QUESTION

Which of the following measures are associated with an increased life span?

A Moderate or regular exercise for 30 min

B Decrease stress

C **Decreasing calorie intake by 30 percent** CORRECT

D Pharmacological intervention with proton pump inhibitors

RIGHT ANSWER

OK

C. Decreasing calorie intake by 30 percent

WHY THIS IS RIGHT

Caloric restriction is the most consistently proven intervention to increase lifespan in multiple experimental models.
Reducing calorie intake by about 30% slows metabolic rate and decreases oxidative stress.
It activates longevity pathways involving sirtuins and AMPK, delaying cellular aging.

Exercise and stress reduction improve health span, but calorie restriction shows the strongest lifespan extension effect.

QUESTION

Which of the following proteins functions as a pro-apoptotic factor?

A MCL

B BCL-2

C **BAX** CORRECT

D BCL-XL

OK RIGHT ANSWER

C. BAX

WHY THIS IS RIGHT

Cell stress / DNA damage
↓
BAX / BAK activation
↓
Mitochondrial membrane permeabilization
↓
Cytochrome c release
↓
Caspase activation
↓
Apoptosis

BAX is a pro-apoptotic member of the BCL-2 family that promotes mitochondrial outer membrane permeabilization. It facilitates release of cytochrome c from mitochondria, activating the intrinsic apoptotic pathway.
This leads to caspase activation and programmed cell death.
BCL-2, BCL-XL, and MCL-1 are anti-apoptotic proteins that inhibit mitochondrial apoptosis.

Pro-apoptotic Genes (BH1-3)	Anti-apoptotic Genes	Apoptosis Initiators/Sensors
BAK gene	BCL-2 gene (most important)	BIM gene
BAX gene	BCL-XL gene	BAD gene
p53 gene	MCL1 gene	PUMA gene
Glucocorticoids	Sex (Love) steroids	NOXA gene

QUESTION

Which of the following statements is false regarding neutrophil extracellular trapping (NET)?

A It is detected in blood during sepsis

B It is produced in response to bacterial infection.

C **Mitochondrial DNA is seen.** CORRECT

D It is chromatin with antibacterial enzymes.

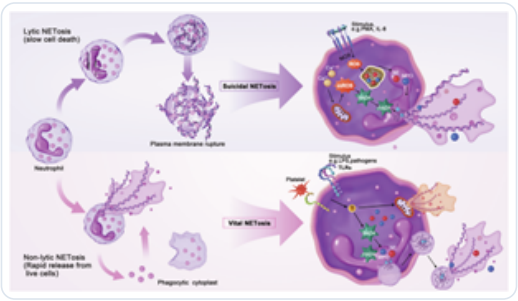
OK RIGHT ANSWER

C. Mitochondrial DNA is seen.

WHY THIS IS RIGHT

Bacterial infection
↓
Neutrophil activation
↓
Nuclear chromatin decondensation
↓
DNA + enzymes released
↓
NET formation -> traps microbes

Neutrophil extracellular traps (NETs) consist of decondensed nuclear chromatin decorated with antimicrobial enzymes like elastase and myeloperoxidase.
They are released in response to bacterial infections and are detectable in sepsis.
NETs are composed primarily of nuclear DNA, not mitochondrial DNA.
Hence, the statement regarding mitochondrial DNA is false.



QUESTION

Choose the correct statement regarding the telomerase theory of aging?

A Telomere stability is associated with aging

B Abnormal telomerase activation is associated with aging

C **Decreased telomere length is associated with aging** CORRECT

D Increased telomere length is associated with aging

RIGHT ANSWER

OK

C. Decreased telomere length is associated with aging

WHY THIS IS RIGHT

Telomeres shorten progressively with each cell division due to incomplete replication of chromosome ends.
Reduced telomere length leads to replicative senescence and loss of cellular proliferative capacity.
This cumulative telomere attrition is a key mechanism underlying cellular aging.
Telomerase activity maintains telomere length in germ cells and cancers, not in normal aging cells.

Feature	Aging	Cancer
Telomere length	□ Decreased	Maintained/increased
Telomerase	Low	High
Cell fate	Senescence	Immortality

QUESTION

Caspases are involved in which of the following types of programmed cell death?

A Necroptosis and Apoptosis

B **Pyroptosis and Apoptosis** CORRECT

C Ferroptosis and Apoptosis

D Necrosis and Apoptosis

OK RIGHT ANSWER

B. Pyroptosis and Apoptosis

WHY THIS IS RIGHT

Caspases = cysteine proteases
• Exist as inactive precursors
• Get activated during programmed cell death

Caspases are central mediators of apoptosis, with initiator and executioner caspases driving programmed cell death.
Pyroptosis is a caspase-dependent inflammatory cell death mediated mainly by caspase-1.
Necroptosis and ferroptosis are caspase-independent forms of regulated cell death.
Hence, caspases are involved in apoptosis and pyroptosis.

QUESTION

Which of the following is not helpful for differentiating between necrosis and apoptosis?

A Intact cell organelle

B **Altered cell shape** CORRECT

C Enzyme mediated degradation

D Internal organelle disintegration with lipid change

OK RIGHT ANSWER

B. Altered cell shape

WHY THIS IS RIGHT

Both necrosis and apoptosis show altered cell shape, making it non-discriminatory.
Apoptosis preserves cell membrane integrity and organelles until late stages, unlike necrosis.
Apoptosis is enzyme-mediated via caspases, whereas necrosis is unregulated.
Necrosis shows organelle disintegration with lipid peroxidation and membrane rupture.

Feature	Necrosis	Apoptosis
Cell size	Enlarged (swelling)	Reduced (shrinkage)
Nucleus	Pyknosis → karyorrhexis → karyolysis	Fragmentation into nucleosome-sized fragments
Plasma membrane	Disrupted	Intact; altered lipid orientation
Cellular contents	Enzymatic digestion	Intact
Inflammation	Frequent	Absent
Role	Invariably pathologic	Often physiologic

QUESTION

Which type of programmed cell death primarily involves the action of caspase-1?

A Apoptosis

B **Pyroptosis** CORRECT

C Necroptosis

D Necrosis

RIGHT ANSWER

OK **B. Pyroptosis**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following statements about free radicals is incorrect?

A Lipid peroxidation

B SOD inactivates free radicals

C Produced by neutrophils

D **Causes Fibrinoid necrosis** CORRECT

RIGHT ANSWER

OK

D. Causes Fibrinoid necrosis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

All tissues are correctly matched to their stem cell location, except:

A Intestine - Paneth cells near base of crypt

B Skin - Bulge region of hair follicle

C Liver - Canal of Hering

D **Cornea - Ciliary body** CORRECT

RIGHT ANSWER

OK **D. Cornea - Ciliary body**

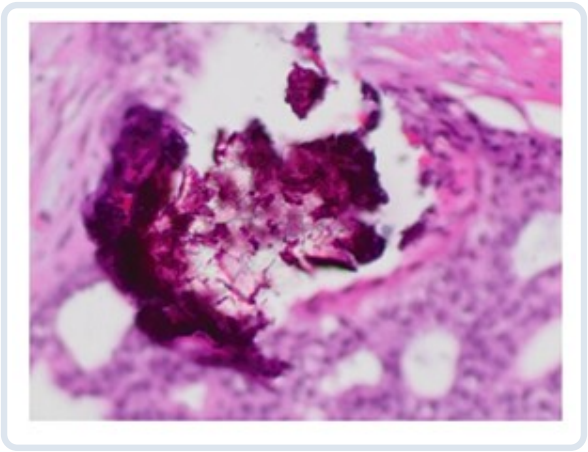
WHY THIS IS RIGHT

Adult stem cells reside in specific tissue niches responsible for regeneration and repair.
Correct stem cell locations:
• Intestine: Stem cells located at the base of crypts of Lieberkühn, closely associated with Paneth cells.
• Skin: Stem cells present in the bulge region of hair follicles.
• Liver: Hepatic progenitor cells (oval cells) located in the Canal of Hering.

Incorrect match:
• Cornea - Ciliary body: Corneal stem cells are actually located in the limbus (limbal stem cells) at the junction of cornea and sclera, not the ciliary body.

QUESTION

A 55-year-old woman presents to her primary care physician with a lump in her right breast that she discovered during a self-examination. She reports that the lump is painless and has been present for about three months. A mammogram shows a suspicious mass. The biopsy shows the following image. What is the likely diagnosis?



A **Dystrophic calcification** — CORRECT

B Ductal degeneration

C Lipofuschin deposition

D Metastatic calcification

RIGHT ANSWER

OK **A. Dystrophic calcification**

WHY THIS IS RIGHT

Dystrophic calcification refers to calcium deposition in damaged or necrotic tissues despite normal serum calcium levels.
• Occurs in areas of tissue injury, necrosis, or degeneration.
• Common sites include atherosclerotic plaques, damaged heart valves, necrotic tumors, and breast lesions.
• Appears histologically as basophilic granular calcium deposits.
• In the breast, dystrophic calcification often produces microcalcifications on mammography, which can indicate underlying malignancy (e.g., DCIS).

QUESTION

All of the following are crosslinking fixatives except:

A Glutaraldehyde

B Formaldehyde

C **Methanol** CORRECT

D Osmium tetraoxide

RIGHT ANSWER

OK

C. Methanol

WHY THIS IS RIGHT

Crosslinking fixatives preserve tissue by forming covalent cross-links between proteins, stabilizing cellular structures and preventing degradation.

Examples of crosslinking fixatives:
• Formaldehyde
• Glutaraldehyde
• Osmium tetroxide
• These fixatives maintain tissue architecture by linking amino groups of proteins.
• Methanol, however, is a coagulant (precipitating) fixative, which works by denaturing and precipitating proteins rather than crosslinking them.

QUESTION

Which is the most important light microscopic feature of irreversible cell injury?

A Amorphous densities in mitochondria

B Cell membrane blebs and loss of microvilli

C Ribosomal detachment from endoplasmic reticulum

D **Nuclear karyolysis** CORRECT

RIGHT ANSWER

OK

D. Nuclear karyolysis

WHY THIS IS RIGHT

The most reliable light microscopic indicator of irreversible cell injury is nuclear damage, particularly karyolysis.
Sequence of nuclear changes in irreversible injury:
1. Pyknosis - nuclear shrinkage with dense chromatin.
2. Karyorrhexis - fragmentation of the nucleus.
3. Karyolysis - dissolution of the nucleus due to DNA degradation by DNases.
Changes such as cell membrane blebbing, ribosomal detachment, and mitochondrial swelling occur in reversible injury.

QUESTION

What characteristics define necrosis among the options provided?
1. Disrupted cell membrane
2. Induces inflammation
3. Swelling of the cell
4. Can be seen physiologically
5. Causes nuclear fragmentation

A 1, 2, 4, 5

B **1, 2, 3** CORRECT

C 2, 3, 4, 5

D 1, 3, 4

OK RIGHT ANSWER

B. 1, 2, 3

WHY THIS IS RIGHT

Necrosis is a form of pathologic cell death caused by severe injury such as ischemia, toxins, or infection. It is characterized by loss of membrane integrity, cell swelling, and leakage of intracellular contents, which triggers inflammation.

Key features of necrosis:
• Disrupted cell membrane
• Cell swelling (oncosis)
• Induction of inflammation

Important distinction:
• Necrosis: pathologic, inflammatory.
• Apoptosis: programmed, physiologic or pathologic, cell shrinkage, no inflammation.

QUESTION

Which of the following correctly describes the changes in intracellular sodium (Na□), calcium (Ca²□), and pH during cell injury?

A **Increase, increase, decrease** CORRECT

B Increase, decrease, increase

C Decrease, increase, increase

D All increase

OK RIGHT ANSWER

A. Increase, increase, decrease

WHY THIS IS RIGHT

During reversible cell injury (especially ischemia), ATP depletion leads to failure of membrane ion pumps.

Key intracellular changes:
• Na□ increases -> due to failure of Na□/K□-ATPase -> water influx -> cell swelling.
• Ca²□ increases -> due to membrane damage and impaired Ca²□ pumps, activating destructive enzymes (proteases, phospholipases).
• pH decreases -> due to anaerobic glycolysis -> lactic acid accumulation.

Therefore: Na□ ↑, Ca²□ ↑, pH ↓ (Option A).

QUESTION

A patient with acute viral infection shows extensive cell death associated with cell swelling, plasma membrane rupture, and release of intracellular contents, leading to marked inflammation. Caspase-8 activity is found to be inhibited in the affected tissues. Which of the following molecular pathways most likely mediates this form of cell death?

A Cytochrome-c-Apaf-1-caspase-9 pathway

B Fas-FADD-caspase-8-dependent apoptosis

C **RIPK1-RIPK3-MLKL signaling pathway** CORRECT

D p53-mediated intrinsic apoptosis

OK RIGHT ANSWER

C. RIPK1-RIPK3-MLKL signaling pathway

WHY THIS IS RIGHT

Necroptosis is a form of programmed necrotic cell death characterized by cell swelling, plasma membrane rupture, and release of intracellular contents, which leads to marked inflammation.

It typically occurs when caspase-8 is inhibited, preventing normal apoptosis. In this situation, signaling shifts to the RIPK1-RIPK3-MLKL pathway, which causes membrane disruption and necrotic cell death.

Why not other options:
A. Cytochrome-c-Apaf-1-caspase-9: Intrinsic apoptosis -> non-inflammatory.
B. Fas-FADD-caspase-8-dependent apoptosis: Extrinsic apoptosis, requires caspase-8 (here it is inhibited).
D. p53-mediated intrinsic apoptosis: Also intrinsic apoptosis, non-inflammatory.

QUESTION

Which of the following proteins functions as a GTP-binding molecular switch in intracellular signaling?

A Protein kinase C

B Adenylate cyclase

C **Ras** CORRECT

D Phospholipase C

OK RIGHT ANSWER

C. Ras

WHY THIS IS RIGHT

Ras is a small GTP-binding protein (small GTPase) that functions as a molecular switch in intracellular signaling pathways controlling cell growth, proliferation, and differentiation.

Mechanism:
• Inactive state: Ras bound to GDP
• Active state: Ras bound to GTP
• Activation occurs after growth factor receptor (RTK) stimulation.
• Active Ras triggers downstream pathways such as the MAPK signaling cascade, leading to gene transcription and cell proliferation.

