**Lecture 1: Introduction to Cancer Biology & Fundamentals of Biology – Dr. Chakravarty**

1. How cells are organized into tissue that make up an organ that forms a component of an organ system within an organism.
	1. Examples of organ systems
2. The Central Dogma: DNA – RNA – protein
	1. Transcription: DNA - RNA
	2. Translation: RNA – Protein
	3. Codes and anticodons – know how to read the genetic code table
3. Know that cancer is a collection of different diseases
	1. Know the definition of the followings
		* Carcinomas
		* Leukemias
		* Lymphomas
		* Sarcomas
4. Know that cancer is a genetic disease due to mutations in the DNA that lead to changes in the RNA and consequently the protein that creates a different phenotype
5. Know what cancer driver mutations and passenger mutations are
6. Know that different types of cells and stroma (supportive tissue surrounding cells) make up the cancer microenvironment that surrounds cancer cells which affects the ability of cancer cells to grow and spread locally and distantly (distant spread = metastasis)
7. Gene Expression: All the cells in a person's body have the same DNA and the same genes
	1. However, the difference between cells in different tissues and organs is that the "expression" of the genes differs between cells
8. The Central Dogma: DNA – RNA – protein
	1. Transcription: DNA - RNA
	2. Translation: RNA – Protein
	3. Codes and anticodons – know how to read the genetic code table
9. DNA and RNA structure and properties
	1. Know the difference between DNA and RNA
	2. Know what DNA and RNA stand for
	3. Know the difference between mRNA and tRNA
10. Proteins – types and function
	1. Amino acids
	2. Polypeptide Chains
11. The human genome
	1. Chromosomes
	2. Homologous pairs
	3. Genes
	4. Alleles
	5. Introns
	6. Exons
	7. Histones
12. Mutations
	1. Somatic vs germline
	2. Point mutations (silent, missense, nonsense)
	3. Frameshift mutation
	4. Chromosomal mutations (duplication, deletion, inversion, translocation)
	5. Be familiar with the BCR-ABL1 translocation in CML
13. Oncogenes vs tumor suppressor genes

**Lecture 2: The Nature of Cancer: Hallmarks of Cancer – Dr Rodrigo Romero**

Be familiar with the hallmarks of cancer summary page at the end of the lecture slide deck. Also focus on the following:

1. Reviewing difference between tumor suppressors, oncogenes and protooncogenes
2. The role of P53
3. Function of telomeres, specifically in cancer
4. The role of angiogenesis in metastasis of primary tumors

**Lecture 3: Cancer as a Genetic Disease – Ms. Akano**

1. How do mutations lead to cancer?
2. Somatic vs germline mutations
3. DNA damage
4. DNA repair
5. Oncogenes
6. Tumor suppressors
7. Can you inherit cancer

