Robbins Review Questions - Chapter 7

1. Cancer is the _____ cause of death in the US.
    a. Leading
    b. Second leading
    c. Third most common
    d. Fourth most common
2. What are the two basic components of any tumor? Classification of the tumor is dependent on which component?
3. What is desmoplasia?
4. Desmoplastic tumors that are “stony hard” are often referred to as
    a. Rock hard
    b. Fibrotic
    c. Desmoplastic
    d. Scirrhous
5. Generally speaking, the suffix -oma describes a _____ (benign/malignant) tumor.
6. What term is applied to benign epithelial neoplasms derived from glands, that may or may not form glandular structures?
7. When a neoplasm—benign or malignant—produces a macroscopically visible projection above a mucosal surface and projects, for example, into the gastric or colonic lumen, it is termed a/an
    a. Adenoma
    b. Carcinoma
    c. Polyp
    d. Sarcoma
8. What is the nomenclature associated with tumors arising in mesenchymal tissues, epithelial tissues and blood forming tissue?
9. Define hamartoma and choristoma.
10. Define differentiation and anaplasia. Which of these is a hallmark of malignancy?
11. What are common morphologic changes are associated with is anaplasia often associated that are also hallmarks of malignancy?
12. Which of the following features is not seen in malignant tumors?
    a. Local invasion
    b. Metastasis
    c. Confined within/by the basement membrane
    d. None of the above
13. _____ (Metaplasia/Dysplasia) is defined as the replacement of one type of cell with another type and is nearly always found in association with tissue damage, repair, and regeneration. In contrast, _____ (metaplasia/dysplasia) means “disordered growth.” It is encountered principally in epithelia and is characterized by a constellation of changes that include a loss in the uniformity of the individual cells as well as a loss in their architectural orientation.
14. When dysplastic changes are marked and involve the full thickness of the epithelium, but the lesion does not penetrate the basement membrane, it is considered a preinvasive neoplasm and is referred to as _____.
15. What are the two most reliable features to determine a tumor as malignant?

16. What are the three pathways through which cancers spread?
17. Lymphatic spread of tumor cells is the most common pathway for the initial dissemination of _____ (carcinomas/sarcomas/leukemias).
18. Which of the following is known as the first node in a regional lymphatic basin that receives lymph flow from the primary tumor?
    a. Primary lymph node
    b. First lymph node
    c. Metastatic lymph node
    d. Sentinel lymph node
19. What are the 3 most common tumor types in men and in women in the US?
20. What are seven environmental risk factors which lead to increased cancer risk?
21. Acquired conditions which predisposes an individual to cancer include all of the following except?
    a. Precursor lesions
    b. Chronic inflammatory state
    c. Immunodeficiency state
    d. All of the above
    e. None of the above
22. A tumor is formed by the _____ (polyclonal/clonal) expansion of a single precursor cell that has incurred _____ (lethal/nonlethal) genetic damage.
23. Describe the eight fundamental changes in cell physiology which are considered the hallmarks of cancer.
24. Genes that promote autonomous cell growth in cancer cells are called _____. These have the ability to promote cell growth in the absence of normal growth-promoting signals.
    a. Oncogenes
    b. Proto-oncogenes
    c. Telomerases
    d. Cyclins
25. What is the “two-hit” hypothesis of cancer progression?
26. What is the most frequently mutated gene in human cancers?
27. In stressed cells, p53 is released from inhibition when triggered by
    a. DNA damage and hyperoxia
    b. DNA damage and proto-oncogene activation
    c. Proto-oncogene activation and hypoxia
    d. DNA damage, hypoxia and oncogene activation
28. What are the two effects of activated p53 on neoplastic transformation?
29. What is Li-Fraumeni syndrome?
30. Mutations in the tumor suppressor gene _____ are associated with familial adenomatous polyposis where individuals born with one mutant allele develop thousands of adenomatous polyps in the colon during their teens or 20s.
    a. P53
    b. RB
    c. APC
    d. MYC