QUESTION

A 55-year-old male with a history of hypertension and smoking presents with chest pain and shortness of breath. ECG shows ST-segment elevation in inferior leads. He undergoes PCI but, within hours, develops deteriorating cardiac function. What is the most likely primary mechanism of cardiac deterioration in this patient?

A Oxidative stress

B **Intracellular calcium overload** CORRECT

C Activation of coagulation cascade

D Activation of the complement system

OK RIGHT ANSWER

B. Intracellular calcium overload

WHY THIS IS RIGHT

During myocardial ischemia followed by reperfusion (e.g., after PCI in STEMI), the most critical mechanism of early myocardial cell injury is intracellular calcium overload.

Pathophysiology:
1. Ischemia -> ATP depletion -> failure of Na□/K□ ATPase.
2. Na□ accumulates intracellularly, which activates the Na□/Ca²□ exchanger.
3. This causes excess Ca²□ influx into cardiomyocytes.
4. Calcium overload leads to:
• Activation of proteases, phospholipases, and endonucleases
• Mitochondrial damage
• Hypercontracture of myofibrils
• Cell death

QUESTION

Which of the following cell types is classified as stable cells in terms of their proliferative capacity and turnover rate?

A Neurons in the brain

B Hematopoietic cells in bone marrow

C Stratified squamous epithelia of the skin

D **Endothelial cells in blood vessels** CORRECT

OK RIGHT ANSWER

D. Endothelial cells in blood vessels

WHY THIS IS RIGHT

Labile Cells
• These cells are continuously being lost and replaced by maturation from tissue stem cells and by the proliferation of mature cells.
• Labile cells include hematopoietic cells in the bone marrow and the majority of surface epithelia.

Stable Cells
• They are quiescent and have minimal proliferative activity from progenitor cells.
• They divide only when required but have limited regeneration compared to labile cells.
• Stable cells constitute the parenchyma of most solid tissues (e.g., liver), endothelial cells, fibroblasts, and smooth muscle cells.

Permanent Cells
• These cells are considered to be terminally differentiated and non-proliferative in postnatal life.
• Permanent cells include neurons and cardiac and skeletal muscle cells.

QUESTION

A 52-year-old woman is diagnosed with an invasive ductal carcinoma of the breast. Molecular analysis of the tumor reveals loss of a specific cell-death pathway that normally occurs when epithelial cells lose contact with the extracellular matrix. Loss of which of the following forms of programmed cell death most directly facilitates metastasis in this patient?

A Necroptosis

B Pyroptosis

C **Anoikis** CORRECT

D Ferroptosis

OK RIGHT ANSWER

C. Anoikis

WHY THIS IS RIGHT

Anoikis is a specialized form of apoptosis that occurs when epithelial or anchorage-dependent cells lose attachment to the extracellular matrix (ECM). This mechanism prevents detached cells from surviving and colonizing other tissues.

In malignant tumors, especially carcinomas, cancer cells often develop resistance to anoikis, allowing them to survive after detaching from the primary tumor, circulate in blood or lymph, and establish metastases at distant sites.

Other options:

A. Necroptosis: programmed inflammatory necrosis mediated by RIPK1/RIPK3.

B. Pyroptosis: inflammatory cell death mediated by caspase-1 with IL-1β release.

D. Ferroptosis: iron-dependent cell death due to lipid peroxidation.

QUESTION

Arrange the steps of the extrinsic pathway of apoptosis in the correct order.
i. Binding of Fas ligand of T cells to Fas
ii. Trimerization of Fas
iii. Activation of caspase 8, 10.
iv. Formation of FADD

A i > ii > iii > iv

B iii > i > ii > iv

C **i > ii > iv > iii** CORRECT

D iv > i > ii > iii

OK RIGHT ANSWER

C. i > ii > iv > iii

WHY THIS IS RIGHT

The extrinsic pathway of apoptosis (death receptor pathway) is triggered when death ligands bind to death receptors on the cell surface, leading to activation of initiator caspases (caspase-8 and caspase-10).

Correct sequence:

i. Binding of Fas ligand (FasL) on T cells to Fas (CD95) receptor on the target cell.
ii. Trimerization of Fas receptors after ligand binding.
iii. Recruitment of adaptor protein FADD (Fas-associated death domain) forming the death-inducing signaling complex (DISC).
iv. Activation of initiator caspases (caspase-8 and caspase-10) -> activation of executioner caspases (3, 6, 7) -> apoptosis.

Thus, the correct order: i -> ii -> iv -> iii

QUESTION

Which of the following statements about apoptosis is false?

A Characterized by the activation of caspases leading to DNA fragmentation.

B **Inflammatory response and release of damage-associated molecular patterns (DAMPs).** CORRECT

C Chromatin condensation and formation of cytoplasmic blebs.

D Eliminates cells that are injured beyond repair without eliciting a host reaction.

RIGHT ANSWER

OK **B. Inflammatory response and release of damage-associated molecular patterns (DAMPs).**

WHY THIS IS RIGHT

Apoptosis is a form of programmed cell death characterized by activation of caspases, DNA fragmentation, chromatin condensation, and membrane blebbing with formation of apoptotic bodies that are rapidly phagocytosed. Because the cell contents remain membrane-bound and are cleared efficiently, apoptosis does not trigger an inflammatory response. In contrast, necrosis leads to cell membrane rupture, release of intracellular components (including DAMPs), and inflammation.
• Apoptosis: caspase activation, cell shrinkage, chromatin condensation, membrane blebbing, no inflammation.
• Necrosis: cell swelling, membrane rupture, DAMP release -> inflammation.

So, the false statement is B, because inflammatory response with DAMP release is a feature of necrosis, not apoptosis.

QUESTION

A patient is scheduled for coagulation testing, including prothrombin time (PT) and activated partial thromboplastin time (APTT). Which one of the following vacutainers is the most appropriate choice for this purpose?

A Lavender-colored tube

B **Blue-colored tube** CORRECT

C Red-colored tube

D Gray-colored tube

RIGHT ANSWER

OK

B. Blue-colored tube

WHY THIS IS RIGHT

Vial Color & Additive	Uses
Blue (Trisodium citrate)	• Coagulation studies (PT, aPTT, D-dimer) • Platelet function studies
Red (Plain vial / Clot activator)	• Serum samples • Biochemistry, serology
Yellow (SPS / ACD)	• Blood culture (SPS) • HLA typing (ACD)
Green (Heparin)	• Light green (Lithium heparin): ABG, electrolytes • Dark green (Sodium heparin): Biochemistry, osmotic fragility
Lavender / Purple (EDTA)	• CBC (Complete blood count) • Peripheral smear • HbA1c • ESR (Wintrobe method)
Grey (Sodium fluoride + Potassium oxalate)	• Glucose estimation • Lactate

QUESTION

A 30-year-old man develops fever, chills, back pain, and hemoglobinuria within 1 hour of receiving a blood transfusion. The transfusion is immediately stopped. Laboratory investigations reveal elevated serum LDH. Which of the following best explains the underlying immunopathogenesis of this reaction?

A Type 1 hypersensitivity reaction

B **Type 2 hypersensitivity reaction** CORRECT

C Type 3 hypersensitivity reaction

D Type 4 hypersensitivity reaction

OK RIGHT ANSWER

B. Type 2 hypersensitivity reaction

WHY THIS IS RIGHT

Type II Hypersensitivity- Antibodies (IgG/IgM) target cell surface antigens
Leads to:
o Complement activation
o Cell destruction

Wrong blood transfusion (ABO mismatch)
↓
Recipient antibodies (IgM)
↓
Bind donor RBC antigens
↓
Complement activation
↓
Intravascular hemolysis
↓
Hemoglobinuria + ↑ LDH
The presentation is consistent with an acute hemolytic transfusion reaction.
Preformed recipient IgM or IgG antibodies react against donor RBC antigens.
This causes complement-mediated intravascular hemolysis with hemoglobinuria and raised LDH.
Such antibody-mediated cytotoxic reactions are classified as type II hypersensitivity.

QUESTION

A patient with renal disease undergoes a biopsy. On Congo red staining, the deposits show apple-green birefringence under polarized light. What is the most likely diagnosis?

A Minimal change disease

B Diabetic nephropathy

C **Amyloidosis** CORRECT

D Sarcoidosis

RIGHT ANSWER

OK

C. Amyloidosis

WHY THIS IS RIGHT

Apple-green birefringence on Congo red staining under polarized light is pathognomonic for amyloid deposition. Amyloid fibrils form β-pleated sheets that bind Congo red in a characteristic way. Renal amyloidosis causes proteinuria and progressive renal dysfunction.

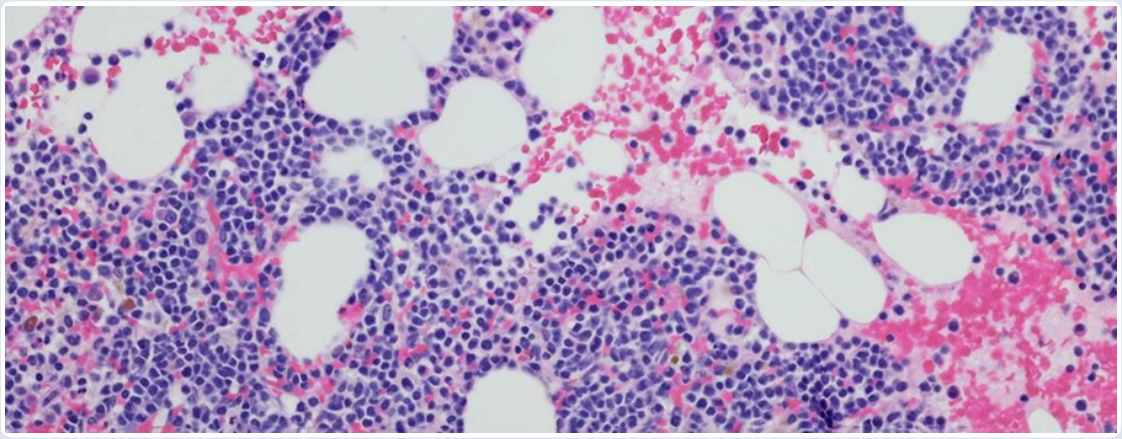
A. Minimal change disease
Seen on:
Electron microscopy -> foot process effacement
• No Congo red positivity

B. Diabetic nephropathy
Shows:
• Kimmelstiel-Wilson nodules

D. Sarcoidosis
Shows:
• Non-caseating granulomas

QUESTION

A 53-year-old woman presents with complaints of fatigue, jaundice, edema, and hepatosplenomegaly. She had anemia and neutropenia. Her serum creatinine is 3.2 mg/dL, and her bone marrow examination showed 22% plasma cells, and her serum Ig levels were 3.0 g/dL. The bone marrow biopsy is shown below. What is the associated histology?



A Monoclonal gammopathy of undetermined significance

B Bone marrow - fibrinoid necrosis

C **Amyloidosis** CORRECT

D Myxomatous degeneration of the bone marrow

RIGHT ANSWER

OK

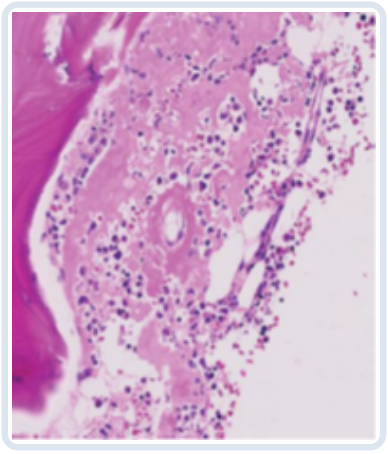
C. Amyloidosis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A patient presents with hepatosplenomegaly with bone marrow having 22% plasma cells. The bone marrow biopsy had a pink acellular deposition of material as shown. What is the likely diagnosis?



A Bone marrow necrosis

B T.B.

C Myxoid degeneration

D **Amyloidosis** CORRECT

RIGHT ANSWER

OK

D. Amyloidosis

WHY THIS IS RIGHT

Plasma cell proliferation
↓
Excess light chains
↓
Misfolding -> β-pleated sheets
↓
Extracellular deposition
↓
Organ dysfunction (liver, spleen, marrow)

The pink, amorphous, acellular extracellular deposition in bone marrow is characteristic of amyloid.

Amyloidosis is commonly associated with plasma cell dyscrasias due to AL (light chain) protein deposition.
Hepatosplenomegaly and increased plasma cells support an underlying plasma cell disorder.
Bone marrow necrosis shows ghost cells, TB shows granulomas, and myxoid degeneration has basophilic stroma.

QUESTION

Match the mode of inheritance with the disorder in the appropriate column:

1. Autosomal dominant	A. Duchene Muscular dystrophy
2. X Linked Recessive	B. Lebers disease
3. Autosomal Recessive	C. Cystic fibrosis
4. Mitochondrial	D. Myotonia dystrophica

A 1-A, 2-D, 3-C, 4-B

B **1-D, 2-A, 3-C, 4-B** CORRECT

C 1-C, 2-A, 3-D, 4-B

D 1-A, 2-D, 3-B, 4-C

OK RIGHT ANSWER

B. 1-D, 2-A, 3-C, 4-B

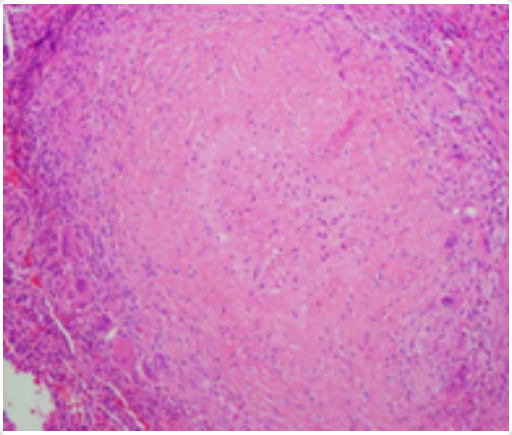
WHY THIS IS RIGHT

Myotonic dystrophy shows autosomal dominant inheritance due to trinucleotide repeat expansion, CTG trinucleotide repeat expansion.
Duchenne muscular dystrophy is X-linked recessive, caused by dystrophin gene deletion.
Cystic fibrosis is an autosomal recessive disorder due to CFTR mutation.
Leber's hereditary optic neuropathy follows mitochondrial inheritance via maternal transmission.

