BIMM134 SPRING 2012

Lectures:

Tuesdays/Thursdays, April 3 – June 7 5:00 – 6:20 pm Center Hall 101 Podcast available

Exams:

Midterm: May 10, in-class, 40% of grade. Final: June 14, 7-10 pm, comprehensive, 60% of grade. Absolutely NO Make-Up Exams

Midterm review session: Wednesday, May 9, 5 -7 pm, venue to be determined. **Final review session**: Wednesday, June 13, 5-7 pm, venue to be determined.

Recommended Textbook:

The Biology of Cancer Robert A. Weinberg

Instructor: Jean Y. J. Wang, PhD Office Hour: Fridays 5-6 pm, Moores Cancer Center, Room 4328

ASEs: (Discussion sessions to be held in Weeks 2 to 10)

FullName	EmailAddress	Sessions
Chang, Aaron Yu-Yuan	ayc001@ucsd.edu	Fridays; 12-1, 1-2 pm; WLH 2114
Tsai, Aaron	aatsai@ucsd.edu	Wednesdays; 5-6, 6-7 pm; WLH 2112
Ding, Eric Yuxiao	yud001@ucsd.edu	Mondays; 2-3 pm; HSS 1315
Reddy, Kritika	kgreddy@ucsd.edu	Mondays; 12-1 pm; HSS 1315

BIMM134 Biology of Cancer

Learning Goals:

- 1. What is cancer? (15 lectures)
- 2. How does cancer kill? (2 lectures)
- 3. Cancer therapy (2 lectures)

Lectures

April 3 – Organization and Overview

What is Cancer?

- 1. Clinical types
 - 2. Solid tumors are complex tissues

April 5 – What are Cancer Stem Cells?

The right and the wrong concepts.

April 10 - What are the Cell-Intrinsic Characteristics of Malignancy?

Malignant and necessary characteristics of cancer initiating cells.

April 12, 17 & 19 – What are Cancer Genes and How are They Discovered?

Oncogenes and tumor suppressors – past, present and future.

April 19, 26 & May 1, 3 – How do Cancer Genes Deregulate Cell Proliferation?

<u>Mitogenesis</u> – Receptor tyrosine kinase, Ras (NF-1), MAP-kinase cascade, Myc, transcription. <u>Cell Growth</u> – Receptor tyrosine kinase, PI3K (PTEN), Akt, mTOR (TSC1/2), protein synthesis.

<u>Cell Cycle</u> – Cyclin-dependent protein kinases (p16lnk4a, p21Cip1), E2F (RB). <u>Terminal Differentiation & Senescence</u> – (p16lnk4a, p19Arf, p21Cip1, RB, p53). [*in parenthesis are tumor suppressor genes*]

<u>May 8 – Coupling Mitogenesis to Apoptosis: a Failsafe Mechanism of Tumor</u> <u>Suppression</u>

Activation of a single oncogene, or inactivation of a single tumor suppressor gene is not sufficient to convert a normal proliferating cell into a cancer initiating cell.

May 10 - In Class Midterm (40% of the grade)

May 15 & 17. What is Apoptosis and How do Cancer Cells Escape It?

Apoptosis is a form of cell death that is genetically programmed. Apoptosis is required for normal development, tissue homeostasis and the elimination of damaged cells. The genetic program of apoptosis is compromised, but not blocked, in cancer cells.

May 22 & 24. What Causes Genome and Transcriptome Instability in Cancer <u>Cells?</u>

Defects in DNA repair mechanisms – mismatch repair in HNPCC, homologous recombination repair in ovarian and breast cancer. RNA quality control – splicing, non-sense mediated decay.

May 29 & 31. Cancer Kills when It Colonizes Vital Organs

The unsolved problems of angiogenesis, invasion, metastasis and cachexia.

June 5 & 7. Cancer Therapeutics

Past and Present - Surgery, radiation, chemo, hormone antagonists, tyrosine kinase inhibitors.

Future – Genetics based treatment strategy, personalized cancer medicine.

June 14. FINAL EXAM (comprehensive, 60% of the grade)