**Lecture 4: Cell Cycle - Dr. Jose Reyes**

1. **Cell division is a fundamental property of all life, and it is necessary for the development and function of multicellular organisms.**
	1. During multicellular development, a single cell gives rise to trillions of cells through cell division. These cells specialize and collectively allow for the organism to function as a whole.
	2. In adult multicellular organisms, cell division is necessary to replace dead cells, therefore maintaining tissue homeostasis. Different tissues regenerate with different frequencies: epithelial tissues, such as the lining of the intestine are constantly shedding dead cells and replacing them with new ones.
	3. Cell division is necessary to replace cells which have been lost through injury. For example, the liver has an outstanding regenerative capacity.
2. **In order to divide, cells transition through phases of growth, DNA replication and chromosome segregation. Collectively, these distinct phases form the cell cycle.**
	1. *G1 phase (first Gap):* Newborn cells enter this phase, during which they increase in size and prepare to replicate DNA.
	2. *S phase (Synthesis):* Cells replicate DNA. There are mechanisms to ensure that DNA is replicated only once per cell cycle.
	3. *G2 phase (second Gap):* Cells continue growing and prepare to divide, provided that there is no DNA damage.
	4. *M phase (mitosis)*: Cells break the nuclear envelope and condense their chromosomes to segregate them to opposite poles of the cell. The cytoplasm is then partitioned in half, yielding two nearly identical cells.
	5. The M phase lasts only ~1h. Cells spend most of their lives in G1, S and G2 phases. These three phases are collectively known as interphase.
3. **Cells integrate information from their internal state and their environment to proceed through the cell cycle, or halt cell cycle progression,**
	1. Cells have mechanisms to control halt or progress through the cell cycle depending on proliferative and antiproliferative signals.
	2. The presence of growth factors in the extracellular environment promotes cell cycle progression from G1 to S phase. The presence of DNA damage halts progression at all phases of the cell cycle. The presence of many neighboring cells also prevents progression through the division cycle (this is known as contact inhibition).
	3. Signal transduction pathways allow cells to:
		1. Sense the presence of these signals
		2. Relay this information through the intracellular environment, by chemically modifying proteins to change their function or activity.
		3. Generate a response, by inducing the production of protein which will promote or stop transition through specific phases of the cell cycle.
4. **Understanding the development of cancer through the lens of the cell cycle.**
	1. Errors in the cell cycle lead to genetic changes, which can generate cancer. These errors are more frequent during DNA replication (S phase) and chromosome segregation (M phase).
	2. Some genetic changes can alter signal transduction pathways to sustain aberrant proliferation. For example:
		1. Cells can become independent of the presence of growth factors to enter the S phase.
		2. Cells can stop responding to the presence of DNA damage by inactivating a protein that senses the DNA damage or relays this signal to other proteins.
	3. Frequently dividing cells are more prone to have errors in cell division just by chance. This in part can explain cancer incidence in different tissues.
5. **Understanding cancer therapy through the lens of the cell cycle.**
	1. Cancer cells tend to proliferate at a higher rate than their normal counterparts. This means that they will have a higher probability of being in S or M phase at any given time. Classic chemotherapeutic agents leverage this difference by inhibiting proper DNA replication and segregation, or directly damaging DNA.
	2. Some common side effects of chemotherapy are due to damage to fast proliferating normal cells, such as hair follicles or blood cells.
	3. DNA damaging therapies also bring about the risk of inducing genetic changes which may lead to another cancer.
	4. Cancer cells differ from their normal counterparts because they have specific mutations that allow them to sustain aberrant proliferation. Sometimes, they become dependent on the activity of these mutant proteins. Novel cancer therapies specifically inhibit the activity of these mutant proteins, and therefore target cancer cells selectively.

**Lecture 5: Liquid Tumors – Dr. Omar Abdel-Wahab**

1. Hematologic malignancies (or blood cancers) are cancers that arise from hematopoietic cells within the bone marrow, blood, or lymphatic system (lymph nodes and spleen).
2. The hematologic malignancies are divided into “myeloid” and “lymphoid” malignancies.
3. Myeloid malignancies include acute myeloid leukemia, myelodysplastic syndromes, and myeloproliferative neoplasms.
4. Lymphoid malignancies are divided into lymphoid leukemias and lymphomas and are cancers of the B- or T-lymphocytes.
5. The most common blood cancers are multiple myeloma (which are cancers of the plasma cells that normally produce antibodies) and diffuse large B cell lymphoma.
6. The most serious form of blood cancer, with the worst 5-year survival rates on average, are acute myeloid leukemia, acute lymphoblastic leukemia, and relapsed/refractory diffuse large B cell lymphoma.
7. Analysis of genetics is important in the diagnosis, risk prediction, and treatment selection of hematologic malignancies.
8. One example of an important genetic change driving blood cancer diagnosis and treatment that we discussed was identification of the Philadelphia chromosome which is a translocation between chromosomes 9 and 22. This results in an abnormally activated protein (the “ABL kinase”) which drives the development of certain forms of leukemia. There are now several drugs which effectively block the ABL kinase and leukemias with the Philadelphia chromosome.
9. A second example of an important genetic change driving blood cancer diagnosis and treatment that we discussed was the PML-RARA translocation that occurs with translocations of chromosomes 15 and 17. This genetic change causes the disease Acute Promyelocytic Leukemia which is a rapidly fatal leukemia if not identified quickly and treated properly, but >95% are cured if the disease is diagnosed and treated correctly. This disease is uniquely treated with the drugs All Trans Retinoic Acid and arsenic.