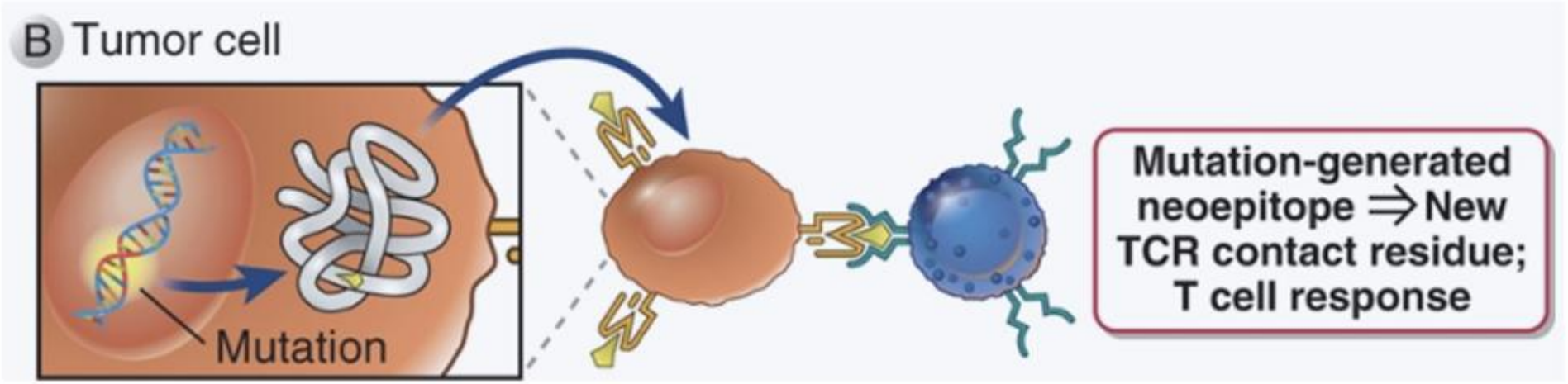
Which statement best describes neoantigens?

A. They are foreign viral proteins.
B. They are self-proteins that T cells are tolerized against.
C. They arise from mutations and are not tolerogenic to T cells.
D. They are found only in germ cells.

Answer & Explanation

Correct Answer: C. They arise from mutations and are not tolerogenic to T cells.

Neoantigens come from mutated self genes and are *not* part of central tolerance, so T cells can recognize them as foreign.



Which virus promotes cancer mainly through persistent inflammation?

A. HPV
B. EBV
C. Hepatitis C virus
D. CMV

Answer & Explanation

Correct Answer: C Hepatitis C virus

Hepatitis C virus promotes cancer not through viral oncogenes but through chronic, long-lasting liver inflammation, which leads to:

MAGE is an example of which antigen class?