31. Loss of function mutations of the ____ gene are associated with hereditary renal cell cancers, pheochromocytomas, hemangioblastomas of the central nervous system, retinal angiomas, and renal cysts.
    a. E-cadherin
    b. VHL
    c. APC
    d. NF2
32. What is the Warburg effect and what is its significance in tumor cells?
33. Of the two pathways of apoptosis, it is the _____ (intrinsic/extrinsic) pathway, sometimes referred to as the mitochondrial pathway, that is most frequently disabled in cancer.
34. What is the major driving force of neoplastic angiogenesis?
35. What is anoikis?
36. What is the proper order of events that underlie invasion of tumor cells prior to metastatic spread? (place in order from first to last)
    a. Degradation of basement membrane and interstitial connective tissue
    b. Migration
    c. Attachment to novel ECM components
    d. Dissociation of cancer cells from one another
37. Adhesion molecules that allow epithelial cells to bind to each other are known as _____.
38. Individuals with HNPCC are prone to developing cancers of the
    a. Head and neck
    b. Lung
    c. Colon
    d. Prostate
39. What is the difference between an initiator and a promoter?
40. What type of UV radiation has the greatest association for increased risk of the development of skin cancers?
41. UV damage increases cancer risk by leading to the DNA damaging formation of
    a. Pyrimidine dimers
    b. Viral tax protein
    c. TNF and EGF
    d. Mutated p53
42. HTLV-1 is a retrovirus that ultimately causes _____ (B cell/T cell) leukemia/lymphoma.
43. Which forms of HPV are “high risk” and have been implicated in the genesis of squamous cell carcinomas of the cervix, anogenital region and head and neck?
    a. 1 and 2
    b. 4 and 7
    c. 6 and 11
    d. 16 and 18
44. Burkitt lymphoma is a type of cancer whose development is related to which of the following oncogenic viruses
    a. HPV 16
    b. HPV 18

    c. Epstein-Barr virus
    d. Kaposi sarcoma virus
45. What bacteria is implicated in the development of gastric adenocarcinomas and gastric lymphomas?
46. These 2 viruses are associated with 75-80% of hepatocellular carcinomas.
47. Progressive loss of body fat and lean body mass accompanied by profound weakness, anorexia, and anemia is known as
    a. Anorexia
    b. Starvation
    c. Cachexia
    d. Wasting
48. What is paraneoplastic syndrome?
49. _____ (Grade/Stage) of a cancer is based on the degree of differentiation of tumor cells while the _____ (grade/stage) of a cancer is based on the size of the primary lesion/depth of invasion, extent of spread to lymph nodes and presence/absence of metastasis.
50. Detecting cell products or surface markers using specific antibodies is known as
    a. Flow cytometry
    b. Immunohistochemistry
    c. Histology
    d. Cytology

1. B (pg 265)
2. Neoplastic cells which make up the tumor *parenchyma* and *reactive stroma* made up of connective tissue, blood vessels, nerves and a variable number of cells from the immune system. Classification is dependent on the parenchymal component (pg 266)
3. Desmoplasia is the formation of fibrous connective (collagenous) tissue, stimulated by the growth of neoplastic parenchyma, typically occurring around the borders of a tumor giving it a firm/hard exterior (pg 266)
4. D (pg 266)
5. Benign (pg 266)
6. Adenoma (pg 266)
7. C (pg 266)
8. Sarcomas (mesenchymal), carcinomas (epithelial) and leukemias (blood-forming) (pg 266)
9. A hamartoma is a disorganized but benign mass composed of cells indigenous to the involved site. A choristoma is the term applied to a heterotopic rest of cells - aka normal cells outside their normal location/in an abnormal spot (pg 267)
10. Differentiation is the degree to which neoplastic tissue resembles its progenitor parenchyma. Anaplasia is a lack of differentiation and is a hallmark of malignancy (pg 268)
11. Pleomorphism (varied size and shape of cells), abnormal nuclear morphology (high nuclear:cytoplasmic ratio-approaching 1:1, hyperchromatic nuclei, variable shape of nucleus), frequent/irregular mitosis, loss of polarity and other changes (hemorrhage/necrosis) (pg 270)
12. C (pg 270-272)
13. Metaplasia. Dysplasia (pg 270-271)
14. Carcinoma in situ (pg 271)
15. Invasiveness and metastasis (pg 271-272)
16. Direct extension/seeding of body cavities and surfaces, lymphatic spread, hematogenous spread (pg 273)
17. Carcinomas (pg 273)
18. D (pg 273)
19. Men - prostate, lung and colon/rectum. Women - breast, lung, colon/rectum (pg 275)
20. Infectious agents, smoking, alcohol consumption, diet, obesity, reproductive history, environmental carcinogens (pg 277-278)
21. D (pg 278-279)
22. Clonal. Nonlethal (pg 280)
23. Eight fundamental changes as follows:
    a. *Self-sufficiency in growth signals* - tumors have the capacity to proliferate without external stimuli, usually as a consequence of oncogene activation.
    b. *Insensitivity to growth-inhibitory signals* - tumors may not respond to molecules that inhibit the proliferation of normal cells, usually because of inactivation of tumor suppressor genes that encode components of these growth inhibitory pathways.

