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INTRODUCTION TO MEDICAL IMMUNOLOGY

MID AND FINAL MATERIAL QUESTIONS

43 Questions with answers and Explanations

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Mid Material Questions

Q1: One of the following is not a part of complement system:

A: ADCC

B: C3bBb

C: MAC

D: Mannose-Binding Lectin

E: $C1qr^2s^2$

Correct answer: A.

C3bBb is C3 convertase of the alternative pathway; membrane attack complex is formed after C3 convertase of any pathway is formed; MBL is the protein that binds mannose on pathogen surface and has its own pathway, and $C1qr^2s^2$ is the first complement complex to be formed in classical pathway. Antibody-dependent Cell-mediated cytotoxicity is a mechanism by which natural killers bind To IgG antibody to be stimulated TO KILL!

Q2: All sentences are true about NK cells EXCEPT:

A: NKs Mature in the bone marrow

B: NKs kill by apoptosis, mainly through fas-fas ligand

C: NKs Undergo gene recombination

D: Have an inhibitory receptor that recognizes MHC1 on cells

E: ITAMs respond to activating signals when NKs recognize altered proteins on target cells.

Correct answer: C.

All statements other than C are true; they are of hematopoietic origin so they originate and mature in the BM, they use apoptosis to kill when activating receptor is on; they have another inhibitory receptor to tell them to hold on and NOT KILL if the cell is normal, and they have intracellular areas (ITAMs) in activating receptors that get phosphorylated when the activating receptor is bound to altered protein so the NK can do its job and KILL!

Q3: What's the Difference between innate and adaptive immune system?

A: innate immunity cells are hematopoietic in origin while adaptive cells are not.

B: Innate system sees self-antigens but without doing anything but adaptive is self-reacting.

C: innate system is only cells but adaptive system contains cells and proteins.

D: The innate system uses phagocytosis as a defensive mechanism while adaptive system uses apoptosis.

Correct answer: D. **controversial answer**

Both systems' cells are from the BM; both are non-self-reacting "don't attack self-antigens normally"; both have cellular and protein components "innate has complement system as well as cells , and adaptive has cellular and Ab humoral response". D is the most close to right "although I'm not sure" but let's say that D is ture because mainly in adaptive, cells kill by apoptosis, while in innate, cells -excpet Nk- kill by phagocytosis.

Q4: Which of the following statements is incorrect regarding complement system?

A: C3 convertase in the alternative pathway is C3bBb.

B: MAC is only activated in classical pathway.

C: DAF can accelerate destruction of C3 convertase.

D: Activation of C1 complex in the classical pathway occurs when C1q binds to IgM or IgG.

E: Mannose is the sugar that MBL binds to in Mannose-Lectin pathway.

Correct answer: B.

C3bBb is C3 convertase of the alternative pathway; membrane attack complex is formed after C3 convertase of **any pathway** is formed; DAF is actually an abbreviation for "Decay Accelerating Factor"; C1qr2s2 is the complement complex formed in classical pathway after C1q component binds to the Ab bound to its antigen; MBL is the protein that binds mannose on pathogen surface and has its own pathway.

Q5: Which of the following cells possesses Phagocytic activity?

A: neutrophils

B: NK cells

C: B lymphocytes

D: T lymphocytes

E: All of the above

Correct answer: A.

All the other cells kill by APOPTOSIS

Q6: Wrong about BCR:

A: disulfide bonds are between different chains only

B: It's an immunoglobulin, and it's either IgM or IgD

C: It's selected through modular design during B cell maturation in the bone marrow.

D: It has a hydrophobic trans-membrane domain that's not found in circulating Igs

E: Productive rearrangement of light chain **and** heavy chain complementing light chain are needed for the cell to not die by apoptosis.

Correct answer: A.

Disulfide bonds exist **also** between domains of chains; BCR is selected through modular design by VDJ recombination; the hydrophobic domain is what anchors BCR to cell membrane; the 2 conditions of E are the mandatory steps after VDJ recombination for the cell to produce a BCR good enough to be sent out for recognition of its cognate antigen, if only one happened not BOTH, the cell will DIE BY APOPTOSIS!!

Q7: IgG has a long half-life due to

A: Multiple binding sites

B: Flexibility

C: Ability to act as an opsonin

D: Its trans-membrane domain

E: Ability to bind neonatal Fc receptor

Correct answer: E.

