Syllabus, BIOSC 2105: Cell Signaling
Spring Term, 2014
Instructor: Dr. Kirill Kiselyov (kiselyov@pitt.edu)
2 – 3:50 pm, Thursday
Crawford 241

Objectives: to provide an overview of the current concepts of cell signaling, and to understand the experimental design leading to their formulation. This course is based on analysis of current literature; the instructor will moderate the discussion, the group will define key components of the discussion, students will present analysis of the primary literature. At the end of the course, students will formulate questions that challenge the current paradigms in cell signaling.

Background reading: Alberts et al., Molecular Biology of the Cell

Week #	Date	Topic (click to see linked papers or presentations)	Presenter	Note
1	01/09	The central paradigm of cell biology and the definition of cell signaling. Components of a signaling machine. Examples of signaling. GPCR.	* Kirill Kiselyov	* We identify six things we want to know about GPCR and GPCR-driven signaling cascades.
2	01/16	Odor perception and coding, cAMP-dependent signaling	Anthony Amarosa Amber Griffith	
3	01/23	ER Ca depletion and Ca influx, GAPs	KK posted Chong Dai	
4	01/30	Quantitative properties of G protein signaling networks G protein regulation * New discussion: receptors with enzymatic activity. RTK. Signaling by proximity.	Shibin Mathew Sarah Smith * Kirill Kiselyov	* We identify five things we want to know about RTK

				and RTK-driven signaling cascades.
5	02/06	Endocytosis of receptors Growth factor receptors and calcium signaling	Anthony Amarosa Amber Griffith	
6	02/13	Receptor phosphatases Regulation of receptors	Sarah Smith Chong Dai	
7	02/20	Scaffold proteins RTK followup * New discussion: long term effects of GPCR signaling	Shibin Mathew Chong Dai * Kirill Kiselyov	* We identify five things we want to know about how GPCR regulate gene expression.
8	02/27	CREB regulation of target genes CaMK	Sarah Smith Chong Dai	
9	03/06	CREB regulation of target genes Consequences of the removal of auto inhibitory region of CaMKII Does the interchange of CaMKII subunits regulate its activity CaM sensitivity to Calcium fluctuations Examples of CaMK signaling	Sarah Smith: follow up Chong Dai: follow up Tim Mackie Amber Griffith Anthony Amarosa	

		influencing transcription		
10	03/20	cancelled		
11	03/27	Growth factor signaling: how do growth factors regulate cell growth? MAPK and cell death	Shibin Mathew Amber Griffith	We identify five things we want to know about how RTK regulate gene expression.
12	04/03	How Do Growth Factors Stimulate Cell Growth？ The Role of P27 in The Assembly of Cyclin D1-Cdk4 Complex mTOR, autophagy and development	Chong Dai: Chong Dai: follow up Anthony Amarosa	
13	04/10	Temporal coding of Growth Factor Signals. mTORC1 and mTORC2 * New discussion: other receptors with enzymatic activity. Signaling and cell death and proliferation.	Shibin Mathews Anthony Amarosa: follow up. * Kirill Kiselyov	*We identify five things we want to know about signaling in cell death, proliferation and development.
14	04/17	Prepare and send questions; questions assigned		
15	04/24	Last round of presentations		

Exam questions:

1. Aside from keeping PKA physically inactivated, how else is AKAPS important for regulating PKA signaling in cells?

2. RTKs and GPCRs are both capable of utilizing Ca2+ to relay their signaling cascades. Compare and contrast how these two signaling pathways incorporate Ca2+ in relaying their messages within cells.

3. What is the importance of “signaling hubs” in RTK signaling?

4. How do GAPs prevent cancer occurrence?

5. How does CaMKII slow intracellular [Ca] decline?

6. What is the significance of the interaction between EGFR and Notch signaling pathways for neural cells.

7. How does Ca2+ signaling initiate embryonic development in response to sperm fertilization? What changes occur in the cell and is expression of maternal RNA linked to this calcium signaling event.

8. Both GPCRs and RTKs can trigger PLC to hydrolyze PIP2 to create IP3 and DAG. We learned in class that IP3 goes on to initiate Ca2+ release, but what happens to DAG and what roles does it play in the cells.

9. How are the different Ca2+ oscillation frequencies generated by the cell?

10. Explain how cells sense changes in nutrient availability to activate growth or autophagy. Hint: Discuss bi-directional transport of amino acids across cell membrane and how it depends on nutrient availability (i.e. presence and absence of nutrients). Also, explain if this mechanism changes in tumor cells.

11. Explain how amplitude, frequency and duration of Ca2+ signaling influences (1) NFAT de-phosphorylation and (2) NFAT nuclear translocation in cardiac cells.

12. Explain in short how differences in ERK duration gets decoded to c-Fos mediated activation of early- and late-response genes.

13. Does photoreceptor signaling follow any of the signaling models/cascades that we talked about this semester?

14. Can receptor signaling occur through non-genomic (i.e. without activating/repressing transcription) mechanisms?

15. How are gases as signaling molecules?