c. *Altered cellular metabolism* - Tumor cells undergo a metabolic switch to aerobic glycolysis (called the Warburg effect), which enables the synthesis of the macromolecules and organelles that are needed for rapid cell growth.
d. *Evasion of apoptosis* - Tumors are resistant to programmed cell death.
e. *Limitless replicative potential (immortality)* - Tumors have unrestricted proliferative capacity, a stem cell–like property that permits tumor cells to avoid cellular senescence and mitotic catastrophe.
f. *Sustained angiogenesis* - Tumor cells, like normal cells, are not able to grow without a vascular supply to bring nutrients and oxygen and remove waste products. Hence, tumors must induce angiogenesis.
g. *Ability to invade and metastasize* - Tumor metastases are the cause of the vast majority of cancer deaths and arise from the interplay of processes that are intrinsic to tumor cells and signals that are initiated by the tissue environment.
h. *Ability to evade the host immune response* - You will recall that the cells of the innate and adaptive immune system can recognize and eliminate cells displaying abnormal antigens (e.g., a mutated oncoprotein). Cancer cells exhibit a number of alterations that allow them to evade the host immune response (pg 282-283)

24. A (pg 283)
25. Using the retinoblastoma (RB) gene as an example: two mutations (hits) involving both alleles of RB, are required to produce retinoblastoma. In familial cases, children inherit one mutated copy of the RB gene and one normal copy - RB develops when the second, normal gene undergoes spontaneous mutation (i.e. takes a “hit”). In sporadic cases, children have two normal RB genes and both must undergo somatic mutation (ie takes two “hits”) before RB function is lost and cancer develops (pg 291)
26. TP53 (pg 294)
27. D (pg 294)
28. Induces cell cycle arrest and (either transient or permanent) or initiates apoptosis (pg 294)
29. Inheritance of a mutated copy of TP53 which predisposes individuals to malignant tumors because only one additional “hit” in the remaining normal allele is needed to stop TP53 function (pg 294)
30. C (pg 296)
31. B (pg 299)
32. The Warburg effect is the preference for cells, even in the presence of oxygen, to uptake glucose and utilize aerobic glycolysis for cellular metabolism. While not unique only to cancer cells (i.e. all growing cells demonstrate this to a degree), this type of cellular metabolism provides many metabolic intermediates (ie lots of carbon and hydrogen) for growth that are not available in the more energy efficient form of mitochondrial oxidative phosphorylation (pg 300)
33. Intrinsic (pg 302)
34. Hypoxia (pg 306)
35. Apoptosis stimulated by cells losing adhesion to one another (pg 308)
36. D, A, B, C (pg 306-307)
37. Cadherins (pg 306)

38. C (pg 314)
39. An initiator is a sufficient dose of a carcinogenic agent; an initiated cell is altered, making it potentially capable of giving rise to a tumor due a permanent alteration in its DNA. Initiation alone, however, is not sufficient for tumor formation. Promoters can induce cancerous proliferation, but only if the cell has been previously exposed to an initiator (pg 322)
40. UBV (pg 324)
41. A (pg 324)
42. T cell (pg 325)
43. D (pg 326)
44. C (pg 327)
45. Helicobacter pylori (pg 329)
46. Hepatitis B & C (pg 328)
47. C (pg 330)
48. Signs and symptoms that cannot be readily explained by the anatomic distribution of the tumor or by the elaboration of hormones indigenous to the tissue from which the tumor arose. Eg) small cell lung cancers may secrete ACTH and cause Cushing syndrome (pg 330)
49. Grade. Stage (pg 332)
50. B (pg 334)