IgG has only 2 sites and it's the simple typical Ab structure; flexibility due to hinge region exists in all antibodies; being an opsonin has nothing to do with IgG's long half-life because not all IgG subtypes work the same; there's no trans membrane domain for IgG because it only exists in circulating form. So binding nFC receptor sequesters IgG away from lysosomal degradation giving it its long t1/2

Q8: What do B lymphocytes need for activation?

A: Antigen presenting cells

B: Th cells that are strictly required for B cell activation.

C: recognition of the cognate antigen only

D: Cross linking only

E: A+C

Correct answer: B.

B cells need no APCs to present antigens; Recognition of cognate antigen and cross linking are only the first 2 steps to reach activation; T cells are which provide Co-stimulatory signal (CD40-CD40L) needed to initiate B cell activation and further Maturation into either a plasma cell or memory B-cell (class switching…)

Q9: The Hinge region exists between:

A: light chain and heavy chain

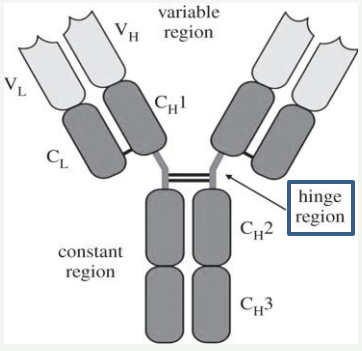
B: Variable region and constant region

C: Variable region VL and Variable region VH

D: C_H1 Region and C_H2 Region

E: It doesn't exist in the Ig structure

Correct answer: D.



Q10: What's the difference between BCR & circulating Ig?

A: BCR has VDJ region while Circulating Igs don't have it

B: Different BCRs were selected through modular design while circulating Igs all have the same Amino Acid sequence.

C: trans-membrane region of hydrophobic amino acid sequences exists only in BCRs but not in circulating Igs.

D: when the cell undergoes class switching, released circulating Igs will recognize an antigen different than the one that BCRs are recognizing.

E: None of the above

Correct answer: C.

Both have VDJ region, both have undergone modular design to recognize different cognate antigens; Circulating antibodies released from a plasma cell will recognize the SAME antigen as the BCR of that plasma cell because only Fc region differ between Abs classes but variable region that recognizes antigens is the same "hence they fight in the same battle!!"

Q11: the most Abundant Antibody in the body is:

A: IgG

B: IgM

C: IgE

D: IgD

E: IgA

Correct answer: E.

IgG is the most abundant Ab in blood, But IgA is the most abundant in the body.

Q12: Which of the following chains isn't polymorphic?

A: HLA-DP β-Chain

B: HLA-C α-Chain

C: HLA-DQ β-Chain

D: β2- microglobulin

E: HLA-B α-Chain

Correct answer: D.

A and C represent chains of MHC 2 molecule, and both chains of MHC 2 molecule are polymorphic; B and E represent alpha chain in MHC 1 molecule which is polymorphic; B2 microglobulin is the small non polymorphic chain of MHC1 molecule.

Q13: Which of the following is true regarding MHC 1?

A: consists of heavy chain and beta microglobulin chain

B: presents exogenous peptides

C: recognized by CD4+ cells

D: Exists only on Antigen presenting cells

E: B+C

Correct answer: A.

All the other statements are features of MHC2 molecule. MHC1 presents endogenous peptides for CD8+ T-cells to recognize, and they exist on all cells of the body.

Q14: A CD8+ cell needs all these for maturation EXCEPT

A: RAG1 + RAG2

B: AID enzyme

C: CD3 molecule

D: αβ receptor

E: CD8 molecule that recognizes MHC 1 molecule on the antigen presenting cell

Correct answer: B. **(Controversial question)**

What you need to understand from this question is that T cells mature in the thymus, and for maturation to be successful they MUST first express surface markers *to look like a T cell*, so CD8+ T cells need to have RAGs (Recombination activating genes) to do a proper TCR, and this TCR will be αβ receptor "because non-traditional T cells that have γδ TCR don't have CD8 molecule", they need CD3 molecule to transmit signals to cell interior, they need CD8 molecule as a co-receptor to recognize MHC 1 molecule on cells. But they NEVER need AID (Activation-induced deaminase) enzyme that exists in B cells because T cells cannot undergo class switching or hypermutation.

Q15: In Asthma, which of the following T cells are activated?

A: Th17

B: Th2

C: Th1

D: Th0

E: A+C

Correct answer: B.

Asthma is an allergic reaction of type 1 hypersensitivity reactions, so it would be typical to find Th2 cells releasing IL-4, IL-5 and IL-13 that all serve to give the typical picture of allergy.

