Test 4 Review

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1 Cancer

1.1 9 Hallmarks

Making more cells isn't enough for cancer to proliferate. These cells start by overdividing; but they will quickly get killed by the various regulatory checkpoints in our body, such as immune system or apoptosis. Thus they need to satisfy all the hallmarks.

1. Proto-oncogens mutate and become hyperactive: proto-oncogenes are genes that can turn into a cancer gene (AKA an oncogene). They usually help the cell go through cell cycle. An example is rtk, a protein associated with a proto-oncegene. (recall that it is the stick thing that has 3 branches, which combine to form a dimer when they receive a growth signal). Once the proto-oncogene becomes mutated, rtk will keep telling the cell to divide.

   See the sheet for more details on this process.

2. Anti-oncagenes mutate and become inhibited: For example, prb proteins, which are master breaks of the cell cycle, or p53, which are DNA repairers, stop working.

3. Apoptotic genes mutate. This blocks apoptosis and fail to trigger it. Example would be bcl-21 proteins block apoptosis, when they activate the cells no longer suicide.

4. Telomerase genes become active and extend telomeres to help cells bypass hayflick limit and make them immortal

5. Cells must acquire the ability to produce VEGF and other growth factors that stimulate growth of blood vessels to nourish tumor (a process sustained angiogenesis)

6. Metastatis: cancer cells separate from their intercellular junctions and invade other tissues.

7. Changes to metabolism. Some cells do anaerobic glyclyosis even in presence of oxygen. They can sometimes even use lactate acid as nutrients.

8. Avoiding immune destruction. Straightforward.
9. Phenotypic plasticity: Cancer cells de-differentiate and return to a more stem-cell like state.

   First six, every cancer cell has to have it. The remaining three, not fully neccessary.

1.2 Causes of mutation

1. Random mutations due to replication errors
2. Ionizing radiation
3. Mutagenic materials, such as arsenic in water
4. Viral infections promote proto-oncogenjes or deactivate tumor suppressor genes. HPV.

1.3 Cancer in old age

most peopole have cancer in old age because the longer you live, the more likely there is for a mutation to occur. It's a chance game.

1.4 Early cancer

1. inheritance of mutated proto-oncogenes (thus present in all cells!)
2. Inheritance of dfective DNA repair genes
3. different versions of detoxifying enzymes that can make more/less harmful molecules from original molecules, or work faster/slower
4. Impaired immune system
5. Exposure to carcinogens
6. disease

1.5 HPV

Uses proteins like E6 and E7 to interfere with anticogenes. For example, E6 binds to p53, which normally helps repair DNA damage or triggers cell death.

Definition 1 (Carcinogens)**.** *Covalent bonds between carcigones and DNA cause mutations*

2 Mitosis

2.1 Histones

Condenses the information of DNA (by letting them wrap around the histones).

2.2 Differentiation

Toti potent: can differentiate into any cell Pluri potent: can differentiate into certain

2.3 Chromos

Chromatin: Loosely packed DNA that exists during G and S phases (interphase). Chromatid: one of two identical halves of a replicated chromosome. when they split, each chromatid becomes an individual chromosome. Chromosomes: during interphase they exist as the less condensed chromatin.

2.4 Mitotic Spindle Fibers

Animal cells have centrioles; plant cells don't. However both cells can make centrosomes. The purpose of centrioles is thus unclear.

Definition 2 (Centrosome)**.** *Organizes the microtubules during cell division. During anaphase, they begin to move to the opposite poles of the cell.*