A. Viral antigen
B. Neoantigen
C. Differentiation antigen
D. Cancer-testis antigen

Answer & Explanation

Correct answer: D Cancer testis antigens

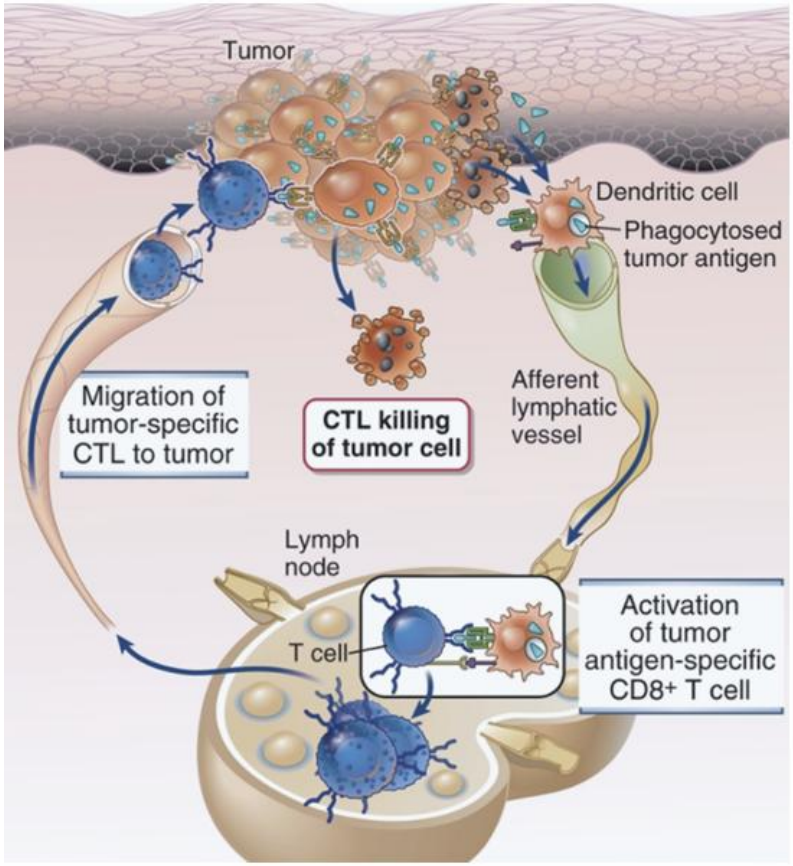
MAGE(melanoma associated antigen) belongs to cancer-testis antigens, expressed in germ cells but silent in normal tissues and re-expressed in tumors

The principal cellular defense against tumors is:

A. NK cells
B. Macrophages
C. B cells
D. CTLs

Answer & Explanation

Correct Answer: D CTLs
CTLs get activated in the lymph node, migrate to tumor site and recognize tumor antigens presented by MHC I and kill tumor cells directly.



Cross-presentation refers to:

A. APCs presenting peptides only to CD4 T cells
B. Tumor cells presenting via MHC II
C. APCs presenting exogenous antigens on MHC I
D. APCs presenting only viral antigens

Answer & Explanation

Correct Answer: C. APCs presenting exogenous antigens on MHC I

DCs take up tumor material and load it onto MHC I, allowing activation of naïve $CD8^+$ T cells even when tumors poorly present antigen.

What occurs in NK cell deficiency?

A. Autoimmunity
B. Increased susceptibility to tumor formation
C. Enhanced CTL activity
D. No major phenotype

Answer & Explanation

Correct Answer: B. Increased susceptibility to tumor formation

NK cells provide an essential layer of tumor surveillance by eliminating cells that have reduced MHC I expression or that display stress-induced ligands. When NK cells are deficient or dysfunctional, this surveillance is weakened, allowing transformed or abnormal cells to persist and expand.

Chronic inflammation promotes tumorigenesis primarily by:

A) Enhancing antigen presentation
B) Improving DNA repair
C) Increasing CTL numbers
D) Expanding NK cell populations
E) Driving ROS/RNI-mediated mutagenesis