Q16: In case there is candida infection, we will find which of the following?

A: Th2

B: Th0

C: Th1

D: Th17

E: All of the Above

Correct answer: D.

Candida is a type of yeast which is a type of fungi, so it would be typical to find Th17 releasing IL-17, IL-21 and IL-22 that work together in fungal infections.

Q17: Th17 is expected to be found in:

A: fungal infections

B: parasitic infections

C: allergy hypersensitivity reactions

D: bacterial infections

E: viral infections

Correct answer: A.

In B and C we expect Th2, while in D and E we would expect Th1.

Q18: Which of the following Cytokines has an Autocrine effect on lymphocytes?

A: IL-1

B: IL-13

C: IL-2

D: A + C

E: IL-22

Correct answer: C.

It's well-known that IL-2 is the most important cytokine for T cells, specially activated ones, because by activation they express receptors for that cytokine on the surface to stimulate their own proliferation in an autocrine fashion.

Q19: All of the following statements are correct EXCEPT?

A: IL-22 is produced by Th17 in cases of fungal infections

B: delta-Gamma TCRs have more diversity than Alpha-Beta

C: IL-4 influences B cells to switch class to produce IgE Antibodies

D: TNF is a factor produced by Th1 in cases of bacterial or viral infections to activate macrophages and natural killer cells.

E: All the above statements are correct

Correct answer: B.

γδ receptors are for Non-traditional T cells that exist in specific places, and in each place, these cells tend to favor certain gene segments during rearrangement, while αβ Receptors of traditional T cells are hugely diverse due to non-specific recombination thus the ability to recognize billions of antigens they'll face throughout life.

Q20: **ALL secondary lymphoid organs have in common:**

A: Afferent lymphatics

B: High endothelial venules

C: Peyer's patches

D: lymphoid follicles

E: Nothing is common!

Correct answer: D.

Afferent lymphatics don't exist in MALT; HEV exist in all Sec.Lymph. Organs EXCEPT the spleen; peyer's patches are type of mucosa associated lymphoid tissue that exists in the gut.

Q21: **Find the mismatched pair:**

A: Th2 --> Allergy and Parasitic infections

B: IgM --> 10 binding sites

C: Antibody-mediated cell-dependent cytotoxicity (ADCC) --> IgG

D: γδ TCR --> Non-Traditional T cells

E: T-cell independent activation --> Affinity maturation

Correct answer: E.

Affinity maturation "also called somatic hyper mutation" happens ONLY IF the B cell is activated through Th cells. IgG3 makes a bridge between Bacteria and NK cell to make Bacteria in closer approximation and make it easier to be killed!

Final Material Questions

Q22: Which of the following is not a mechanism for restraining كبح the immune response?

A: short life span of immune cells.

B: Activation induced cell death.

C: iT-reg cells.

D: INF-γ release.

E: Inhibiting the immune response.

Correct answer: D.

We took that INF-γ is a strong macrophage activator that's released from Th cells, and this contradicts our main aim in restraining the immune system. All the other choices represent the 4 major ways to restrain the immune system.

Q23: iTreg releases which of the following cytokines to make it difficult for dendritic cells to activate T cells?

A: INF-γ

B: IL-2

C: IL-13

D: IL-10

E: TGF β

Correct answer: D.

iTreg releases IL-10 and TGF β; TGF β decreases proliferation of T cells and the ability to T-killer cells to kill. INF-γ is released by Th1; IL-2 is the most important cytokine for T cells; IL-13 is released by Th2 to stimulate production of mucus.

Q24: **Wrong about Attenuated viral vaccine:**

A: Can't produce memory killer T-cells.

B: Can infect Antigen presenting cells.

C: Can spread from vaccinated person to others in a phenomenon called herd immunity.

D: Should NOT be given to immune-suppressed people.

E: Vaccines for measles, rubella, and mumps are famous examples.

Correct answer: A.

The first and clearest difference between killed and life attenuated vaccines is that Life.At.Vs can infect APCs thereby accomplishing the rule needed to produce memory killer T cells, while killed Vs can never have the infectious ability "hence: named killed" to infect APCs or any cell. Because life.At.Vs are infectious, they can cause serious infections to those who are immune compromised, that's why it should not be given to them.

Q25: **If you knocked out transcription factor (Aire) gene, what would you expect to happen?**

A: T-cells will be double positive

B: T-cells will be double negative

C: T-cells will have a BCR receptor

D: T cells will remain non-self

E: T-cells will target self-organs

Correct answer: E.