Pattern	Key Clue	Example
AD	Every generation	Myotonic dystrophy
XLR	Males affected	Duchenne
AR	Skips generations	CF
Mitochondrial	Maternal only	LHON

QUESTION

A patient presents with fever, cough, weight loss, histology of the pathology is as shown below. What is the most likely cytokine involved?



A **Interferon gamma** — CORRECT

B Complement 5a protein

C Histamine

D Prostaglandin

RIGHT ANSWER

OK **A. Interferon gamma**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A physician wanted to perform D-Dimer using the vacutainer shown in the image. Which of the following is the anticoagulant used for this test?

A **Trisodium Citrate** CORRECT

B Potassium oxalate

C Sodium fluoride

D EDTA

RIGHT ANSWER

OK **A. Trisodium Citrate**

WHY THIS IS RIGHT

The vacutainer shown has a light blue cap, which is specifically used for coagulation studies such as D-dimer.

Key fact:
• Light blue top tube -> contains Trisodium citrate (3.2% or 3.8%)
• It works by chelating calcium reversibly, preserving coagulation factors for testing.

Option analysis:
• A. Trisodium citrate -> □ Correct (used for PT, aPTT, D-dimer)
• B. Potassium oxalate -> Used with gray top (glucose estimation)
• C. Sodium fluoride -> Gray top (inhibits glycolysis for glucose)
• D. EDTA -> Purple top (CBC, hematology)

QUESTION

Match the following:

Mode of Inheritance	Disease
A. AD	1. LHON
B. AR	2. Myotonic Dystrophy
C. XR	3. Cystic Fibrosis
D. Mitochondrial	4. Duchenne Muscular Dystrophy

A. A-1, B-2, C-3, D-4

B. A-4, B-3, C-1, D-2

C. **A-2, B-3, C-4, D-1** CORRECT

D. A-2, B-3, C-1, D-4

OK RIGHT ANSWER

C. A-2, B-3, C-4, D-1

WHY THIS IS RIGHT

Myotonic dystrophy follows autosomal dominant inheritance due to trinucleotide repeat expansion.
Cystic fibrosis is an autosomal recessive disorder caused by CFTR gene mutation.
Duchenne muscular dystrophy is inherited in an X-linked recessive pattern due to dystrophin gene deletion.
Leber's hereditary optic neuropathy shows mitochondrial inheritance via maternal transmission.

Pattern	Key Feature	Example
AD	Every generation	Myotonic dystrophy
AR	Skips generations	CF
XLR	Males affected	Duchenne
Mitochondrial	Maternal only	LHON

QUESTION

Which vacutainer is commonly used to collect blood samples for serum electrolyte analysis?

A Blue

B Yellow

C **Green** CORRECT

D Purple

RIGHT ANSWER

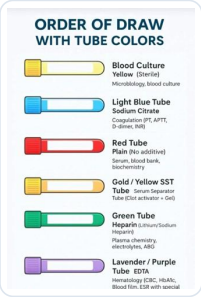
OK

C. Green

WHY THIS IS RIGHT

Need electrolyte measurement
↓
Avoid clotting + avoid ion interference
↓
Use heparin (green tube)
↓
Accurate electrolyte values

Serum electrolyte analysis commonly uses a green-top vacutainer containing heparin as anticoagulant.
Heparin prevents clotting without chelating ions, thus preserving true electrolyte levels.
EDTA (purple) chelates calcium and alters potassium, while citrate (blue) dilutes the sample.
Yellow-top tubes are used for serum separation, but may also be used for routine electrolyte estimation making the answer controversial.



QUESTION

For HbA1c sample, the vacutainer used contains which of the following?

A Warfarin

B Sodium fluoride

C **EDTA** CORRECT

D Heparin

RIGHT ANSWER

OK

C. EDTA

WHY THIS IS RIGHT

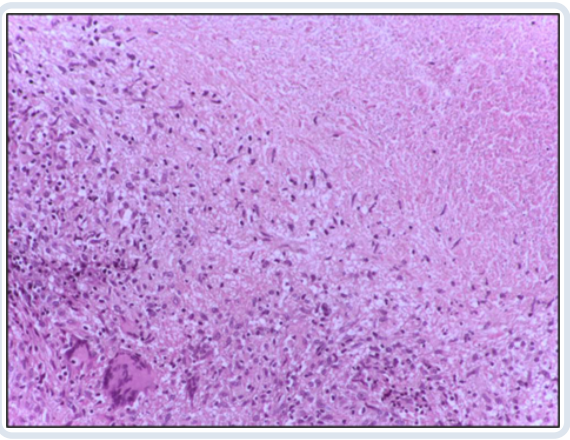
Need to analyze RBCs
↓
Prevent clotting without damaging cells
↓
Use EDTA
↓
Accurate HbA1c measurement

Vacutainer Vials and Their Uses

Vials	Uses
Blue (Trisodium citrate)	Coagulation studies (PT, aPTT studies), Platelet Study (Platelet-rich plasma)
Red (Plain vial)	Serum samples
Yellow	Bacterial culture, HLA typing
Green (Emergency Testing) (Light – Li heparin Dark – Na heparin)	ABG, Electrolytes, Osmotic fragility
Lavender (EDTA)	CBC, Immunohistochemistry studies, ESR - Westergren method, HbA1C
Grey (Additive: NaF) (Anticoagulant: potassium oxalate)	Glucose estimation

QUESTION

Histological examination of a cervical lymph node biopsy is shown below. What is the likely diagnosis?



A Sarcoidosis

B **Tuberculosis** CORRECT

C Langerhans cell histiocytosis

D Hodgkin lymphoma

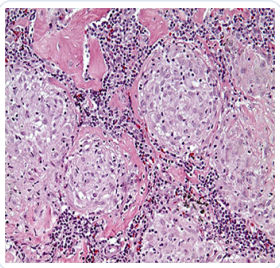
OK RIGHT ANSWER

B. Tuberculosis

WHY THIS IS RIGHT

Mycobacterium tuberculosis
↓
Macrophage ingestion
↓
T-cell activation (Th1)
↓
IFN-γ release
↓
Granuloma formation
↓
Caseous necrosis
Tuberculosis shows caseating granulomatous inflammation with central amorphous necrosis. Granulomas are composed of epithelioid histiocytes and Langhans-type giant cells.
Cervical lymph node involvement is common in tuberculous lymphadenitis.

Sarcoidosis shows non-caseating granulomas as shown below.
Langerhans cell histiocytosis has grooved nuclei, and Hodgkin lymphoma shows Reed-Sternberg cells.



QUESTION

Which of the following is/are criteria for anaphylaxis?
a. Persistent abdominal cramps and vomiting
b. Hypertension
c. Urticaria and swollen lips
d. Respiratory distress

A a, b & c are correct

B b & c are correct

C **a, c & d** CORRECT

D All of the above

OK RIGHT ANSWER

C. a, c & d

WHY THIS IS RIGHT

Allergen exposure
↓
IgE on mast cells cross-links
↓
Mast cell degranulation
↓
Histamine, leukotrienes release
↓
Vasodilation + bronchospasm + edema
↓
Anaphylaxis
Anaphylaxis is an acute, systemic hypersensitivity reaction involving skin, mucosa, respiratory, and gastrointestinal systems.
Diagnostic criteria include urticaria with angioedema, respiratory compromise, and persistent gastrointestinal symptoms like cramps and vomiting.

System	Features
Skin	Urticaria, angioedema
Respiratory	Dyspnea, wheeze, stridor
Cardiovascular	Hypotension
GI	Vomiting, cramps

Hypotension, not hypertension, is the cardiovascular manifestation of anaphylaxis.
Hence, options a, c, and d fulfil criteria for anaphylaxis.

QUESTION

Which of the following should be prevented when collecting a blood sample for serum calcium?

A **Making a fist with forearm exercise** CORRECT

B Tourniquet should be tied and released as soon as possible

C Should be collected in sitting position & better to avoid prolonged immobilization

D Sample should be analyzed within 15-30 min of collection

RIGHT ANSWER

OK **A. Making a fist with forearm exercise**

WHY THIS IS RIGHT

Repeated fist clenching = forearm muscle activity. This causes:
• Local metabolic changes
• Hemoconcentration
• Altered ion balance

Result: Falsely elevated calcium (and other analytes like potassium)
Fist clenching / forearm exercise
↓
Muscle contraction
↓
↑ Local metabolites + hemoconcentration
↓
Altered serum composition
↓
False lab values (↑ Ca, ↑ K)

Factor	Effect
Fist clenching	False ↑ calcium
Prolonged tourniquet	↑ protein-bound calcium
Posture change	Alters calcium levels
Delayed processing	pH changes -> affects Ca binding

QUESTION

Match the following pathological bodies with their associated condition.

Column A	Column B
A. Parkinson's Disease	1. Dutcher body
B. Rabies	2. Negri body
C. Multiple myeloma	3. Lewy body
D. Malakoplakia	4. Michaelis-Gutmann body

A. A-4, B-3, C-1, D-2

B. A-3, B-2, C-4, D-1

C. A-2, B-1, C-3, D-4

D. **A-3, B-2, C-1, D-4** CORRECT

OK

RIGHT ANSWER

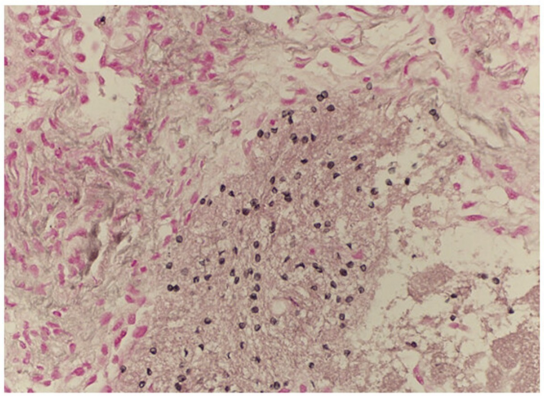
D. A-3, B-2, C-1, D-4

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A post-renal transplant patient presents with fever and breathlessness. Lung biopsy is done. Which of the following stains is least useful in identifying the causative organism?



A **Congo red** — CORRECT

B Periodic Acid-Schiff

C Gomori Methenamine Silver

D Fontana-Masson

RIGHT ANSWER

OK

A. Congo red

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Which of the following are correctly matched with respective stain and its properties?
1. Perl's - Iron, red
2. Alizarin - Calcium, black
3. Giemsa - Mast cells, purple
4. Von Gieson - Elastin fibres, blue

A 1 and 2

B 2 and 4

C **3 only** CORRECT

D All of the above

RIGHT ANSWER

OK **C. 3 only**

WHY THIS IS RIGHT

The source quiz contains the placeholder "$1d" here and does not include a written explanation for this item.

QUESTION

All streptococcal antigen-host antigen-disease associations are true, except:

A M protein - Myocardium - ARF

B Cytoplasmic membrane - Glomerular intima - PSGN

C Peptidoglycan - Skin antigens - Guttate psoriasis

D **Hyaluronic acid - Skin - Impetigo** CORRECT

RIGHT ANSWER

OK

D. Hyaluronic acid - Skin - Impetigo

WHY THIS IS RIGHT

Certain streptococcal antigens cross-react with host tissues, causing immune-mediated diseases due to molecular mimicry.

Important associations:
• M protein -> Myocardium -> Acute rheumatic fever (ARF) due to cross-reactive antibodies.
• Streptococcal cytoplasmic membrane antigens -> Glomeruli -> Post-streptococcal glomerulonephritis (PSGN).
• Peptidoglycan -> Skin antigens -> Guttate psoriasis following streptococcal infection.

Hyaluronic acid capsule of streptococci mainly acts as a virulence factor that prevents phagocytosis and does not cause molecular mimicry leading to impetigo.

QUESTION

All are features of venous thrombus, except:

A **Caused by turbulent blood flow** CORRECT

B Common in deep leg veins

C Propagate in antegrade manner

D Composed mainly of red cells (red thrombi)

RIGHT ANSWER

OK **A. Caused by turbulent blood flow**

WHY THIS IS RIGHT

Venous thrombi (red thrombi) typically form due to blood stasis and hypercoagulability, which are key components of Virchow's triad.

Features of venous thrombi:
• Commonly occur in deep veins of the legs (DVT).
• Propagate in the direction of blood flow (antegrade) toward the heart.
• Rich in red blood cells and fibrin, giving them a red appearance.

Important distinction:
• Turbulent blood flow is mainly associated with arterial thrombus formation, not venous thrombi.

QUESTION

What type of collagen is involved in the classic Ehlers-Danlos syndrome?

A Type 1

B Type 2

C **Type 5** CORRECT

D Type 4

RIGHT ANSWER

OK

C. Type 5

WHY THIS IS RIGHT

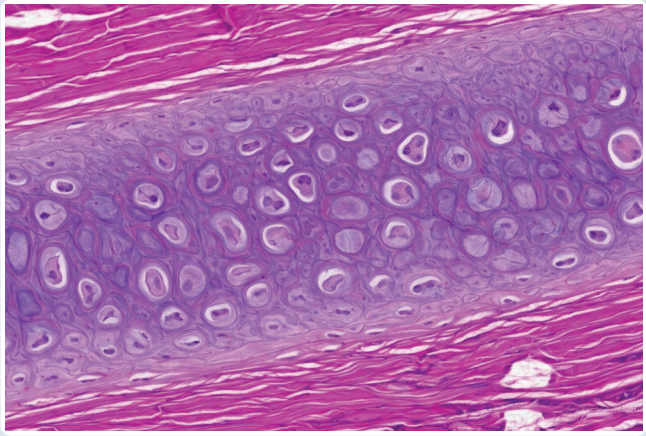
Classic Ehlers-Danlos syndrome (EDS) is caused by mutations in type V collagen genes (COL5A1, COL5A2), leading to defective collagen fibril formation.

Clinical features:
• Hyperextensible skin
• Joint hypermobility
• Easy bruising
• Poor wound healing with atrophic (“cigarette paper”) scars

Other options:
• Type 3 collagen -> Vascular EDS
A. Type 1 collagen -> Osteogenesis imperfecta
B. Type 2 collagen-> Cartilage disorders

QUESTION

Which of the following stains are used to visualize the specific fibers present in the following cartilage?