Answer & Explanation

Correct Answer: E) Driving ROS/RNI-mediated mutagenesis

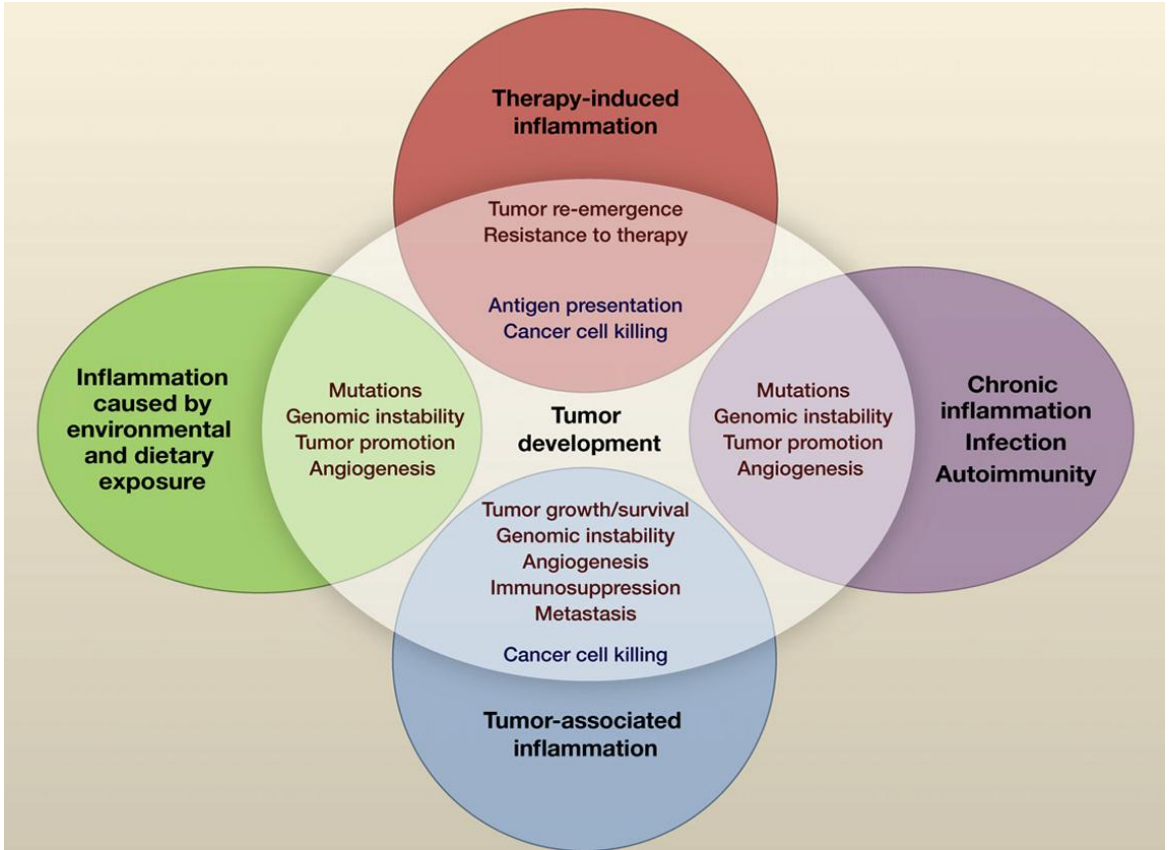
Chronic inflammation promotes genomic instability because activated immune cells generate large amounts of ROS and RNIs, which can directly damage DNA and cause mutations. In addition, inflammatory cytokines can stimulate bystander cells to produce their own ROS, creating indirect mutagenic pressure throughout the tissue.

Which is NOT a type of inflammation involved in tumorigenesis?