You must remember that AIRE is a transcription factor that exists in the medullary thymic epithelial cells in the thymus; these cells contribute in training T cells in the second stage of their development and maturation in the thymus by displaying self-antigens to delete self-reactive T cells. One of the strategies it uses is AIRE transcription factor which up regulates expression of tissue specific antigens like heart or kidney Ag that don't exist elsewhere. Without this strategy, many T cells will target these organs because they didn't get introduced to them in the thymus. T cells enter the thymus Double –ve, and then in the cortex they form TCRs and both co-receptors to become D+ve. T cells never form BCRs because the receptors totally differ in structure.

Q26: **Wrong about nTreg:**

A: Expresses CD8 co-receptor

B: Originate in the thymus

C: nTreg is a T helper cell

D: It functions to eliminate self-reactive T-cells

E: It expresses a gene called FOXp3

Correct answer: A.

nTreg is a Th cell so it expresses CD4 co-receptor.

All other statements are true.

Q27: **Choose the correct statement regarding Central tolerance:**

A: Lack of opsonized self-antigens is a mechanism of B cells central-tolerance

B: Hyper mutation, which is rearrangement of BCRs, happens as a second chance for B cells to avoid being self-reactive.

C: T cells undergo negative selection then positive selection in the thymus.

D: Being a double positive T cell means that the cell has both TCR and CD3 molecule.

E: All statements are incorrect.

Correct answer: B. **(Controversial Question)**

A final exam question that looks like this question had this answer as one, so the question was rewritten for better understanding.

Lack of opsonized self-antigens is a mechanism of B cells peripheral-tolerance to preserve self-tolerance even if B cells had a hyper mutation in the periphery that resulted in recognizing self-antigens; T cells undergo positive then negative selection in the thymus during development; being a double positive T cell means that the cell has both CD4 and CD8 as co-receptors; this happens in early cortical stages of T cell development.

Q28: **Wrong about Amyloidosis:**

A: Amyloid is not a single chemical entity; different proteins can aggregate and form fibrils with the appearance of amyloid

B: Proteins' fibrils are continuous and non-branching.

C: Congo red stain differentiates amyloid from collagen and other substances.

D: Amyloidosis is diagnosed on morphologic grounds

E: It's an intracellular deposition of protein fibrils

Correct answer: E.

Amyloidosis is characterized by extracellular deposits of fibrillary proteins which are responsible for tissue damage and functional compromise.

Q29: **Wrong about Bence-Jones bodies:**

A: They're unpaired κ or λ light chains

B: They're released from malignant plasma cells in multiple myeloma.

C: They can be excreted in urine due to their small molecular size.

D: They're seen in a systemic type of Amyloidosis.

E: They're always related to amyloidosis.

Correct answer: E.

Bence-Jones proteins can cause amyloidosis but the majority of Myeloma patients who have Bence-Jones proteins in serum and urine do not develop amyloidosis. Primary Amyloidosis which these bodies are seen in is systemic.

Q30: In Rheumatoid arthritis, which type of Amyloidosis would complicate the disease?

A: AL Amyloidosis

B: Aβ Amyloidosis

C: AA Amyloidosis

D: β2-microglobulin Amyloidosis

E: All types can be found

Correct answer: C.

AL is seen in plasma cell disorders; Aβ is seen in Alzheimer disease; β2-microglobulin is found in Amyloidosis that complicates long-term hemodialysis.

Q31: The form of amyloid associated with chronic inflammation is?

A: Amyloid Associated

B: Transthyretin (TTR)

C: β2-microglobulin

D: Amyloid Light chain (AL)

E: Aβ (β-amyloid protein)

Correct answer: A.

AL is seen in plasma cell disorders; Aβ is seen in Alzheimer disease; β2-microglobulin is found in Amyloidosis that complicates long-term hemodialysis; and TTR is either deposited in Mutant form in familial amyloid polyneuropathy or in normal form in senile systemic amyloidosis (Amyloid of Aging).

Serum AA increased production in inflammatory states as acute phase response contributes to its deposition in chronic inflammation, so this type of Amyloidosis is often called secondary amyloidosis.

Q32: An endocrine Tumor which Amyloidosis can be seen in is:

A: Melanoma

B: medullary carcinoma of the thyroid

C: Osteosarcoma

D: Neurofibroma

E: None of the above

Correct answer: B.