A Bielchowsky stain

B **Verhoeff's stain** CORRECT

C GMS Silver stain

D Alcian Blue stain

OK RIGHT ANSWER

B. Verhoeff's stain

WHY THIS IS RIGHT

The image shows elastic cartilage, characterized by chondrocytes in lacunae within a matrix rich in elastic fibers (e.g., external ear, epiglottis).

To visualize elastic fibers, special stains are used, the most important being Verhoeff-Van Gieson (Verhoeff's) stain, which stains elastic fibers black.

Other stains:

A. Bielschowsky stain: highlights neurofibrils/axons.

C. GMS (Grocott methenamine silver): used for fungi.

D. Alcian blue: stains acidic mucopolysaccharides and glycosaminoglycans in cartilage matrix.

QUESTION

All of the following conditions are related to the same core pathology except:

A Parkinson's disease

B **Alzheimer’s disease** CORRECT

C Lewy body dementia

D Multisystem atrophy

RIGHT ANSWER

OK **B. Alzheimer’s disease**

WHY THIS IS RIGHT

Parkinson disease, Lewy body dementia, and multisystem atrophy are synucleinopathies, meaning they share the core pathology of abnormal aggregation of α-synuclein protein within neurons or glial cells.
• Parkinson disease: Lewy bodies containing α-synuclein in substantia nigra.
• Lewy body dementia: Diffuse cortical Lewy bodies (α-synuclein).
• Multiple system atrophy: α-synuclein accumulation in oligodendrocytes.

In contrast, Alzheimer disease is a tauopathy/amyloidopathy, characterized by:
• β-amyloid plaques
• Neurofibrillary tangles composed of hyperphosphorylated tau protein

QUESTION

Which of the following is not a hemostatic agent?

A Zeolite

B Chitosan

C **Boric acid** CORRECT

D Kaolin

RIGHT ANSWER

OK

C. Boric acid

WHY THIS IS RIGHT

Hemostatic agents are substances used to promote rapid blood clotting and control bleeding, especially in trauma or surgical settings.

Common topical hemostatic agents include:
• Zeolite: absorbs water from blood -> concentrates clotting factors and platelets.
• Chitosan: promotes platelet adhesion and aggregation.
• Kaolin: activates the intrinsic coagulation pathway (factor XII activation).

Boric acid is primarily used as an antiseptic and antifungal agent, not for promoting coagulation.

QUESTION

Which cyclin-CDK (cyclin-dependent kinase) combination is essential for the G2 to M phase transition of cell cycle?

A Cyclin D-CDK6

B Cyclin A-CDK2

C Cyclin A-CDK1

D **Cyclin B-CDK1** CORRECT

OK RIGHT ANSWER

D. Cyclin B-CDK1

WHY THIS IS RIGHT

The G2 -> M phase transition of the cell cycle is regulated by the Cyclin B-CDK1 complex, also called the Maturation Promoting Factor (MPF).
Mechanism:
• During late G2 phase, Cyclin B accumulates and binds to CDK1.
• Activation of this complex triggers entry into mitosis by promoting:
• Chromosome condensation
• Nuclear envelope breakdown
• Mitotic spindle formation

Other options:
A. Cyclin D-CDK4/6 -> G1 progression~~.~~
B. Cyclin A-CDK2 -> S phase progression.
• Cyclin E-CDK2 -> G1 to S transition.

QUESTION

A diabetic nephropathy patient is an ideal candidate for renal graft transplant. Which of the following statements is true?

A The survival rate of graft is 95% in the first year

B The life expectancy is doubled in a diabetic patient with renal transplant

C **The transplantation is cost effective after the second transplant year** CORRECT

D The treatment of chronic rejection has improved over the last 10 years

OK RIGHT ANSWER

C. The transplantation is cost effective after the second transplant year

WHY THIS IS RIGHT

Renal transplantation is the preferred treatment for end-stage renal disease (ESRD), including patients with diabetic nephropathy, because it provides better quality of life and long-term survival compared with dialysis.

Although the initial cost of transplantation is high (surgery, hospitalization, immunosuppressive therapy), the overall cost becomes lower than long-term dialysis after about 1-2 years.

QUESTION

All of the following conditions are associated with Neurosecretory bodies except:

A Medullary thyroid carcinoma

B Pituitary adenoma

C Paraganglioma

D **Adrenal cortical tumor** CORRECT

RIGHT ANSWER

OK

D. Adrenal cortical tumor

WHY THIS IS RIGHT

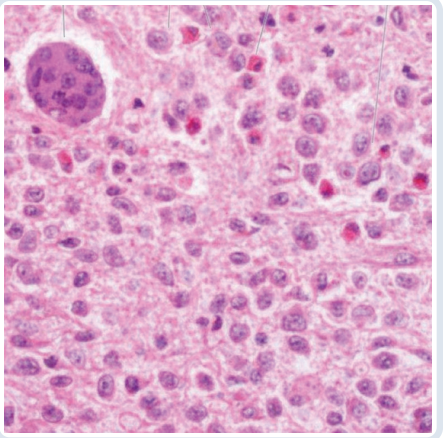
Neurosecretory (dense-core) granules are membrane-bound secretory vesicles present in neuroendocrine cells, storing peptide hormones and amines.

They are typically seen in neuroendocrine tumors, such as:
• Medullary thyroid carcinoma (from parafollicular C cells)
• Pituitary adenoma
• Paraganglioma / pheochromocytoma

Adrenal cortical tumors arise from steroid-producing cortical cells, which synthesize hormones from cholesterol in smooth ER and mitochondria, not from stored secretory granules.

QUESTION

A 7-year-old child brought to OPD with complaints of fever, erythematous rash, and bone pain. X-ray of the skull revealed multiple lytic lesions. Skin biopsy shows prominent nuclear grooves with eosinophils as in the image below. Immunohistochemistry is positive for S100, CD1a. What is the likely diagnosis?



A Rosai-Dorfman disease

B Kimura disease

C **Langerhans cell histiocytosis** CORRECT

D Juvenile Xanthogranuloma

RIGHT ANSWER

OK **C. Langerhans cell histiocytosis**

WHY THIS IS RIGHT

Langerhans cell histiocytosis (LCH) is a disorder characterized by clonal proliferation of Langerhans cells (dendritic antigen-presenting cells).
• Children commonly affected.
• Lytic bone lesions, especially in the skull.
• Histology: Langerhans cells with grooved “coffee-bean” nuclei and numerous eosinophils.
• Immunohistochemistry: S100 positive and CD1a positive.
• Electron microscopy: Birbeck granules (“tennis-racket” shaped).

Why not the other options:
A. Rosai-Dorfman disease: Shows massive painless lymphadenopathy with emperipolesis (intact lymphocytes inside histiocytes). Cells are S100 positive but CD1a negative, unlike this case.
B. Kimura disease: Chronic inflammatory disorder with subcutaneous head-neck masses, lymphadenopathy, and marked eosinophilia, not lytic skull lesions.
D. Juvenile xanthogranuloma: Shows foamy histiocytes and Touton giant cells; CD1a and S100 negative.

QUESTION

A genetic polymorphism leading to deletion of the ATG16L1 gene, which plays a key role in autophagosome formation, predisposes an individual to which of the following diseases?

A Alzheimer disease

B **Inflammatory bowel disease** CORRECT

C Systemic lupus erythematosus

D Rheumatoid arthritis

RIGHT ANSWER

OK **B. Inflammatory bowel disease**

WHY THIS IS RIGHT

ATG16L1 is a key gene involved in autophagy, the cellular process that degrades damaged organelles and intracellular pathogens through autophagosome formation.

Polymorphisms or deletion of ATG16L1 impair autophagy, leading to defective handling of intestinal microbes and abnormal immune responses in the gut. This contributes to chronic intestinal inflammation, particularly Crohn disease, a type of inflammatory bowel disease (IBD).

QUESTION

A 58-year-old man with a 10-year history of rheumatoid arthritis presents with progressive pedal edema, frothy urine, and hepatosplenomegaly. Urinalysis shows nephrotic-range proteinuria. Renal biopsy reveals amorphous eosinophilic deposits in the mesangium that show apple-green birefringence under polarized light after Congo red staining. Which of the following findings is most specific for identifying the type of amyloid protein in this patient?

A Elevated serum β□-microglobulin

B **Immunohistochemical staining for serum amyloid A protein** CORRECT

C Presence of Bence Jones proteins in urine

D Increased transthyretin levels

RIGHT ANSWER

OK

B. Immunohistochemical staining for serum amyloid A protein

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

A child presents with intellectual disability, chronic diarrhea, ichthyosis, peripheral neuropathy, and low serum copper. The most likely diagnosis is:

A Wilson disease

B Menkes disease

C **MEDNIK syndrome** CORRECT

D Zellweger syndrome

RIGHT ANSWER

OK

C. MEDNIK syndrome

WHY THIS IS RIGHT

MEDNIK syndrome is a rare autosomal recessive disorder of copper metabolism caused by mutations in the AP1S1 gene, which affects intracellular trafficking of copper transport proteins.

MEDNIK stands for the major clinical features:
• Mental retardation (intellectual disability)
• Enteropathy (chronic diarrhea)
• Deafness
• Neuropathy (peripheral neuropathy)
• Ichthyosis
• Keratoderma

Patients typically have abnormal copper metabolism with low serum copper and ceruloplasmin, leading to neurologic and dermatologic manifestations.

Why not the other options:
A. Wilson disease: Copper accumulation -> liver disease, neuropsychiatric symptoms, Kayser-Fleischer rings; not ichthyosis or chronic diarrhea.
B. Menkes disease: Infancy onset, kinky hair, severe neurodegeneration.
D. Zellweger syndrome: Peroxisomal disorder with hypotonia, seizures, craniofacial anomalies.

QUESTION

A 42-year-old man presents with chronic abdominal pain, intermittent fever, and loose stools. Colonoscopy reveals patchy transmural inflammation with a cobblestone appearance of the intestinal mucosa. Which of the following biochemical parameters is most likely to be elevated in this patient?

A Serum albumin

B Retinol-binding protein

C Transthyretin (prealbumin)

D **Fibrinogen** CORRECT

OK RIGHT ANSWER

D. Fibrinogen

WHY THIS IS RIGHT

The patient's findings (chronic abdominal pain, intermittent fever, loose stools, patchy transmural inflammation, cobblestone mucosa) are characteristic of Crohn disease, a chronic inflammatory bowel disease. During chronic inflammation, the liver increases production of acute-phase reactants under the influence of cytokines such as IL-6, IL-1, and TNF-α.

Positive acute-phase proteins (increase in inflammation):
• Fibrinogen
• C-reactive protein (CRP)
• Serum amyloid A
• Ferritin
• Haptoglobin

Negative acute-phase proteins (decrease in inflammation):
• Albumin
• Prealbumin (transthyretin)
• Retinol-binding protein

QUESTION

A 36-year-old male presents to the clinic with complaints of bilateral cataracts, short stature, premature greying and loss of hair, and skin changes. He also reports joint stiffness and pain, as well as type 2 diabetes mellitus. On examination, the patient appears older than his stated age, with characteristic "bird-like" facies and a high-pitched voice. Which of the following conditions is most likely to be associated with his presentation?

A Hutchinson-Gilford Syndrome

B **Werner syndrome** CORRECT

C Wiedemann-Rautenstrauch syndrome

D Cockayne Syndrome

OK RIGHT ANSWER

B. Werner syndrome

WHY THIS IS RIGHT

Werner syndrome is an autosomal recessive premature aging (progeroid) disorder caused by mutations in the WRN gene, which encodes a DNA helicase involved in DNA repair and replication. Defective DNA repair leads to genomic instability and accelerated aging.
• Premature graying and loss of hair
• Bilateral cataracts
• Short stature
• Bird-like facies
• High-pitched voice
• Skin atrophy/ulceration
• Joint stiffness
• Early-onset type 2 diabetes mellitus
• Increased risk of malignancies (especially sarcomas)

Why not the other options:

A. Hutchinson-Gilford progeria syndrome: Presents in early childhood with growth failure and premature atherosclerosis; cataracts and diabetes are uncommon.

C. Wiedemann-Rautenstrauch syndrome: Neonatal progeroid syndrome; features are present at birth.

D. Cockayne syndrome: Characterized by photosensitivity, neurologic deficits, microcephaly, and developmental delay, not diabetes or cataracts.

QUESTION

A 4-year-old patient presented with a rectal mass 2.5 cm by 3 cm. On biopsy it had the presence of colonic mucosa with multiple dilated glands filled with mucin and inflammatory cells. Which of the following is a likely diagnosis?

A Choristoma

B **Hamartoma** CORRECT

C Adenoma

D Adenocarcinoma

RIGHT ANSWER

OK

B. Hamartoma

WHY THIS IS RIGHT

Hamartoma is a benign, disorganized overgrowth of tissue native to the organ.
In the colon, it shows normal colonic mucosa with cystically dilated glands filled with mucin and inflammatory cells.
These lesions commonly present as rectal polyps in children.

Feature	Hamartoma	Choristoma	Adenoma	Adenocarcinoma
Tissue type	Native	Ectopic	Neoplastic	Malignant
Organization	Disorganized	Normal	Dysplastic	Atypical
Age	Children	Any	Adults	Adults
Malignancy	□ No	□ No	Premalignant	Yes

QUESTION

Match the following cancers with their respective genes involved:

1. KIT	A. Myeloproliferative proliferative neoplasm
2. K-RAS	B. Adenocarcinoma lung
3. JAK 2	C. Pancreatic Carcinoma
4. ALK	D. GIST

A 1-C, 2-D, 3-A, 4-B

B 1-B, 2-A, 3-D, 4-C

C **1-D, 2-C, 3-A, 4-B** CORRECT

D 1-C, 2-A, 3-D, 4-B

OK RIGHT ANSWER

C. 1-D, 2-C, 3-A, 4-B

WHY THIS IS RIGHT

• KIT mutations are characteristic of gastrointestinal stromal tumors due to constitutive tyrosine kinase activation.

K-RAS mutations are commonly seen in pancreatic adenocarcinoma driving uncontrolled proliferation.
JAK2 mutations are associated with myeloproliferative neoplasms via persistent JAK-STAT signalling. Seen in:
• Polycythemia vera
• Essential thrombocythemia
• Primary myelofibrosis

ALK rearrangements are classically linked to lung adenocarcinoma. EML4-ALK fusion.