A. Therapy-induced
B. Chronic infection-induced
C. Autoimmune-induced
D. Allergy-induced

Answer & Explanation

Correct Answer: D allergy-induced



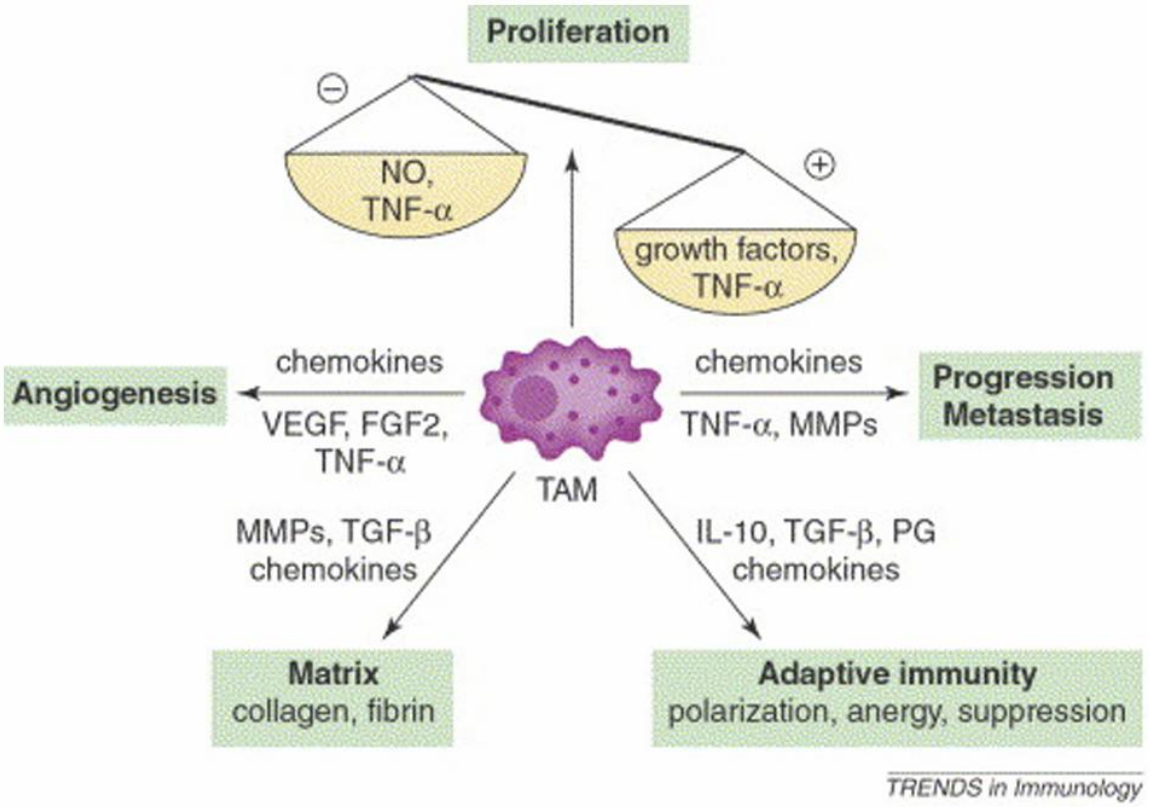
Tumor-associated macrophages (TAMs) are typically:

A) M1 phenotype
B) M2 phenotype
C) γδ lineage
D) NK-like macrophages
E) Plasma-like macrophages

Answer & Explanation

Correct Answer: B
M2 phenotype

TAMs (often M2-like) promote angiogenesis, invasion, and immunosuppression.



Which T cell subset can *promote* tumor progression?

A. Th1
B. CTLs
C. Th2
D. NK-T cells

Answer & Explanation

Correct answer: C) Th2
Explanation: Th2 cytokines can support tumor growth and suppress effective immunity.

JAK/STAT inhibitors in cancer aim to:

A. Increase CTL activity
B. Block inflammatory pro-tumorigenic signaling
C. Improve NK recognition
D. Enhance Treg survival

Answer & Explanation

Correct Answer: B) Block inflammatory pro-tumorigenic signaling

The JAK/STAT pathway drives many pro-tumorigenic processes, including inflammatory cytokine production, survival signaling and proliferation. Inhibiting this pathway reduces the chronic inflammatory environment that supports tumor growth and progression.