In endocrine tumors like medullary carcinoma of the thyroid, as well as islet tumors of the pancreas, and also pheocromocytoma (a neuroendocrine tumor of the medulla of the adrenal glands), you can find microscopic deposits of localized amyloid.

Other options are all non-endocrine tumors "as simple as that".

Q33: Wrong about graft rejection:

A: Hyper acute graft rejection occurs within minutes to few hours after transplantation.

B: Hyper acute graft rejection occurs in presensitized hosts like those who previously rejected a transplant, or had multiple blood transfusions, and in multiparous “many pregnancies” women.

C: Ab-Mediated Chronic rejection occurs insidiously, without preceding acute rejection.

D: In acute rejection, we find a local accumulation of mononuclear cells (Lymphocytes and Macrophages).

E: Hyper Acute rejection induces cellular and Antibody mediated reactions.

Correct answer: E.

Since hyper acute rejection occurs in a very fast onset, it would be reasonable to say it only occurs through Antibodies, and not just any antibodies, they’re antibodies formed even BEFORE transplantation.

Q34: In cases of contact with Poison ivy (السماق السام), which of the Following Th cells do you expect to be activated?

A: Th17

B: Th2

C: Th1

D: B+C

E: Maybe none; I don’t know!

Correct answer: C.

This is a **very clever question**; it depends on your previous knowledge in released factors from Th cells and their effects that are related to hypersensitivity Rxns. How? Let’s break this puzzle into simple pieces!

Immune response directed against contact Ags like Poison Ivy is an example on Delayed type HSRxn (Type 4); Type 4 is characterized by T cell response driving an inflammatory reaction involving **macrophages**. Now is the great question; which type of T cells had the ability to release a cytokine that strongly stimulates macrophages??? Doesn’t this remind you now with a cytokine named INF-γ??

Don’t you just love when your brain rings the bell!?

Q35: All these features are related to Di- George syndrome EXCEPT?

A: Abnormal Facial features

B: Cardiac defects

C: Cleft palate

D: Deletion in chromosome 22, region q11

E: hypercalcemia

Correct answer: E.

It's all about CATCH 22!

Hypocalcemia is what caused Tetany which is the MOST COMMON clinical presentation in infants having Di-George syndrome.

Q36: X-linked SCID is associated with which genetic defect?

A: Btk deficiency in B cell progenitors.

B: abnormal gene coding for IL-7 receptor.

C: Accumulation of toxic adenosine metabolites.

D: Impaired differentiation of Lymphocytes to become IgA-producing cells.

E: Inherited defect in NADPH oxidase enzyme complex.

Correct answer: B.

A is seen in X-linked Bruton's Agammaglobulinemia

C is seen in SCID caused by AUTOSOMAL defect

D is seen in Selective IgA deficiency.

E is seen in Chronic Granulomatous Disease

A Question not mentioned in questions collecting: **In Immunodeficiency..?**

The answer was: Vitamin D deficiency results in macrophages dysfunction.

Another choice was: humoral immunity is the most affected! But **be careful** I fell into this when it was actually wrong; For PIDs, humoral defects are the most common, however, The Dr. was asking about 1ry and 2ry Immune deficiencies altogether, and in Malnutrition "which is the most common of 2ry ID", cellular arm (T cells) of the immune system is affected mostly "remember protein def. being most common and what it affects".

Q37: **Wrong about CGD:**

A: Caused by an inherited defect in NADPH oxidase enzyme complex

B: low IgG levels are found

C: Patients are particularly susceptible to fungal infections

D: Gold standard for CGD diagnosis is NBT assay.

E: All the statements are correct.

Correct answer: B.

CGD mainly affects phagocytes, so B cells are expected to be functioning normally!

This defect greatly lowers available Reactive species needed to kill the fungi; Neutrophils are hugely recruited in cases of fungal infections "remember Th17 and released cytokines", so if these cells were non-functional, well, with what army I'll fight??!

NBT depends on NADPH oxidase existence to change color and give positive sign, so a negative NBT is Diagnostic!

A Question of no certain answer: **SLE accurate confirmatory test?**

Some said it was **ANA** and some said it was **Anti-dsDNA** because it's specific which means it's only positive in SLE. ANA was said to be always positive in SLE, however, it can be positive in OTHER autoimmune diseases. If you came to me I'd agree on ANA because accuracy is needed, and false negativity in Anti-dsDNA "diagnosed as SLE while in fact he's not" happens in 40% of cases.