QUESTION

Match the following:

Column A	Column B
A. AFP	1. Medullary Carcinoma of Thyroid
B. Beta-HCG	2. Hepatocellular carcinoma
C. Catecholamines	3. Choriocarcinoma
D. Calcitonin	4. Pheochromocytoma

A A-3, B-1, C-2, D-4

B A-4, B-2, C-1, D-3

C A-1, B-4, C-3, D-2

D **D. A-2, B- 3, C-4, D-1** CORRECT

OK RIGHT ANSWER

D. D. A-2, B- 3, C-4, D-1

WHY THIS IS RIGHT

AFP is a tumor marker for hepatocellular carcinoma due to fetal liver protein re-expression. Elevated in:
• HCC , Hepatoblastoma
• Yolk sac tumors

β-hCG is characteristically elevated in choriocarcinoma from trophoblastic proliferation & in hydatidiform mole.

Catecholamines -> Pheochromocytoma
Tumor of: Adrenal medulla
Secretes: Epinephrine, norepinephrine
Measured as: Metanephrines / VMA

Calcitonin is produced by parafollicular C cells and is a marker of medullary thyroid carcinoma.

Marker	Source	Tumor
AFP	Fetal liver	HCC
β-hCG	Placenta	Choriocarcinoma
Catecholamines	Adrenal medulla	Pheochromocytoma
Calcitonin	Thyroid C cells	MTC

QUESTION

Epithelial mesenchymal transcription is mediated by which transcription factors?

A Cathepsin D

B **SNAIL/TWIST** CORRECT

C MMP-9

D TTF-1

RIGHT ANSWER

OK **B. SNAIL/TWIST**

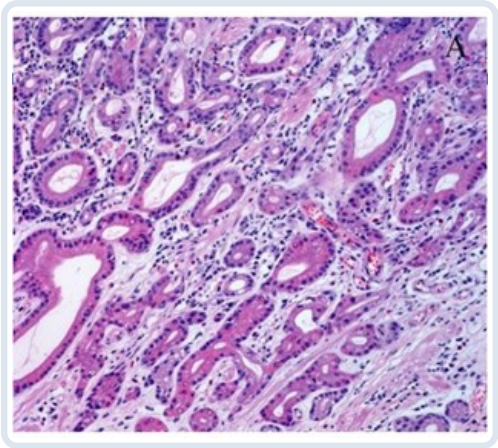
WHY THIS IS RIGHT

Epithelial cell
↓
SNAIL / TWIST activation
↓
↓ E-cadherin (cell adhesion)
↓
Loss of polarity
↓
Gain of motility
↓
Invasion -> Metastasis
Epithelial-mesenchymal transition (EMT) is driven by transcription factors that repress epithelial markers like E-cadherin.
SNAIL and TWIST are key EMT transcription factors that promote loss of cell adhesion and increased motility.
They induce mesenchymal phenotype, facilitating invasion and metastasis.

Cathepsin D and MMP-9 are proteases involved in matrix degradation, while TTF-1 is a lineage-specific transcription factor, not EMT-related.
Tumor cell
↓
EMT (SNAIL/TWIST)
↓
↓ E-cadherin
↓
Basement membrane breakdown (MMPs)
↓
Invasion -> Intravasation -> Metastasis

QUESTION

Which of the following is the most likely tumor arising from the slide shown in the image?



A Squamous cell carcinoma

B **Adenocarcinoma** CORRECT

C Lymphoma

D Sarcoma

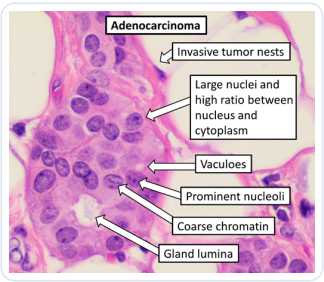
OK RIGHT ANSWER

B. Adenocarcinoma

WHY THIS IS RIGHT

Epithelial malignancy
↓
Gland formation / acinar pattern
↓
Mucin secretion
↓
Cellular atypia
↓
Adenocarcinoma
Adenocarcinoma is characterized by malignant epithelial cells forming glandular or acinar structures. Tumor cells show luminal formation, mucin production, and gland-like architecture.

This reflects origin from glandular epithelium or epithelium with secretory differentiation.
Squamous cell carcinoma shows keratinization, lymphoma shows diffuse sheets of lymphoid cells, and sarcoma is mesenchymal without gland formation.



QUESTION

All of the following are growth promoting proto-oncogenes except?

A FGF

B PDGF

C TGF alpha

D **TGF beta** CORRECT

RIGHT ANSWER

OK

D. TGF beta

WHY THIS IS RIGHT

TGF-β is growth-inhibitory, not growth-promoting.
Proto-oncogenes encode growth factors that stimulate cell proliferation and survival.
FGF, PDGF, and TGF-α act as mitogenic signals promoting cell cycle progression.
Growth factors (FGF, PDGF, TGF-α)
↓
Receptor activation
↓
RAS/MAPK pathway
↓
Cell proliferation

TGF-β, in contrast, inhibits epithelial cell proliferation by inducing cell cycle arrest via SMAD signalling. Loss of TGF-β signalling contributes to oncogenesis, but the gene itself functions as a tumor suppressor.

Factor	Function	Role in Cancer
FGF	Proliferation, angiogenesis	Oncogenic
PDGF	Cell growth	Oncogenic
TGF-α	EGFR activation	Oncogenic
TGF-β
“Beta” = brake □	Growth inhibition	Tumor suppressor

QUESTION

Which of the following malignancies has the least or no established association with obesity?

A Meningioma

B Pancreatic cancer

C **Gastric pyloric cancer** CORRECT

D Multiple myeloma

RIGHT ANSWER

OK **C. Gastric pyloric cancer**

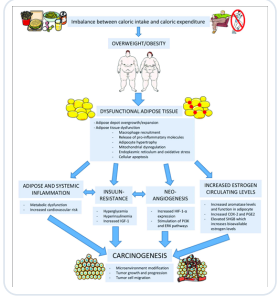
WHY THIS IS RIGHT

Obesity
↓
↑ Insulin & IGF-1
↑ Estrogen (via adipose)
↑ Inflammatory cytokines
↓
Cell proliferation + ↓ apoptosis
↓
Obesity-Associated Cancers

Strong Association	Moderate/Other Associations
Breast (postmenopausal)	Meningioma
Endometrial	Multiple myeloma
Colon	Thyroid
Pancreatic	Kidney
Esophageal adenocarcinoma	Liver

Gastric pyloric cancer is more strongly linked to *H. pylori* infection, nitrosamines, smoking, and dietary salt.
Gastric Cancer Types

Type	Location	Risk Factors
Intestinal type	Distal (pylorus)	H. pylori, diet
Diffuse type	Whole stomach	CDH1 mutation



QUESTION

Which of the following is the best gene analysis investigation for a female patient with history of breast and ovarian cancer?

A PTEN gene

B P53 gene

C **BRCA1** CORRECT

D CDH1 gene

RIGHT ANSWER

OK

C. BRCA1

WHY THIS IS RIGHT

BRCA1 and BRCA2 are tumor suppressor genes involved in homologous recombination DNA repair. Think of them as: *molecular tailors fixing torn DNA strands*
BRCA mutation
↓
Defective DNA repair
↓
Genomic instability
↓
Accumulation of mutations
↓
Cancer (breast, ovary, others)
Germline mutations markedly increase risk of breast and ovarian cancers in women. This combination of cancers is the classic clinical clue for hereditary breast-ovarian cancer syndrome.

Gene	Function	Associated Cancers
BRCA1/2	DNA repair	Breast, ovarian, prostate
PTEN	Cell cycle regulation	Breast, thyroid, endometrium
p53	DNA damage checkpoint	Sarcoma, breast, brain
CDH1	Cell adhesion	Diffuse gastric, lobular breast

QUESTION

Which of the following is the incorrect association?

A PTEN and thyroid

B BRCA2 and prostate

C STK11 and breast cancer

D **p53 and mucosal neuroma** CORRECT

RIGHT ANSWER

OK

D. p53 and mucosal neuroma

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following best describes the mechanism by which mutations in Ras/ MAP kinase gene cause oncogenicity?

A **Constitutive activation of GTP-ase domain leading to continuous cell proliferation** CORRECT

B Enhanced DNA repair leading to genomic instability

C Inactivation of tumor suppressor gene resulting in loss of cell cycle check point

D Increased activity of tyrosine kinase receptors through gene amplification

OK RIGHT ANSWER

A. Constitutive activation of GTP-ase domain leading to continuous cell proliferation

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A scientist is studying the invasion of malignant colon cancer cells into surrounding tissues in an experimental model. Over time, the cancer cells are observed to detach from each other and degrade the extracellular matrix, allowing the tumor cells to invade the basement membrane and access nearby blood vessels. Which of the following substances is most likely upregulated to facilitate basement membrane invasion?

A Acid hydrolases

B E-cadherin

C Hyaluronic acid

D **Metalloproteinases** CORRECT

OK

RIGHT ANSWER

D. Metalloproteinases

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A 58-year-old man presents with a rapidly growing bladder tumor. Genetic analysis will reveal:

A Supression of HRAS

B **Activation of HRAS** CORRECT

C Supression of KRAS

D Activation of KRAS

OK RIGHT ANSWER

B. Activation of HRAS

WHY THIS IS RIGHT

Bladder carcinoma, particularly low-grade papillary urothelial tumors, is commonly associated with activating mutations in the HRAS proto-oncogene.

Mechanism:
• Point mutation in HRAS leads to constitutive activation of the Ras signaling pathway.
• This causes continuous cell proliferation and tumor formation independent of normal growth factor signaling.

QUESTION

The mutations/changes that convert proto-oncogene to oncogene include all except:

A Promoter insertion

B **Enhancer deletion** CORRECT

C Gene amplification

D Point mutation

RIGHT ANSWER

OK

B. Enhancer deletion

WHY THIS IS RIGHT

Proto-oncogenes become oncogenes through genetic changes that increase their expression or activity, leading to uncontrolled cell proliferation.

Common activating mechanisms include:
• Point mutations -> produce constitutively active proteins (e.g., RAS).
• Gene amplification -> increased copies of the gene (e.g., HER2/ERBB2).
• Promoter/enhancer insertion -> increased transcription of the gene (often via viral insertion).

Enhancer deletion would reduce gene expression, not activate it, so it does not convert a proto-oncogene into an oncogene.

QUESTION

Which of these options is not a mechanism contributing to thalidomide's teratogenic effects?

A Release of reactive oxygen species and free radicles

B Suppression of angiogenesis

C Cereblon binding

D **Inhibition of IL2, IL4, IL5** CORRECT

OK RIGHT ANSWER

D. Inhibition of IL2, IL4, IL5

WHY THIS IS RIGHT

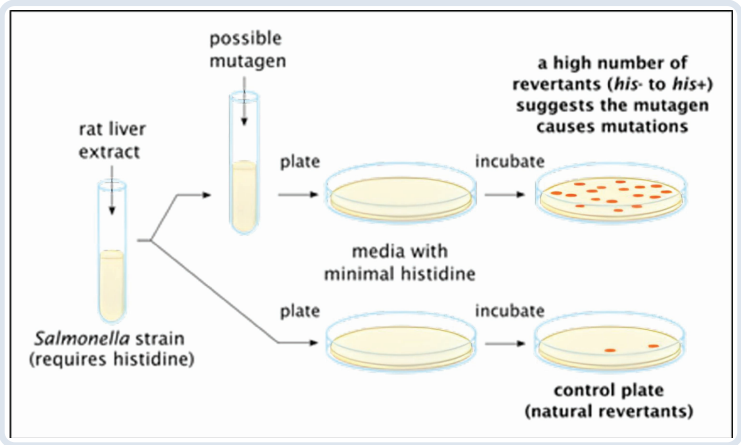
Thalidomide teratogenicity occurs through multiple mechanisms that disrupt limb bud development and embryonic vascular formation.

Key mechanisms include:
• Binding to cereblon (CRBN), a protein involved in ubiquitin ligase complexes, altering gene expression important for limb development.
• Anti-angiogenic effects, which impair formation of new blood vessels in the developing limb bud.
• Generation of reactive oxygen species (ROS) leading to oxidative stress and embryonic cell damage.

Inhibition of IL-2, IL-4, and IL-5 relates mainly to thalidomide's immunomodulatory effects, not its teratogenic mechanism.

QUESTION

You work under a research scientist who is working on determining the association of adenocarcinoma of pancreas with a compound X. He uses a test whose steps are shown in the image below to determine the mutagenic effect and potential carcinogenicity of compound X. What is this test that he has used?



A CGH

B Micro nucleus test

C Microtox bio-assay

D **Ames test** CORRECT

OK RIGHT ANSWER

D. Ames test

WHY THIS IS RIGHT

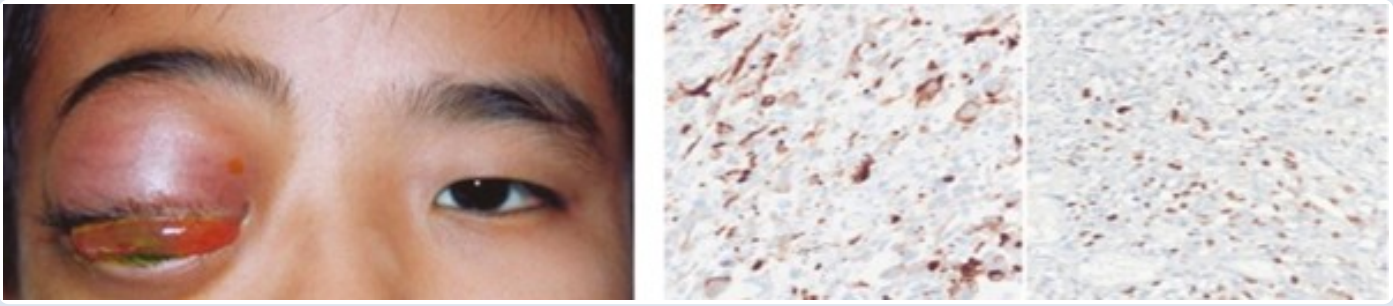
The Ames test is a screening test used to detect mutagenic and potential carcinogenic substances.
• Uses a histidine-requiring mutant strain of Salmonella typhimurium (his□).
• Bacteria are exposed to the test compound along with rat liver extract (S9 fraction) to mimic metabolic activation in humans.
• They are plated on media lacking histidine.
• If the compound is mutagenic, it causes reverse mutation (his□ -> his□), allowing bacteria to grow and form colonies.
• More revertant colonies compared to control -> mutagenic substance -> potential carcinogen.