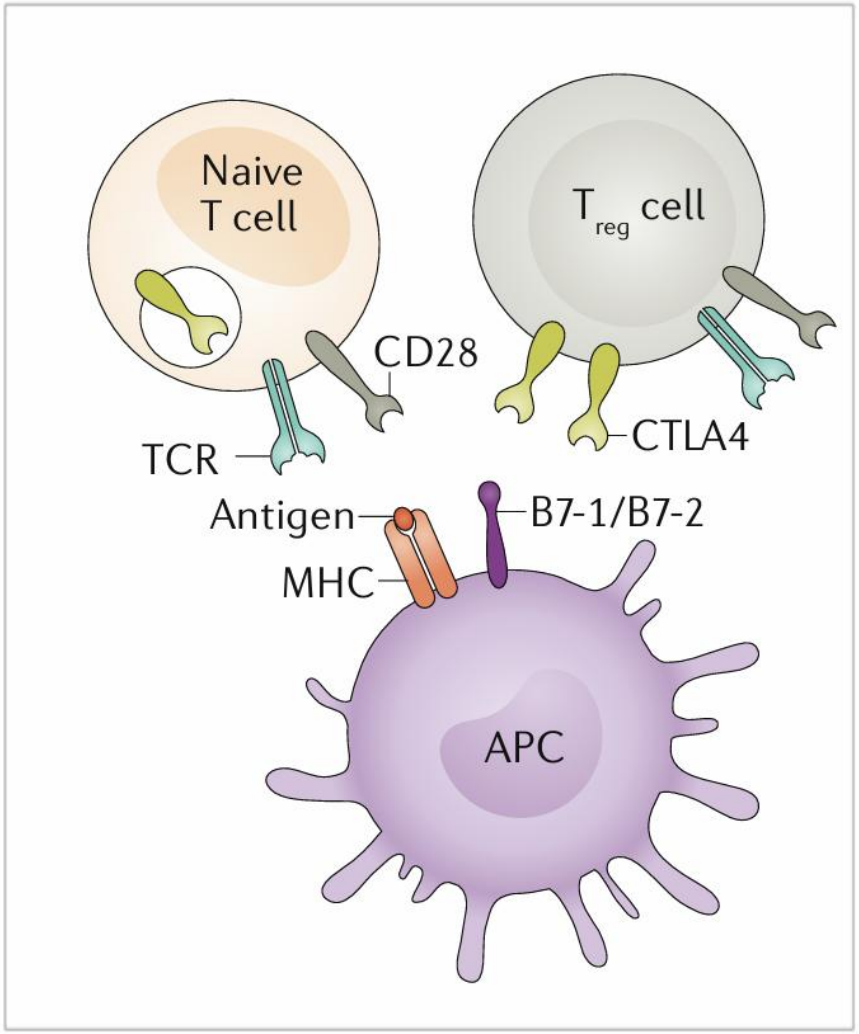
CTLA4 is structurally similar to:

A. PD-1
B. CD80
C. CD40L
D. CD28

Answer & Explanation

Correct answer: D) B28

CTLA4 and CD28 share strong extracellular homology and bind B7 ligands.
CTLA4 binds B7 molecules with higher affinity than CD28



CTLA4 knockout mice develop:

A. Severe autoimmunity
B. Immunodeficiency
C. Tumors
D. Mild inflammation

Answer & Explanation

Correct Answer: A. severe autoimmunity

Ctla4-knockout mice develop T cell-mediated lymphoproliferative autoimmune disease. because their T cells lack the inhibitory signals normally provided by CTLA4, leading to uncontrolled activation, excessive proliferation, and widespread immune attack on self tissues.

CTLA4 inhibits T cells by:

A. Activating NF-κB
B. Promoting AP-1 activation
C. Recruiting SHP2 and PP2A phosphatases
D. Enhancing CD28 signaling

Answer & Explanation

Correct Answer: C) Recruiting SHP2 and PP2A phosphatases
Explanation: CTLA4 recruits SHP2 and PP2A, blocking CD3ζ and ERK signaling → anergy

Anti-CTLA4 therapy works best when tumors have:

A. Low mutation burden
B. High neoantigen burden
C. No CTLs
D. High Treg infiltration
.

Answer & Explanation

Correct Answer: B more neoantigens

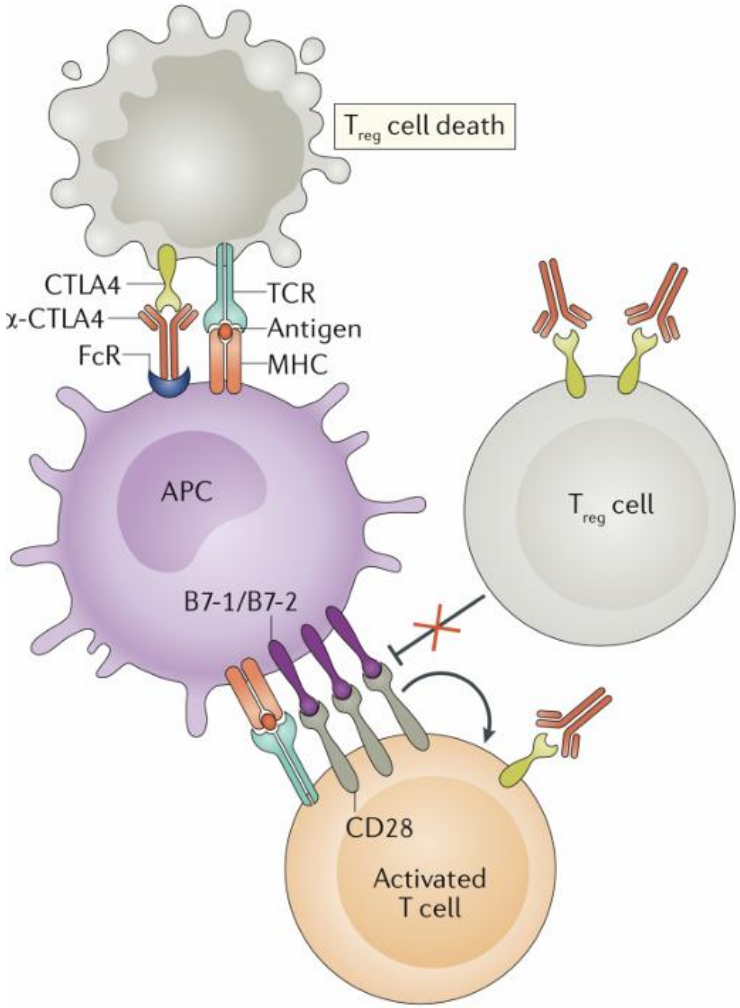
Explanation: More neoantigens → more T cell priming → stronger anti-CTLA4 benefit

Anti-CTLA4 therapy depletes intratumoral Tregs via:

A. Complement activation
B. ADCC
C. Apoptosis induction
D. PD-1 activation

Answer & Explanation

Correct Answer: B) ADCC
Explanation: Anti-CTLA4 antibodies bind CTLA4 on Tregs, coating these Tregs with IgG1. NK cells and macrophages have Fcγ receptors that recognize the Fc portion of those antibodies. So the antibody is on the Treg, not on the NK cell → the NK cell target the antibody-coated Treg and kills it through ADCC.

