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Personal Immunotherapy: A New Frontier in Cancer Treatment. Review

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Abstract: Cancer immunotherapy covers tests and approved treatments. It taps the body's defenses to fight tumors. Labs explore new methods and animal models. Clinics use drugs like immune checkpoint inhibitors (ICIs). This review covers how it works, and main methods. It notes patient uses and lab gains. Anticancer defenses rely on active dendritic cells, T cells priming, and immune surveillance. Tumors fight back with evasion tricks. Key methods include cancer vaccines, tumor-killing viruses, cell transfer, and ICIs. Nano-tools, cytokines, chemokines, and boosters sharpen these treatments. Mixes with radiation or chemo add power. They need care to cut side effects. New factors shape results. Tumor variety, gut microbes, gene shifts, and epi changes play roles. The review checks immune hides and drug blocks. Noncoding RNAs and epi shifts cause issues. New fixes target them. Biomarkers guide better outcomes. Problems stand out. Toxicity hurts. Solid tumors resist. Making drugs limits use. Future work eyes personal meds and smart delivery. These boost power and reach. The review builds knowledge for better, accessible treatments.

Keywords: Cancer immunotherapy, Tumor microenvironment remodeling, Immune evasion, Clinic and lab work, Biomarkers

I. Introduction

T cells drive three main cancer immunotherapy types now. They are: vaccines, cell transfers, and checkpoint blocks. These show strong clinical effectiveness and draw focus. The "Cancer-Immunity Cycle" explains antitumor mechanisms. Tumors work with tricks. Some pick cancer cells that block immunity. This is cancer immunoediting. Other paths block attacks too. Paths vary by person. Personal therapy fits needs. It finds each patient's blocks. It pushes the immunity cycle ahead. Cancer cells hold gene changes. These flag them for immune kill. Neoantigens are changed proteins only on cancer cells. The body spots them. New sequencing maps cancer genes. It finds tumor-only neoantigens. Therapies target block paths and these flags [1]. Tumor mixes hurt personal plans. Subgroups inside tumors differ in genes. Sequencing compares sites in one patient. Studies show gene and trait shifts in blood and solid cancers. Mutations and DNA copies vary. One biopsy misses the full picture. These risks failed care and return. It questions biomarkers too. Antigen loss under immune push adds trouble. Low HLA aids CAR-T over TCR-T cells. Tumor clone shifts need deep study across sites and time [2]. New work boosts CTL attacks on cancer. TIL transfers help melanoma patients. Trials show 22% full wins, 56% responses. Side effects hit like low white cells and leaks. Cancer vaccines target neoantigens or shared marks. They use peptides or DCs. A phase II test for esophagus cancer upped 5-year life. Best in low-CD8 or no-PD-L1 tumors. Personal neoantigen shots test in phase I. They work for melanoma and brain tumors. Patients grew T cell hits. PFS stretched, more with anti-PD-1. Low-mutation tumors like glioblastoma suit them. Some patients still worsen. Sides stay mild. Shots cause grade 1-2 skin flares, flu feels. Bad events hit under 10% [3].

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1.Tumor Type

Tumour type shapes immunotherapy success. Each cancer has unique biology and molecules that change how the immune system spots and fights it. The tumor's antigen set matters most. It differs by cancer type. Cancers like melanoma, lung cancer, and MSI-high tumors have many mutations. They make lots of neoantigens. This helps immune drugs, mainly ICIs, work well. Low-mutation tumors like pancreatic or some breast cancers make few neoantigens. They spark weak immune attacks on immunotherapy. The TME also decides outcomes. Melanoma and lung cancers have a "hot" TME. It shows heavy immune cell entry. This boosts ICI response. "Cold" TMEs in pancreatic cancer, glioblastoma, and ovarian cancer have few immune cells. They pack immunosuppressive cells like Tregs and MDSCs. This blocks immune drugs.

2. TME Heterogeneity

The TME forms a busy, shifting space. It holds tumor cells, immune cells, stromal cells, blood vessels, ECM parts, and fluids. Heterogeneity means shifts in cell mixes, signals, and immune presence. These occur across one tumor's zones or between main tumors and spread sites. Such changes harm immunotherapy results. Immune setups in tumors differ a lot. Some spots pack active T cells and DCs. They yield to ICB or other treatments. Other spots turn hostile. They raise MDSCs, Tregs, and TAMs. These curb key immune cells. Low oxygen zones add woes. They boost VEGF and HIF-1 alpha. Both worsen immune block. Varied TME spots make treatment hard. Immune-tough areas linger while others shrink.

