Chapter 7 Neoplasia

1. Introduction
    a. "Neo"=new; "Plasia"=growth
    b. Tumor
        i. Any abnormal cell growth
        ii. Malignant: cancer, altered cellular gene expression
        iii. Benign
2. Benign vs. Malignant Tumors
    a. Malignant
        i. Possibly fatal if untreated (because of the actual tumor)
        ii. Invasion, dissemination, metastases
        iii. Anaplasia (lacks differentiation) (it doesn't look like normal cells anymore)
        iv. Rapid growth, angiogenesis, necrotic, dysfunctional tissue
        v. Carcinoma: epithelial
        vi. Sarcoma: mesenchymal
        vii. Leukemia: WBC
    b. Benign
        i. Life-threatening if compressive or obstructive (if it takes up too much room)
        ii. Local, noninvasive, **encapsulated**
        iii. **Well differentiated** (doesn't invade adjacent tissue)
        iv. Slow growth
        v. "-oma"
3. Cancer Cell Characteristics
    a. Autonomous: proliferate without growth signals (independent of everything)
    b. Insensitive to growth inhibitory signals, evade apoptosis
    c. Unlimited replication
    d. Genetically unstable, rapid accumulation of mutations (too rapid, too many replications)
    e. Lose differentiated features, lose tissue function
    f. Invade local tissue, malignant cells migrate and metastasize
    g. Angiogenesis
4. Cancer Epidemiology
    a. Cancer is the 2nd leading cause of death in the U.S.
        i. 25% of general population

ii. 77% of population over 55 years
iii. Risk: men 1:2, women 1:3

b. 66% 5 year survival rate

5. Cancer Epidemiology: CDC
6. Cancer Risk Factors
    a. Lifestyle factors = 40-50% of deaths of cancer due to lifestyle choices (preventable and avoidable cancers)
        i. Contribute 1/3 of cancer-related deaths
        ii. Tobacco
        iii. Nutrition: (high) fat, (low) fiber, (high) alcohol, (low) antioxidants
        iv. Obesity
        v. Sun exposure
        vi. HPV – STD, normal contact with a patient can spread to health care workers, it does not have to always be sexually. HPV can cross condoms, so abstinence is the only way to 100% prevent contracting HPV. One type of strain causes "cauliflower" type of looking warts that protrude out of the vagina for females and out of the anus for males. A different strain can cause cancers (cervical, pharyngeal = oral sex, and penile).
            1. HIV- does not always cross condoms so it is considered "condom safe" but should be discussed and agreed upon with your partner.
    b. Early screening=early detection
7. Genetic Mechanisms of Cancer
    a. Carcinogens: potential cancer-causing agents
        i. carcin/o= cancer
        ii. –gens= creating
    b. Proto-oncogene: gain-of-function mutation normal genes- physiological code for signals/factors- we need them! They contribute for cellular growth. If they are overexpressed, it means they have gain too much expression and hyperactivate. These become oncogenes- which are abnormal and cause cancer.
        i. Oncogenes: overactive proto-oncogene mutations of proto/
            1. Retrovirus- HIV
    c. Tumor suppressor gene: loss-of-function mutation- initiate apoptosis, the genes must be there for it to occur, if not there than apoptosis cant occur and cells will accumulate. So they are normally suppose to be there, if not then they are deficient or defective.
8. Genetic Mechanisms of Cancer
    a. Proto-oncogenes

i. Normal genes that are transformed into oncogenes by activating mutations
ii. Proto-oncogenes normally code for: growth factors (mitogens), growth factor receptors, cytoplasmic signaling pathways, nuclear transcription factors

9. Genetic Mechanisms of Cancer
    a. Oncogenes
        i. Mutations alter proto-oncogene activity by abnormally enhancing proliferation-promoting signals, forming activated oncogenes
        ii. 4 paths of activation:
            1. Oncogenes introduced into host by retrovirus
            2. Intracellular proto-oncogene suffers mutagenic event
            3. Abnormal proto-oncogene activation via DNA loss/damage
            4. Chromosome replication errors form extra copies of proto-oncogenes (amplification)- produce more and wont stop
    b. Tumor suppressor genes
        i. Contribute to physiological inhibitory pathways
        ii. Cancer develops when both maternal and paternal copies are defective: chromosomal deletions, point mutations, chromosomal nondisjunction
        iii. Rb gene, p53, BRCA-1/BRCA-2
            1. Rb Gene- causes retinal blastoma- tumor in the retina of the eye
            2. P53- breast/colon cancers
                a. BRCA 1/2- breast cancer, affect both male and female, being more severe in males. Also causes ovarian cancer in females too which can lead to both a hysterectomy and a mastectomy in that affected female

10. Multistep Nature of Carcinogenesis
    a. Initiation –start of production of dysplastic or anaplastic cells
        i. Initiating events: inappropriately activate proto-oncogenes and inactivate tumor suppressor genes
        ii. Carcinogens: complete/partial
            1. Partial: needs to be combined with some other factor, tendency to be combined with tumor (genetic) suppressor gene, or environment.
                a. Ex. You have the partial gene and add in a bad lifestyle diet=cancer
            2. Complete: inherit the right mutation to form cancer
                a. Inherit colon cancer