Why not the other options:

A. CGH (Comparative Genomic Hybridization): Used to detect DNA copy number changes (gene amplification or deletion) in tumors, not mutagenicity.
B. Micronucleus test: Detects chromosomal damage or fragments forming micronuclei in dividing cells, usually in bone marrow or cultured cells.
C. Microtox bioassay: Measures general toxicity using bioluminescent bacteria, not specifically mutations.

QUESTION

4-year-old child was brought to OPD with proptosis of left eye. On investigation, it was found to be a Desmin positive tumour. What can be the likely diagnosis?



A Ewing's sarcoma

B **Embryonal rhabdomyosarcoma** CORRECT

C Leukemia

D Retinoblastoma

RIGHT ANSWER

OK **B. Embryonal rhabdomyosarcoma**

WHY THIS IS RIGHT

Embryonal rhabdomyosarcoma is the most common soft-tissue sarcoma in children, frequently arising in the head and neck region, including the orbit, where it presents with rapidly progressive unilateral proptosis.

Key diagnostic features:
• Occurs in young children.
• Orbital mass -> proptosis.
• Tumor cells show skeletal muscle differentiation.
• Immunohistochemistry: Desmin positive, often myogenin and MyoD1 positive.

QUESTION

Which of the following statements is true about proto-oncogenes?
1. KIT has increased expression in GIST.
2. KRAS mutation is more commonly associated with small-cell lung carcinoma.
3. ERBB2 over-expression is associated with breast carcinomas.
4. PDGFR-β has suppressed expression in colorectal cancers.

A 2 only

B 1 only

C **1 and 3** CORRECT

D 2 and 3

OK RIGHT ANSWER

C. 1 and 3

WHY THIS IS RIGHT

Proto-oncogenes are normal cellular genes that regulate cell growth, proliferation, and survival. When they undergo mutation, amplification, or overexpression, they become oncogenes, promoting uncontrolled cell proliferation.
• KIT overexpression/mutation -> Gastrointestinal stromal tumors (GIST).
• ERBB2 (HER2/neu) amplification -> Breast carcinoma.

Why others are false:

2. KRAS mutations are common in adenocarcinoma of lung, pancreatic, and colorectal cancers-not typically small-cell lung carcinoma.
4. PDGFR-β acts as a growth factor receptor proto-oncogene; cancer usually involves activation/overexpression, not suppression.

QUESTION

Which of the following statements regarding phosphorylation of the RB (Retinoblastoma) protein is true?

A Hypophosphorylated RB allows progression from G1 to S phase

B **Phosphorylation of RB prevents binding to E2F transcription factors** CORRECT

C RB is phosphorylated by cyclin B-CDK1 complex

D RB phosphorylation leads to cell cycle arrest

OK RIGHT ANSWER

B. Phosphorylation of RB prevents binding to E2F transcription factors

WHY THIS IS RIGHT

The retinoblastoma (RB) protein is a tumor suppressor that regulates the G1 -> S phase transition of the cell cycle.
• In its hypophosphorylated (active) state, RB binds to E2F transcription factors, preventing transcription of genes required for S-phase entry.
• When RB is phosphorylated by cyclin D-CDK4/6 and cyclin E-CDK2, it becomes inactive and releases E2F.
• Free E2F activates transcription of genes necessary for DNA synthesis and progression into S phase.

QUESTION

A 46-year-old woman presents with progressive proximal muscle weakness, difficulty climbing stairs, and a violaceous rash over the eyelids. Examination reveals erythematous papules over the knuckles and periungual telangiectasia. Laboratory evaluation shows elevated creatine kinase levels, and muscle biopsy demonstrates perifascicular atrophy. Further evaluation is most likely to reveal an underlying malignancy of which of the following organs?

A Lung

B **Ovary** CORRECT

C Thyroid

D Pancreas

OK RIGHT ANSWER

B. Ovary

WHY THIS IS RIGHT

• Dermatomyositis is an autoimmune inflammatory myopathy characterized by proximal muscle weakness and characteristic skin findings, including heliotrope rash (violaceous rash on eyelids) and Gottron papules (erythematous papules over knuckles).
• Muscle biopsy classically shows perifascicular atrophy.
• In adults, dermatomyositis may occur as a paraneoplastic syndrome, so patients should be screened for an underlying malignancy.
• In women, dermatomyositis is most commonly associated with ovarian carcinoma. Therefore, the malignancy most likely to be detected is ovarian cancer.

QUESTION

Which of the following statements is/are True?
1. The basement membrane remains intact in dysplasia.
2. Dysplasia is an adaptive response to stress or injury.
3. Pleomorphism is observed in metaplasia.
4. Myositis ossificans is an example of dysplasia.

A 1, 3 and 4

B **1 only** CORRECT

C 2 and 4

D 1, 2 and 3

OK RIGHT ANSWER

B. 1 only

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Following the resection of the pituitary adenoma in a patient with Cushing's disease, which intracellular change is most likely in residual adenoma cells?

A Mallory-Denk bodies

B Dutcher bodies

C **Crooke hyaline change** CORRECT

D Woronin bodies

OK RIGHT ANSWER

C. Crooke hyaline change

WHY THIS IS RIGHT

Crooke hyaline change refers to the accumulation of cytokeratin filaments in the cytoplasm of pituitary corticotroph cells due to prolonged exposure to high glucocorticoid levels. Excess cortisol suppresses ACTH production, leading to perinuclear hyaline (glassy) cytoplasmic changes in corticotrophs.

In Cushing disease (ACTH-secreting pituitary adenoma), surrounding normal corticotroph cells show Crooke hyaline change because of chronic cortisol excess.

Other options:

A. Mallory-Denk bodies: cytokeratin aggregates in alcoholic liver disease / steatohepatitis.

B. Dutcher bodies: intranuclear immunoglobulin inclusions in plasma cell disorders.

D. Woronin bodies: structures in fungal cells, not human pathology.

QUESTION

Which of the following measures are associated with an increased life span?

A Moderate or regular exercise for 30 min

B Decrease stress

C **Decreasing calorie intake by 30 percent** CORRECT

D Pharmacological intervention with proton pump inhibitors

OK RIGHT ANSWER

C. Decreasing calorie intake by 30 percent

WHY THIS IS RIGHT

Caloric restriction is the most consistently proven intervention to increase lifespan in multiple experimental models.
Reducing calorie intake by about 30% slows metabolic rate and decreases oxidative stress.
It activates longevity pathways involving sirtuins and AMPK, delaying cellular aging.

Exercise and stress reduction improve health span, but calorie restriction shows the strongest lifespan extension effect.

QUESTION

Which of the following proteins functions as a pro-apoptotic factor?

A MCL

B BCL-2

C **BAX** CORRECT

D BCL-XL

OK RIGHT ANSWER

C. BAX

WHY THIS IS RIGHT

Cell stress / DNA damage
↓
BAX / BAK activation
↓
Mitochondrial membrane permeabilization
↓
Cytochrome c release
↓
Caspase activation
↓
Apoptosis

BAX is a pro-apoptotic member of the BCL-2 family that promotes mitochondrial outer membrane permeabilization. It facilitates release of cytochrome c from mitochondria, activating the intrinsic apoptotic pathway.
This leads to caspase activation and programmed cell death.
BCL-2, BCL-XL, and MCL-1 are anti-apoptotic proteins that inhibit mitochondrial apoptosis.

Pro-apoptotic Genes (BH1-3)	Anti-apoptotic Genes	Apoptosis Initiators/Sensors
BAK gene	BCL-2 gene (most important)	BIM gene
BAX gene	BCL-XL gene	BAD gene
p53 gene	MCL1 gene	PUMA gene
Glucocorticoids	Sex (Love) steroids	NOXA gene

QUESTION

Which of the following statements is false regarding neutrophil extracellular trapping (NET)?

A It is detected in blood during sepsis

B It is produced in response to bacterial infection.

C **Mitochondrial DNA is seen.** CORRECT

D It is chromatin with antibacterial enzymes.

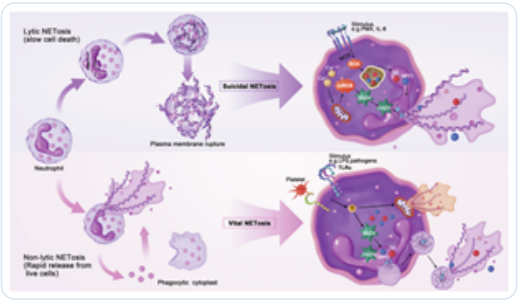
OK RIGHT ANSWER

C. Mitochondrial DNA is seen.

WHY THIS IS RIGHT

Bacterial infection
↓
Neutrophil activation
↓
Nuclear chromatin decondensation
↓
DNA + enzymes released
↓
NET formation -> traps microbes

Neutrophil extracellular traps (NETs) consist of decondensed nuclear chromatin decorated with antimicrobial enzymes like elastase and myeloperoxidase.
They are released in response to bacterial infections and are detectable in sepsis.
NETs are composed primarily of nuclear DNA, not mitochondrial DNA.
Hence, the statement regarding mitochondrial DNA is false.



QUESTION

Choose the correct statement regarding the telomerase theory of aging?

A Telomere stability is associated with aging

B Abnormal telomerase activation is associated with aging

C **Decreased telomere length is associated with aging** CORRECT

D Increased telomere length is associated with aging

RIGHT ANSWER

OK

C. Decreased telomere length is associated with aging

WHY THIS IS RIGHT

Telomeres shorten progressively with each cell division due to incomplete replication of chromosome ends.
Reduced telomere length leads to replicative senescence and loss of cellular proliferative capacity.
This cumulative telomere attrition is a key mechanism underlying cellular aging.
Telomerase activity maintains telomere length in germ cells and cancers, not in normal aging cells.

Feature	Aging	Cancer
Telomere length	□ Decreased	Maintained/increased
Telomerase	Low	High
Cell fate	Senescence	Immortality

QUESTION

Caspases are involved in which of the following types of programmed cell death?

A Necroptosis and Apoptosis

B **Pyroptosis and Apoptosis** CORRECT

C Ferroptosis and Apoptosis

D Necrosis and Apoptosis

RIGHT ANSWER

OK

B. Pyroptosis and Apoptosis

WHY THIS IS RIGHT

Caspases = cysteine proteases
• Exist as inactive precursors
• Get activated during programmed cell death

Caspases are central mediators of apoptosis, with initiator and executioner caspases driving programmed cell death.
Pyroptosis is a caspase-dependent inflammatory cell death mediated mainly by caspase-1.
Necroptosis and ferroptosis are caspase-independent forms of regulated cell death.
Hence, caspases are involved in apoptosis and pyroptosis.

QUESTION

Which of the following is not helpful for differentiating between necrosis and apoptosis?

A Intact cell organelle

B **Altered cell shape** CORRECT

C Enzyme mediated degradation

D Internal organelle disintegration with lipid change

OK RIGHT ANSWER

B. Altered cell shape

WHY THIS IS RIGHT

Both necrosis and apoptosis show altered cell shape, making it non-discriminatory.
Apoptosis preserves cell membrane integrity and organelles until late stages, unlike necrosis.
Apoptosis is enzyme-mediated via caspases, whereas necrosis is unregulated.
Necrosis shows organelle disintegration with lipid peroxidation and membrane rupture.

Feature	Necrosis	Apoptosis
Cell size	Enlarged (swelling)	Reduced (shrinkage)
Nucleus	Pyknosis → karyorrhexis → karyolysis	Fragmentation into nucleosome-sized fragments
Plasma membrane	Disrupted	Intact; altered lipid orientation
Cellular contents	Enzymatic digestion	Intact
Inflammation	Frequent	Absent
Role	Invariably pathologic	Often physiologic

QUESTION

Which type of programmed cell death primarily involves the action of caspase-1?

A Apoptosis

B **Pyroptosis** CORRECT

C Necroptosis

D Necrosis

RIGHT ANSWER

OK

B. Pyroptosis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following statements about free radicals is incorrect?

A Lipid peroxidation

B SOD inactivates free radicals

C Produced by neutrophils

D **Causes Fibrinoid necrosis** CORRECT

RIGHT ANSWER

OK

D. Causes Fibrinoid necrosis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A patient is scheduled for coagulation testing, including prothrombin time (PT) and activated partial thromboplastin time (APTT). Which one of the following vacutainers is the most appropriate choice for this purpose?

A Lavender-colored tube

B **Blue-colored tube** CORRECT

C Red-colored tube

D Gray-colored tube

RIGHT ANSWER

OK **B. Blue-colored tube**

WHY THIS IS RIGHT

Vial Color & Additive	Uses
Blue (Trisodium citrate)	• Coagulation studies (PT, aPTT, D-dimer) • Platelet function studies
Red (Plain vial / Clot activator)	• Serum samples • Biochemistry, serology
Yellow (SPS / ACD)	• Blood culture (SPS) • HLA typing (ACD)
Green (Heparin)	• Light green (Lithium heparin): ABG, electrolytes • Dark green (Sodium heparin): Biochemistry, osmotic fragility
Lavender / Purple (EDTA)	• CBC (Complete blood count) • Peripheral smear • HbA1c • ESR (Wintrobe method)
Grey (Sodium fluoride + Potassium oxalate)	• Glucose estimation • Lactate

QUESTION

A 30-year-old man develops fever, chills, back pain, and hemoglobinuria within 1 hour of receiving a blood transfusion. The transfusion is immediately stopped. Laboratory investigations reveal elevated serum LDH. Which of the following best explains the underlying immunopathogenesis of this reaction?