3.Gut Microbiota

Gut bacteria now shape cancer immunotherapy success, above all ICIs for PD-1/PD-L1. Three studies lit this up in melanoma patients and others. Gopalakrishnan saw responders with rich gut diversity. They had more Ruminococcaceae and Faecalibacterium. Non-responders lacked variety. They had extra Bacteroidales . Matson spotted Bifidobacterium longum in winners. Ruminococcus obeum and Roseburia intestinalis filled losers. Responders showed high diversity too. Akkermansia muciniphila tied to good results. Antibiotics during treatment cut gains [4]. Gut mix and variety drive anti-PD-1 replies.

Gut bugs aid local gut immunity via PRRs and SCFAs. PRRs spot germ signs. They ripen DCs. These guide naive T cells to Tregs or Th17. Microbiome sets whole-body immunity. Good balance lifts immune work and shots. Imbalance breaks gut walls. It sparks body-wide swelling [5]. Gut flora tweaks replies to ACT, TLR boosters, and ICB. Drugs like ciprofloxacin or vancomycin scramble flora. This hits ACT via LPS, TLR4 paths, and type 1 DCs. Good bugs make SCFAs. They amp CD8+ T cells and CAR-T via HDAC block and IL-12 rise. CpG-ODNs need gram-negative bugs. They wake myeloid cells for TNF and IL-12. In ICB, Ruminococcaceae, Lachnospiraceae, and Bifidobacteriaceae link to win on anti-PD-1/PD-L1. Antibiotics wreck this. Study clashes call for strain-level digs on bug genes or paths. This could spark gut tweaks to lift immunotherapy [6].

Mouse and human melanoma work links T cell hits on Bacteroides thetaiotaomicron and Bacteroides fragilis to better CTLA-4 block. Stool genus clusters and 16S RNA sequencing made three groups. One led with Alloprevotella or Prevotella. Two held Bacteroides types [7]. FMT from donors to germ-free mice tested this. Transplants hit two weeks pre-tumour. Then came anti-CTLA-4 shots . Cluster C donors gave mice strong anti-cancer wins. Cluster B ones failed [8]

4.Neoantigens

Neoantigens are odd proteins that cancer cells make alone. They arise from mutations that alter the protein code. This sets them apart from normal cell proteins. They trigger strong immune reactions. They help spark immune attacks. TAAs show up in both healthy and cancer cells. Neoantigens mark only cancer cells. Spotting them needs a few steps. Take a tumour biopsy sample. Sequence it to find changed genes and proteins. Run computer models to pick likely immune targets. Use mass spectrometry to check immune effects. Neoantigen therapies stir good immune replies. They work best in high-mutation tumors like melanoma [9].

Fast cancer genome sequencing has sped up neoantigen hunts. It spots body mutations that birth these proteins. Each tumour has its own set from random DNA flaws or repair errors. Some high-mutation cancers like MSI-H types share neoantigens from set gene spots. Whole-exome or RNA sequencing of spread tumors finds targets. Neoantigen counts differ by cancer. Astrocytomas have few. Melanoma and lung cancers have many [10]. A study of 221 PDAC samples gave key facts. Most held usable neoantigens.

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Tumour T cells sat inside but stayed weak. Good antigen display signs linked to low killer T cell work. PDAC curbs T cell drive even with cancer-only targets [11].

Neoantigens aid cancer immune treatments like CAR-T cells and PD-1/PD-L1 blockers. CAR-T cells hit neoantigens to kill cancer but skip healthy spots. Claudin 6 fits. It sits in many solid tumors yet lacks in adult normal tissues. It makes a good aim. In blockers, mutation load predicts reply. High load means more neoantigens and better drug effects. NSCLC patients with high load live longer on PD-1/PD-L1 drugs. Still, some gain resistance. They lose old neoantigens or grow new ones that bind MHC tighter. This aids cancer escape. Neoantigens shift and shape treatment success [12].

PD-1 block boosts CD8 T cells against mutation neoantigens. Yet tumour surroundings can block this. Cell studies show weak TILs. Neoantigen TILs look like memory cells in tissue. They ignore IL-7 signals. In poor-reply tumors, they lack signal paths and gain brake receptors. Some mutations like TP53 hide inside cells. But their bits show on HLA. The p53R175H form works as a target. A H2 antibody grabs its HLA setup. It guides T cells to kill cancer in tests and animals. RAS cancer mutations get like treatment. Diabodies wake T cells to hit mutant RAS on HLA. This tap shared neoantigens from driver flaws for wide use. Better HLA-peptide hunts with pull-down and mass scans refine vaccines and shots. These steps push neoantigen vaccines, T cell tweaks, and aimed drugs past tumour mixes and escapes [13–17].