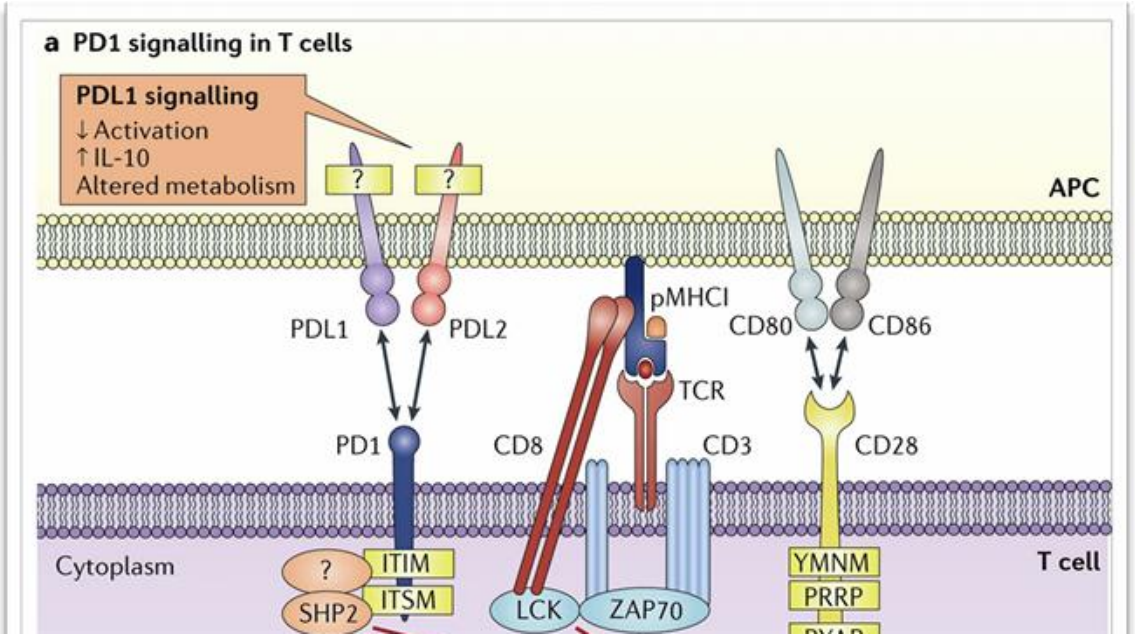


PD-1 is upregulated on T cells:

A) Before activation
B) Immediately after thymic maturation
C) on NK cells
D) Only during apoptosis
E) After TCR stimulation

Answer & Explanation

Correct Answer: E) after TCR stimulation
Explanation: PD-1 is a brake induced after TCR activation, so it's upregulated only once a T cell has been stimulated, helping prevent excessive or prolonged activation. For its activation it bind to PDL1/L2 on APCs.



Overexpression of PD-L1/2 on tumors results in:

A. Increased CTL killing
B. Tumor protection from CTLs
C. Increased NK cell killing
D. Reduced Treg recruitment

Answer & Explanation

Correct Answer: B) Tumor protection from CTLS
Explanation: PD-L1 on tumors drives T cell exhaustion and allows immune evasion

Compared to CTLA4 blockade, PD-1/PD-L1 blockade causes:

A. More autoimmunity
B. Less systemic autoimmunity
C. No T-cell activation
D. Only innate immune activation

Answer & Explanation

Answer: B) Less systemic autoimmunity

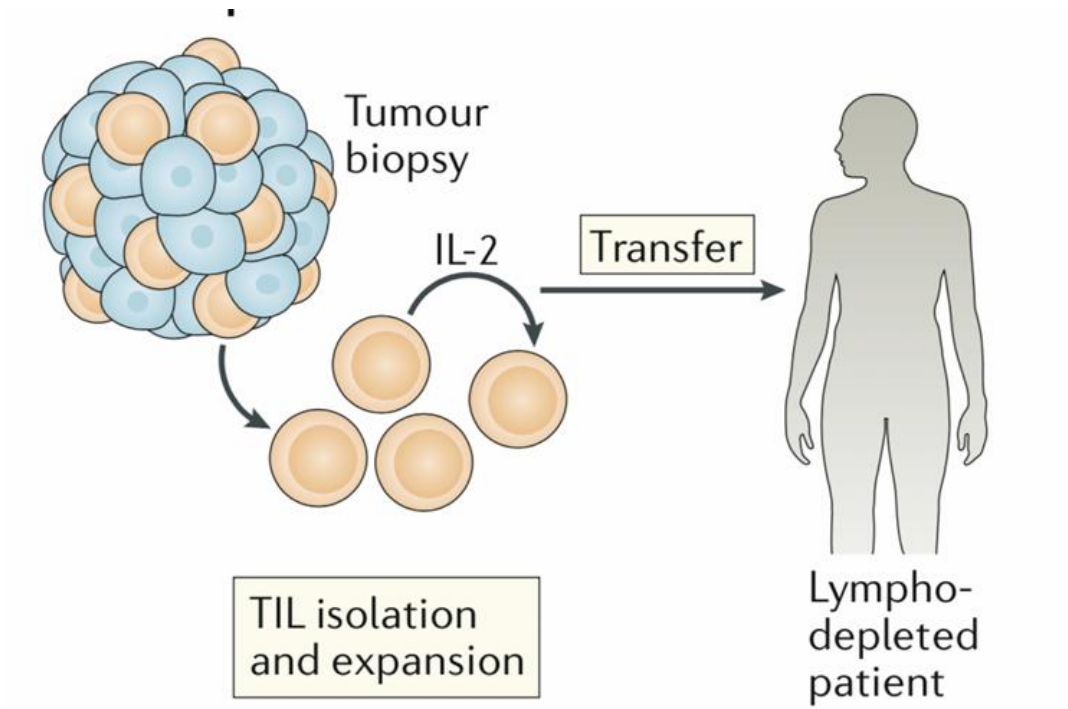
Explanation: PD-1 mainly restrains T cells in peripheral tissues, so blocking it disrupts local regulation rather than systemic activation, leading to less widespread autoimmunity than CTLA4 blockade