A Type 1 hypersensitivity reaction

B **Type 2 hypersensitivity reaction** CORRECT

C Type 3 hypersensitivity reaction

D Type 4 hypersensitivity reaction

OK RIGHT ANSWER

B. Type 2 hypersensitivity reaction

WHY THIS IS RIGHT

Type II Hypersensitivity- Antibodies (IgG/IgM) target cell surface antigens
Leads to:
o Complement activation
o Cell destruction

Wrong blood transfusion (ABO mismatch)
↓
Recipient antibodies (IgM)
↓
Bind donor RBC antigens
↓
Complement activation
↓
Intravascular hemolysis
↓
Hemoglobinuria + ↑ LDH
The presentation is consistent with an acute hemolytic transfusion reaction.
Preformed recipient IgM or IgG antibodies react against donor RBC antigens.
This causes complement-mediated intravascular hemolysis with hemoglobinuria and raised LDH.
Such antibody-mediated cytotoxic reactions are classified as type II hypersensitivity.

QUESTION

A patient with renal disease undergoes a biopsy. On Congo red staining, the deposits show apple-green birefringence under polarized light. What is the most likely diagnosis?

A Minimal change disease

B Diabetic nephropathy

C **Amyloidosis** CORRECT

D Sarcoidosis

OK RIGHT ANSWER

C. Amyloidosis

WHY THIS IS RIGHT

Apple-green birefringence on Congo red staining under polarized light is pathognomonic for amyloid deposition. Amyloid fibrils form β-pleated sheets that bind Congo red in a characteristic way. Renal amyloidosis causes proteinuria and progressive renal dysfunction.

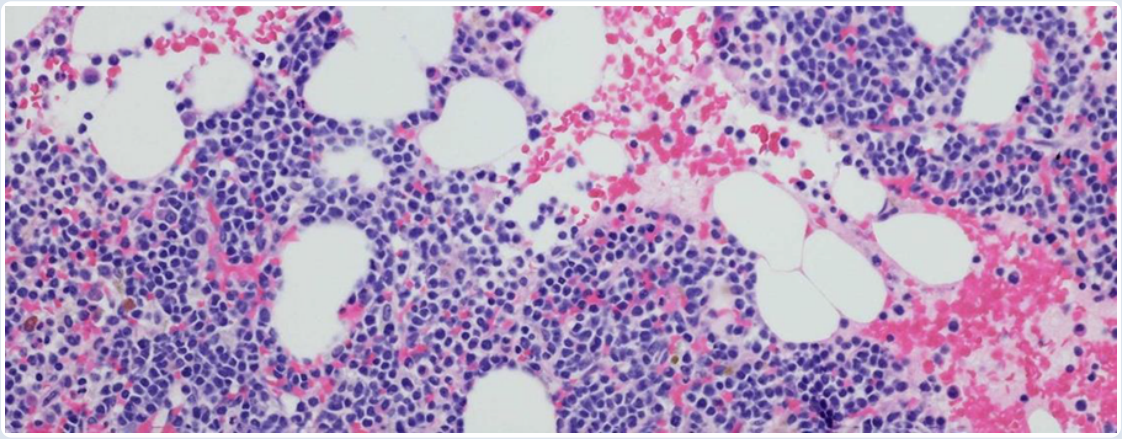
A. Minimal change disease
Seen on:
Electron microscopy -> foot process effacement
• No Congo red positivity

B. Diabetic nephropathy
Shows:
• Kimmelstiel-Wilson nodules

D. Sarcoidosis
Shows:
• Non-caseating granulomas

QUESTION

A 53-year-old woman presents with complaints of fatigue, jaundice, edema, and hepatosplenomegaly. She had anemia and neutropenia. Her serum creatinine is 3.2 mg/dL, and her bone marrow examination showed 22% plasma cells, and her serum Ig levels were 3.0 g/dL. The bone marrow biopsy is shown below. What is the associated histology?



A Monoclonal gammopathy of undetermined significance

B Bone marrow - fibrinoid necrosis

C **Amyloidosis** CORRECT

D Myxomatous degeneration of the bone marrow

RIGHT ANSWER

OK

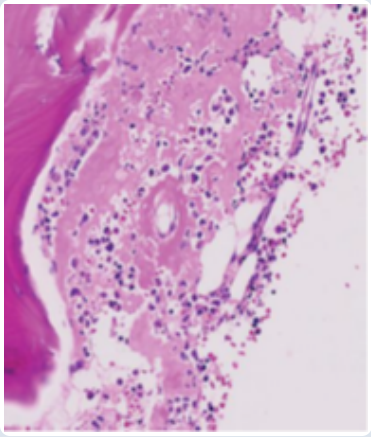
C. Amyloidosis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A patient presents with hepatosplenomegaly with bone marrow having 22% plasma cells. The bone marrow biopsy had a pink acellular deposition of material as shown. What is the likely diagnosis?



A Bone marrow necrosis

B T.B.

C Myxoid degeneration

D **Amyloidosis** CORRECT

RIGHT ANSWER

OK **D. Amyloidosis**

WHY THIS IS RIGHT

Plasma cell proliferation
↓
Excess light chains
↓
Misfolding -> β-pleated sheets
↓
Extracellular deposition
↓
Organ dysfunction (liver, spleen, marrow)

The pink, amorphous, acellular extracellular deposition in bone marrow is characteristic of amyloid.

Amyloidosis is commonly associated with plasma cell dyscrasias due to AL (light chain) protein deposition.
Hepatosplenomegaly and increased plasma cells support an underlying plasma cell disorder.
Bone marrow necrosis shows ghost cells, TB shows granulomas, and myxoid degeneration has basophilic stroma.

QUESTION

Match the mode of inheritance with the disorder in the appropriate column:

1. Autosomal dominant	A. Duchene Muscular dystrophy
2. X Linked Recessive	B. Lebers disease
3. Autosomal Recessive	C. Cystic fibrosis
4. Mitochondrial	D. Myotonia dystrophica

A 1-A, 2-D, 3-C, 4-B

B **1-D, 2-A, 3-C, 4-B** CORRECT

C 1-C, 2-A, 3-D, 4-B

D 1-A, 2-D, 3-B, 4-C

OK RIGHT ANSWER

B. 1-D, 2-A, 3-C, 4-B

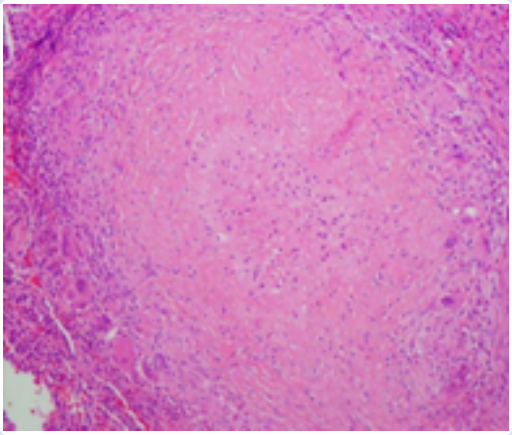
WHY THIS IS RIGHT

Myotonic dystrophy shows autosomal dominant inheritance due to trinucleotide repeat expansion, CTG trinucleotide repeat expansion.
Duchenne muscular dystrophy is X-linked recessive, caused by dystrophin gene deletion.
Cystic fibrosis is an autosomal recessive disorder due to CFTR mutation.
Leber's hereditary optic neuropathy follows mitochondrial inheritance via maternal transmission.

Pattern	Key Clue	Example
AD	Every generation	Myotonic dystrophy
XLR	Males affected	Duchenne
AR	Skips generations	CF
Mitochondrial	Maternal only	LHON

QUESTION

A patient presents with fever, cough, weight loss, histology of the pathology is as shown below. What is the most likely cytokine involved?



A **Interferon gamma** CORRECT

B Complement 5a protein

C Histamine

D Prostaglandin

RIGHT ANSWER

OK **A. Interferon gamma**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A physician wanted to perform D-Dimer using the vacutainer shown in the image. Which of the following is the anticoagulant used for this test?

A **Trisodium Citrate** CORRECT

B Potassium oxalate

C Sodium fluoride

D EDTA

RIGHT ANSWER

OK **A. Trisodium Citrate**

WHY THIS IS RIGHT

The vacutainer shown has a light blue cap, which is specifically used for coagulation studies such as D-dimer.

Key fact:
• Light blue top tube -> contains Trisodium citrate (3.2% or 3.8%)
• It works by chelating calcium reversibly, preserving coagulation factors for testing.

Option analysis:
• A. Trisodium citrate -> □ Correct (used for PT, aPTT, D-dimer)
• B. Potassium oxalate -> Used with gray top (glucose estimation)
• C. Sodium fluoride -> Gray top (inhibits glycolysis for glucose)
• D. EDTA -> Purple top (CBC, hematology)

QUESTION

Match the following:

Mode of Inheritance	Disease
A. AD	1. LHON
B. AR	2. Myotonic Dystrophy
C. XR	3. Cystic Fibrosis
D. Mitochondrial	4. Duchenne Muscular Dystrophy

A. A-1, B-2, C-3, D-4

B. A-4, B-3, C-1, D-2

C. **A-2, B-3, C-4, D-1** CORRECT

D. A-2, B-3, C-1, D-4

RIGHT ANSWER

OK **C. A-2, B-3, C-4, D-1**

WHY THIS IS RIGHT

Myotonic dystrophy follows autosomal dominant inheritance due to trinucleotide repeat expansion.
Cystic fibrosis is an autosomal recessive disorder caused by CFTR gene mutation.
Duchenne muscular dystrophy is inherited in an X-linked recessive pattern due to dystrophin gene deletion.
Leber's hereditary optic neuropathy shows mitochondrial inheritance via maternal transmission.

Pattern	Key Feature	Example
AD	Every generation	Myotonic dystrophy
AR	Skips generations	CF
XLR	Males affected	Duchenne
Mitochondrial	Maternal only	LHON

QUESTION

Which vacutainer is commonly used to collect blood samples for serum electrolyte analysis?

A Blue

B Yellow

C **Green** CORRECT

D Purple

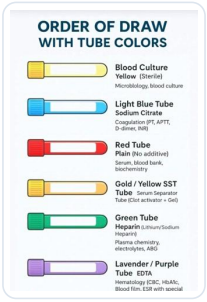
OK RIGHT ANSWER

C. Green

WHY THIS IS RIGHT

Need electrolyte measurement
↓
Avoid clotting + avoid ion interference
↓
Use heparin (green tube)
↓
Accurate electrolyte values

Serum electrolyte analysis commonly uses a green-top vacutainer containing heparin as anticoagulant.
Heparin prevents clotting without chelating ions, thus preserving true electrolyte levels.
EDTA (purple) chelates calcium and alters potassium, while citrate (blue) dilutes the sample.
Yellow-top tubes are used for serum separation, but may also be used for routine electrolyte estimation making the answer controversial.



QUESTION

For HbA1c sample, the vacutainer used contains which of the following?

A Warfarin

B Sodium fluoride

C **EDTA** CORRECT

D Heparin

RIGHT ANSWER

OK

C. EDTA

WHY THIS IS RIGHT

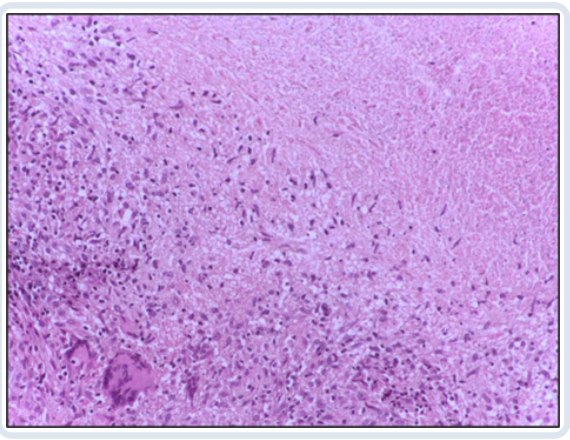
Need to analyze RBCs
↓
Prevent clotting without damaging cells
↓
Use EDTA
↓
Accurate HbA1c measurement

Vacutainer Vials and Their Uses

Vials	Uses
Blue (Trisodium citrate)	Coagulation studies (PT, aPTT studies), Platelet Study (Platelet-rich plasma)
Red (Plain vial)	Serum samples
Yellow	Bacterial culture, HLA typing
Green (Emergency Testing) **(Light – Li heparin** **Dark – Na heparin)**	ABG, Electrolytes, Osmotic fragility
Lavender (EDTA)	CBC, Immunohistochemistry studies, ESR - Westergren method, HbA1C
Grey (Additive: NaF) **(Anticoagulant: potassium oxalate)**	Glucose estimation

QUESTION

Histological examination of a cervical lymph node biopsy is shown below. What is the likely diagnosis?



A Sarcoidosis

B **Tuberculosis** CORRECT

C Langerhans cell histiocytosis

D Hodgkin lymphoma

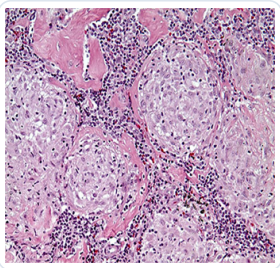
RIGHT ANSWER

OK **B. Tuberculosis**

WHY THIS IS RIGHT

Mycobacterium tuberculosis
↓
Macrophage ingestion
↓
T-cell activation (Th1)
↓
IFN-γ release
↓
Granuloma formation
↓
Caseous necrosis
Tuberculosis shows caseating granulomatous inflammation with central amorphous necrosis. Granulomas are composed of epithelioid histiocytes and Langhans-type giant cells.
Cervical lymph node involvement is common in tuberculous lymphadenitis.

Sarcoidosis shows non-caseating granulomas as shown below.
Langerhans cell histiocytosis has grooved nuclei, and Hodgkin lymphoma shows Reed-Sternberg cells.



QUESTION

Which of the following is/are criteria for anaphylaxis?
a. Persistent abdominal cramps and vomiting
b. Hypertension
c. Urticaria and swollen lips
d. Respiratory distress

A a, b & c are correct

B b & c are correct

C **a, c & d** CORRECT

D All of the above

OK RIGHT ANSWER

C. a, c & d

WHY THIS IS RIGHT

Allergen exposure
↓
IgE on mast cells cross-links
↓
Mast cell degranulation
↓
Histamine, leukotrienes release
↓
Vasodilation + bronchospasm + edema
↓
Anaphylaxis
Anaphylaxis is an acute, systemic hypersensitivity reaction involving skin, mucosa, respiratory, and gastrointestinal systems.
Diagnostic criteria include urticaria with angioedema, respiratory compromise, and persistent gastrointestinal symptoms like cramps and vomiting.