A Question of no certain answer: **HIV confirmatory test? "READ ONLY"**

It was HIV antibodies vs. viral antigens. The most accepted Answer is by far **viral antigens**; which is true since now the most reliable determinant for progression of HIV is viral load. So for diagnosis of HIV it's better to see HIV antigens load because if high they would greatly indicate disease existence. **NOTE: I didn't find a reference for this question!**

Immune-Pharma

Q38: **Drug of choice for RA patient with B- cell selectivity:**

A: Azathioprine

B: Rituxizumab

C: Mycophenolate

D: Methotrexate

E: Sirolimus

Correct answer: B.

Rituxizumab is an anti-CD20 drug; CD20 is a famous B-cell marker, so it would be really convincing to say that it's selective to B-cells, and thus it would be beneficial in rheumatoid arthritis involving B cells more than T cells.

Azathioprine, Mycophenolate and Methotrexate are antimetabolites and they're non-selective to either B cell or T cell; Sirolimus is an m-TOR inhibitor that serves to inhibit **T cells** proliferation.

Q39: Which of the following statements is Incorrect?

A: Glucocorticoids are used in idiopathic thrombocytopenic Purpura and in Rheumatoid Arthritis

B: Azathioprine is more selective than Mycophenolate

C: One of the side effects of glucocorticoids is Cataracts

D: Methotrexate is an example on anti-metabolites that work as false nucleotides to terminate DNA replication

E: Calcineurin inhibitors are more selective to the real cause of organ rejection which is T cells.

Correct answer: B.

For sure Azathioprine is less selective than Mycophenolate, because Mycophenolate is more selective towards T and B cells because it inhibits an enzyme specifically existing there, but azathioprine inhibits as well all other replicating cells so it's less selective. For sure since activated Calcineurin exists in T cells to bind the nuclear factor of activated T cells (NFAT) to stimulate production of IL-2, it would be very reasonable to predict that Calcineurin INHs will target the cells that Calcineurin exists in, which are T-cells!!

Q40: Drug of choice for patients with high risk of rejection is?

A: Basiliximab

B: Sirolimus (Rapamune©)

C: Muromonab

D: Azathioprine

E: Cyclosporine

Correct answer: A.

If we are highly expecting an acute rejection "high risk of rejection", IL-2 receptor INHs will serve us BEST before the transplantation; Basiliximab is an example on these monoclonal Antibodies (-mabs).

Muromonab is Anti-CD3 drug that's not used anymore in the market because it causes an immunity flare and this is exactly the OPPOSITE of what we want; Cyclosporine is used mostly in GVHD cases "when the organ transplanted attacks the recipient's body"; Sirolimus is used as an alternative to calcineurin INHs.

Q41: Drug of choice for transplantation patients with signs of rejection:

A: Cyclosporine

B: Methotrexate

C: Mycophenolate

D: Daclizumab

E: Sirolimus (Rapamune©)

Correct answer: C.

If AFTER transplantation and starting drug regimen rejection appeared, preventing this acute rejection would be better done by administering Mycophenolate (That's why if rejection appears, we change azathioprine to Mycophenolate).

NOTE HERE: The difference between this question and the previous one is that here we didn't expect any rejection and we started our normal therapy THEN rejection appeared, while in the previous Q. we did expect a high risk of rejection and we gave pre OP Basilixizumab. That's why here Daclizumab which is of the same family of Basilixizumab was a wrong answer. Methotrexate is a DMART used for RA.

Q42: Psychosis and personality changes are side effects of which drug?

A: Tacrolimus

B: Cyclosporine

C: Sirolimus (Rapamune©)

D: prednisolone

E: Infliximab

Correct answer: D.

Even if you thought that Cyclosporine is the answer, it's true that it causes mental confusion but a real side effect that is well-known for Glucocorticoids is psychosis, personality changes and depression which are really serious! This question just wants you to be reminded that prednisolone is one of this family "as well as cortisone and prednisone".

Q43: Reactivation of TB (Break of TB latency) is a side effect of which drug?

A: Anti TNF – alpha

B: Calcineurin inhibitors

C: Anti- CD3

D: m-TOR inhibitors

E: IL-2 Receptor inhibitors

Correct answer: A.

TNF-α serves in long-term controlling of progression of TB infection by activating Macrophages that form granulomas around TB and seize infections (A.K.A TB latency), so upon administering Anti-TNF-α, This job will have no one to do, so reactivation of TB will happen. All other options act on T cells mainly which are of less importance in TB infection.