Adoptive T-cell therapy expands T cells in vitro using:

A. IL-4
B. IL-2
C. TNF
D. GM-CSF

Answer & Explanation

Correct Answer: C) IL-2
Explanation:IL-2 drives strong ex vivo expansion of tumor-reactive lymphocytes.



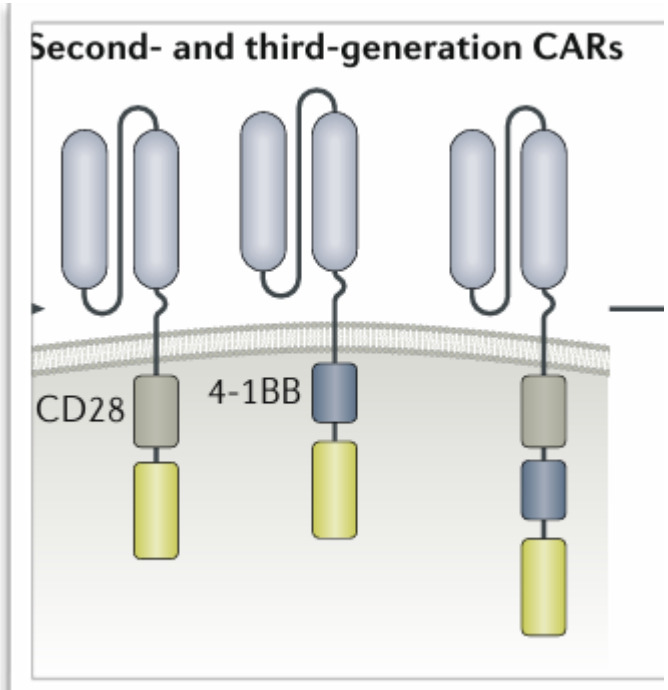
CAR T cells overcome TCR engineering limitations because:

A. They use MHC I only
B. They only target viral antigens .
C. They require tumor APCs
D. They recognize antigen independently of MHC

Answer & Explanation

Answer: D) They recognize antigen independently of MHC

Explanation: CARs bind surface antigens directly without MHC restriction, important since tumors often downregulate MHC I.



Major limitations of CAR T therapy include:

A. Lack of TCR expression
B. Inability to generate memory T cells
C. Poor penetration into solid tumors, exhaustion, CRS
D. Only works on CD4 T cells

Answer & Explanation

Answer: C.) Poor penetration into solid tumors, exhaustion, CRS

CAR T therapy works very well in blood cancers but faces several major limitations in solid tumors: poor T-cell infiltration, T-cell exhaustion in the suppressive tumor microenvironment, and severe side effects such as cytokine release syndrome (CRS) and off-target toxicity

Required components of a cancer vaccine include:

A. Antigen + adjuvant + formulation
B. Antigen + cytokines only
C. Adjuvant + checkpoint inhibitor
D. Tumor lysate without delivery system
E. Antigen + any immune cell population

Answer & Explanation

Answer: A Antigen + adjuvant + formulation

Explanation:
A functional cancer vaccine always requires three elements:
1.a tumor antigen,
2.a delivery/formulation system (e.g., RNA, vector, nanoparticles), and
3.an adjuvant to activate APCs.