System	Features
Skin	Urticaria, angioedema
Respiratory	Dyspnea, wheeze, stridor
Cardiovascular	Hypotension
GI	Vomiting, cramps

Hypotension, not hypertension, is the cardiovascular manifestation of anaphylaxis.
Hence, options a, c, and d fulfil criteria for anaphylaxis.

QUESTION

Which of the following should be prevented when collecting a blood sample for serum calcium?

A **Making a fist with forearm exercise** CORRECT

B Tourniquet should be tied and released as soon as possible

C Should be collected in sitting position & better to avoid prolonged immobilization

D Sample should be analyzed within 15-30 min of collection

OK RIGHT ANSWER

A. Making a fist with forearm exercise

WHY THIS IS RIGHT

Repeated fist clenching = forearm muscle activity. This causes:
• Local metabolic changes
• Hemoconcentration
• Altered ion balance

Result: Falsely elevated calcium (and other analytes like potassium)
Fist clenching / forearm exercise
↓
Muscle contraction
↓
↑ Local metabolites + hemoconcentration
↓
Altered serum composition
↓
False lab values (↑ Ca, ↑ K)

Factor	Effect
Fist clenching	False ↑ calcium
Prolonged tourniquet	↑ protein-bound calcium
Posture change	Alters calcium levels
Delayed processing	pH changes -> affects Ca binding

QUESTION

Match the following pathological bodies with their associated condition.

Column A	Column B
A. Parkinson's Disease	1. Dutcher body
B. Rabies	2. Negri body
C. Multiple myeloma	3. Lewy body
D. Malakoplakia	4. Michaelis-Gutmann body

A A-4, B-3, C-1, D-2

B A-3, B-2, C-4, D-1

C A-2, B-1, C-3, D-4

D **A-3, B-2, C-1, D-4** CORRECT

OK RIGHT ANSWER

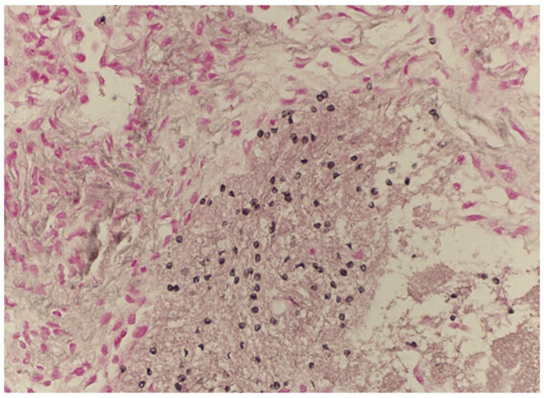
D. A-3, B-2, C-1, D-4

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

A post-renal transplant patient presents with fever and breathlessness. Lung biopsy is done. Which of the following stains is least useful in identifying the causative organism?



- A **Congo red** — CORRECT
- B Periodic Acid-Schiff
- C Gomori Methenamine Silver
- D Fontana-Masson

RIGHT ANSWER

OK **A. Congo red**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

Which of the following are correctly matched with respective stain and its properties?
1. Perl's - Iron, red
2. Alizarin - Calcium, black
3. Giemsa - Mast cells, purple
4. Von Gieson - Elastin fibres, blue

A 1 and 2

B 2 and 4

C **3 only** CORRECT

D All of the above

RIGHT ANSWER
OK
C. 3 only

WHY THIS IS RIGHT

The source quiz contains the placeholder "$1f" here and does not include a written explanation for this item.

QUESTION

A 4-year-old patient presented with a rectal mass 2.5 cm by 3 cm. On biopsy it had the presence of colonic mucosa with multiple dilated glands filled with mucin and inflammatory cells. Which of the following is a likely diagnosis?

A Choristoma

B **Hamartoma** CORRECT

C Adenoma

D Adenocarcinoma

RIGHT ANSWER

OK

B. Hamartoma

WHY THIS IS RIGHT

Hamartoma is a benign, disorganized overgrowth of tissue native to the organ.
In the colon, it shows normal colonic mucosa with cystically dilated glands filled with mucin and inflammatory cells.
These lesions commonly present as rectal polyps in children.

Feature	Hamartoma	Choristoma	Adenoma	Adenocarcinoma
Tissue type	Native	Ectopic	Neoplastic	Malignant
Organization	Disorganized	Normal	Dysplastic	Atypical
Age	Children	Any	Adults	Adults
Malignancy	□ No	□ No	Premalignant	Yes

QUESTION

Match the following cancers with their respective genes involved:

1. KIT	A. Myeloproliferative proliferative neoplasm
2. K-RAS	B. Adenocarcinoma lung
3. JAK 2	C. Pancreatic Carcinoma
4. ALK	D. GIST

A 1-C, 2-D, 3-A, 4-B

B 1-B, 2-A, 3-D, 4-C

C **1-D, 2-C, 3-A, 4-B** CORRECT

D 1-C, 2-A, 3-D, 4-B

OK RIGHT ANSWER

C. 1-D, 2-C, 3-A, 4-B

WHY THIS IS RIGHT

• KIT mutations are characteristic of gastrointestinal stromal tumors due to constitutive tyrosine kinase activation.

K-RAS mutations are commonly seen in pancreatic adenocarcinoma driving uncontrolled proliferation.
JAK2 mutations are associated with myeloproliferative neoplasms via persistent JAK-STAT signalling. Seen in:
• Polycythemia vera
• Essential thrombocythemia
• Primary myelofibrosis

ALK rearrangements are classically linked to lung adenocarcinoma. EML4-ALK fusion.

QUESTION

Match the following:

Column A	Column B
A. AFP	1. Medullary Carcinoma of Thyroid
B. Beta-HCG	2. Hepatocellular carcinoma
C. Catecholamines	3. Choriocarcinoma
D. Calcitonin	4. Pheochromocytoma

A A-3, B-1, C-2, D-4

B A-4, B-2, C-1, D-3

C A-1, B-4, C-3, D-2

D **D. A-2, B- 3, C-4, D-1** CORRECT

OK RIGHT ANSWER

D. D. A-2, B- 3, C-4, D-1

WHY THIS IS RIGHT

AFP is a tumor marker for hepatocellular carcinoma due to fetal liver protein re-expression. Elevated in:
• HCC , Hepatoblastoma
• Yolk sac tumors

β-hCG is characteristically elevated in choriocarcinoma from trophoblastic proliferation & in hydatidiform mole.

Catecholamines -> Pheochromocytoma
Tumor of: Adrenal medulla
Secretes: Epinephrine, norepinephrine
Measured as: Metanephrines / VMA

Calcitonin is produced by parafollicular C cells and is a marker of medullary thyroid carcinoma.

Marker	Source	Tumor
AFP	Fetal liver	HCC
β-hCG	Placenta	Choriocarcinoma
Catecholamines	Adrenal medulla	Pheochromocytoma
Calcitonin	Thyroid C cells	MTC

QUESTION

Epithelial mesenchymal transcription is mediated by which transcription factors?

A Cathepsin D

B **SNAIL/TWIST** CORRECT

C MMP-9

D TTF-1

RIGHT ANSWER

OK **B. SNAIL/TWIST**

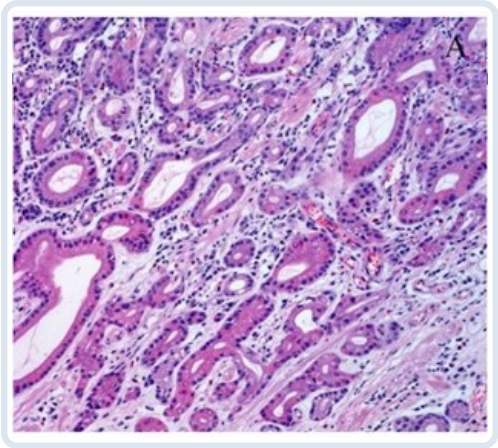
WHY THIS IS RIGHT

Epithelial cell
↓
SNAIL / TWIST activation
↓
↓ E-cadherin (cell adhesion)
↓
Loss of polarity
↓
Gain of motility
↓
Invasion -> Metastasis
Epithelial-mesenchymal transition (EMT) is driven by transcription factors that repress epithelial markers like E-cadherin.
SNAIL and TWIST are key EMT transcription factors that promote loss of cell adhesion and increased motility.
They induce mesenchymal phenotype, facilitating invasion and metastasis.

Cathepsin D and MMP-9 are proteases involved in matrix degradation, while TTF-1 is a lineage-specific transcription factor, not EMT-related.
Tumor cell
↓
EMT (SNAIL/TWIST)
↓
↓ E-cadherin
↓
Basement membrane breakdown (MMPs)
↓
Invasion -> Intravasation -> Metastasis

QUESTION

Which of the following is the most likely tumor arising from the slide shown in the image?



A Squamous cell carcinoma

B **Adenocarcinoma** CORRECT

C Lymphoma

D Sarcoma

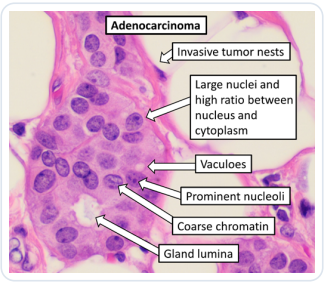
OK RIGHT ANSWER

B. Adenocarcinoma

WHY THIS IS RIGHT

Epithelial malignancy
↓
Gland formation / acinar pattern
↓
Mucin secretion
↓
Cellular atypia
↓
Adenocarcinoma
Adenocarcinoma is characterized by malignant epithelial cells forming glandular or acinar structures. Tumor cells show luminal formation, mucin production, and gland-like architecture.

This reflects origin from glandular epithelium or epithelium with secretory differentiation.
Squamous cell carcinoma shows keratinization, lymphoma shows diffuse sheets of lymphoid cells, and sarcoma is mesenchymal without gland formation.



QUESTION

All of the following are growth promoting proto-oncogenes except?

A FGF

B PDGF

C TGF alpha

D **TGF beta** CORRECT

RIGHT ANSWER

OK

D. TGF beta

WHY THIS IS RIGHT

TGF-β is growth-inhibitory, not growth-promoting.
Proto-oncogenes encode growth factors that stimulate cell proliferation and survival.
FGF, PDGF, and TGF-α act as mitogenic signals promoting cell cycle progression.
Growth factors (FGF, PDGF, TGF-α)
↓
Receptor activation
↓
RAS/MAPK pathway
↓
Cell proliferation

TGF-β, in contrast, inhibits epithelial cell proliferation by inducing cell cycle arrest via SMAD signalling. Loss of TGF-β signalling contributes to oncogenesis, but the gene itself functions as a tumor suppressor.

Factor	Function	Role in Cancer
FGF	Proliferation, angiogenesis	Oncogenic
PDGF	Cell growth	Oncogenic
TGF-α	EGFR activation	Oncogenic
TGF-β
“Beta” = brake □	Growth inhibition	Tumor suppressor

QUESTION

Which of the following malignancies has the least or no established association with obesity?

A Meningioma

B Pancreatic cancer

C **Gastric pyloric cancer** CORRECT

D Multiple myeloma

RIGHT ANSWER

OK **C. Gastric pyloric cancer**

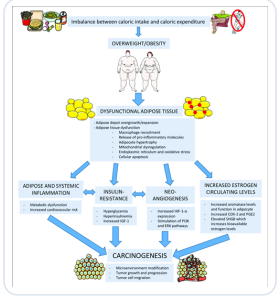
WHY THIS IS RIGHT

Obesity
↓
↑ Insulin & IGF-1
↑ Estrogen (via adipose)
↑ Inflammatory cytokines
↓
Cell proliferation + ↓ apoptosis
↓
Obesity-Associated Cancers

Strong Association	Moderate/Other Associations
Breast (postmenopausal)	Meningioma
Endometrial	Multiple myeloma
Colon	Thyroid
Pancreatic	Kidney
Esophageal adenocarcinoma	Liver

Gastric pyloric cancer is more strongly linked to *H. pylori* infection, nitrosamines, smoking, and dietary salt.
Gastric Cancer Types

Type	Location	Risk Factors
Intestinal type	Distal (pylorus)	H. pylori, diet
Diffuse type	Whole stomach	CDH1 mutation



QUESTION

Which of the following is the best gene analysis investigation for a female patient with history of breast and ovarian cancer?

A PTEN gene

B P53 gene

C **BRCA1** CORRECT

D CDH1 gene

OK RIGHT ANSWER

C. BRCA1

WHY THIS IS RIGHT

BRCA1 and BRCA2 are tumor suppressor genes involved in homologous recombination DNA repair. Think of them as: *molecular tailors fixing torn DNA strands*
BRCA mutation
↓
Defective DNA repair
↓
Genomic instability
↓
Accumulation of mutations
↓
Cancer (breast, ovary, others)
Germline mutations markedly increase risk of breast and ovarian cancers in women. This combination of cancers is the classic clinical clue for hereditary breast-ovarian cancer syndrome.

Gene	Function	Associated Cancers
BRCA1/2	DNA repair	Breast, ovarian, prostate
PTEN	Cell cycle regulation	Breast, thyroid, endometrium
p53	DNA damage checkpoint	Sarcoma, breast, brain
CDH1	Cell adhesion	Diffuse gastric, lobular breast

QUESTION

Which of the following is the incorrect association?

A PTEN and thyroid

B BRCA2 and prostate

C STK11 and breast cancer

D **p53 and mucosal neuroma** CORRECT

RIGHT ANSWER

OK

D. p53 and mucosal neuroma

WHY THIS IS RIGHT

The source quiz contains the placeholder “$20” here and does not include a written explanation for this item.

QUESTION

Which of the following best describes the mechanism by which mutations in Ras/ MAP kinase gene cause oncogenicity?

A **Constitutive activation of GTP-ase domain leading to continuous cell proliferation** CORRECT

B Enhanced DNA repair leading to genomic instability

C Inactivation of tumor suppressor gene resulting in loss of cell cycle check point

D Increased activity of tyrosine kinase receptors through gene amplification

OK RIGHT ANSWER

A. Constitutive activation of GTP-ase domain leading to continuous cell proliferation

WHY THIS IS RIGHT

The source quiz contains the placeholder “$21” here and does not include a written explanation for this item.