., Kotwaliwale, A., Vass, K., and Janosch, P. (2002). The role of Raf kinases in malignant transformation. *Expert Reviews in Molecular Medicine* 4: 1-18.

Frame 38:

En.wikipedia.org/wiki/Growth_cone

Frame 56:

Figure 1 from Bullock, A.N., and Fersht, A.R. (2001). Rescuing the function of mutant p53. *Nature Reviews Cancer* 1: 68-76.

Frame 57:

Figure 4 from Meek, D. W. (2009). Tumour suppression by p53: a role for the DNA damage response? *Nature Reviews Cancer* 9: 714-723.

Frame 58:

Figure 8 from Knights, C.D., Catania, J., Di Giovanni, S., Muratoglu, S., Perez, R., Swartzbeck, A., Quong, A.A., Zhang, X., Beerman, T., Pestell, R.G., and Avantaggiati, M.L. (2006). Distinct p53 acetylation cassettes differentially influence gene-expression patterns and cell fate *The Journal of Cell Biology* 173: 533-544.

Figures used in synthesis map from Figure 3.

Most images are from the textbook used for the course:

Weinberg, R.A. (2007). *The Biology of Cancer*. Garland Science: New York. Figures include 2-2, 2-25, 3-19, 4-6, 4-10, 4-11, 4-13, 5-1, 5-10, 5-12, 5-17, 6-12, 6-14, 6-15, 6-24, 6-26, 6-29, 7-3, 7-7, 7-8, 7-17, 8-3, 8-10, 8-11, 8-12, 8-13, 8-14, 8-19, 8-24, 8-28, 10-10, 10-13, 10-14, 10-16, 10-19, 10-23, 11-8, 11-10, 11-28, 11-41, 12-1, 12-2, 12-20, 12-21, 12-22, 12-26, 12-31, 13-10, 13-14, 13-25, 13-42, 14-4, 14-15

Other images are from the following sources:

Frames 4, 6, 7, 9, 11, 17, 19, 20:

Slides 5, 6, 19, 20, 21, 26, 28, 29 from the National Cancer Institute. Understanding Cancer Series. <http://www.cancer.gov/cancertopics/understandingcancer/cancer/AllPages>.

Frame 51:

Cell Signaling Technology MAPK/Erk in Growth and Differentiation Signaling Pathway. <http://www.cellsignal.com/common/content/content.jsp?id=pathways-mapk-erk>.

Frames 53 and 54:

Figure 15-64 from Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., and Walter, P. (2008). *Molecular Biology of the Cell*. Garland Science: New York.

Frame 59:

Figure 15-68 from Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., and Walter, P. (2008). *Molecular Biology of the Cell*. Garland Science: New York.

Frame 75:

Figure 1 from Bullock, A.N., and Fersht, A.R. (2001). Rescuing the function of mutant p53. *Nature Reviews Cancer* 1: 68-76.