Each patient's neoantigen set, the neoantigenome, tests custom immune drugs. Hard part one: true ID and guess work. It needs deep gene and RNA scans to sort cancer changes from normal. This takes skill and power. Not all guesses spark immunity. Picking strong ones stays tough. Tumour mixes and shifting surrounds add woes. Neoantigens shift over time or by site. Custom vaccines cost much and take time. Immune tricks like weak display or suppressor cues cut gains. Mix therapies fight this.

5.Mutations

Cancer is known for evading the immune system. Tumors use checkpoint proteins like PD-1, PD-L1 and CTLA-4 to dodge T cell attacks. ICIs work by boosting T cell function. Their success depends on MHC molecules showing immunogenic neoantigens. High TMB links to better ICI results. More mutations raise chances of strong neoantigens. TMB has flaws as a sole biomarker. High-TMB tumors show about 45% response rates. Issues include neoantigen spread, tumour traits and host immunity. Immune actions involve T cell travel, cytokine levels and MHC-TCR links. Pair TMB with other markers to predict ICI response better [18].

PD-L1 levels and TMB both guide ICB response predictions on their own. PD-L1 offers some help as a marker. But uneven expression and test issues cut its value. TMB stands out as another option. High TMB ties to stronger results in ICB trials like Checkmate 026 and IMvigor211. Patients with high TMB and high PD-L1 gain most from ICIs. Yet TMB and PD-L1 do not match up. In combo therapy like anti-PD-1 with anti-CTLA-4, TMB matters more than PD-L1. It flags tumors where CTLA-4 curbs T cell start-up. These points show value in mixing TMB and PD-L1 for better patient picks in ICB. They also stress immune complexity and need for full biomarker plans [19].

Researchers built a tool to find tumour-specific neoantigens from SNVs in tumour DNA. It aims for use in cancer vaccines. Steps include DNA sequencing, SNV spotting and peptide picks that bind well to common HLA class I types in groups like Costa Rica's Central Valley. One case found 28 non-silent SNVs in 17 genes on chromosome one. This made 23 strong-binding peptides for HLA class I. This marks the first computer-based look at a vaccine from HLA-matched DNA data. Human cancers hold many somatic changes. Some show on MHC class I as foreign to the body. A simple pipeline uses exome sequencing, RNA checks and mass spec to spot and test neoantigens. It skips slow old methods for mutant peptides. Tests in mouse tumors proved it. Predicted peptides sparked T cell fights. Dextramers track T cell action directly. This aids custom vaccines and live checks of their effects. The method succeeds in neoantigen hunts and custom immunotherapy [20, 21].

Engineers made a fusion protein: scFv_RD_IL-15. It pairs an antibody with cytokine. An IL-15Rα piece targets IL-15 to tumors. This cuts body-wide harm and boosts tumor kill in mouse lung spread models. SGN-00101 (HspE7) blends heat shock protein 65 with HPV 16 E7 for cervical pre-cancer grade III. In 64 women, it showed promise. 22.5% had full pathology fix. 55% saw partial gains. Still, it's not clear if vaccine or natural fade caused this. It crossed reacts with other HPV types. Local swelling tied to immune strength. These ideas offer hope. More work must lift results and find best patient groups [22, 23].

A study checked SNVs in PD-1 path genes for melanoma patients on PD-1 drugs. It eyed prediction and survival links. PD-L1 +8293 C/A cut irAE risk vs C/C. PD1.5 T allele showed fewer irAEs too. No tie to drug response. PD1.7 C/C boosted overall survival. PD1.5 and PD-L1 +8293 may predict irAEs. PD1.7 hints at survival odds. More tests must back this up [24].

Colorectal cancer grows by two main paths of gene faults. Most show chromosome issues. About 15% are MSI-H from MMR breaks. MSI-H types build insert-delete errors in microsatellites. They link to Lynch syndrome often. These tumours have good outlooks and spread less. They hold special antigens that stir big immune hits. This leads to escape tricks. This review covers new causes, immune draw and dodges in MSI-H colorectal cancers. It looks at HLA changes too. Escape helps local defense but may limit spread [25].

MSI CRCs show a high load of neoantigen mutations from faulty DNA MMR. This leads to strong immune cell influx and ways to dodge immunity. One study looked at high endothelial venules (HEVs). These are special blood vessels that help lymphocytes move into tissues. In MSI CRCs (n=48) and stable microsatellite CRCs (n=35), HEV counts were much higher in MSI tumors. This held true most in Lynch syndrome cases. CRCs tied to Lynch syndrome with B2M changes had the top HEV levels. This points to a link between drawing in lymphocytes and dodging immune attack. The results stress how lymphocyte movement aids anti-tumour immunity in MSI CRCs. They also point to tumour immunoediting effects [26].

Many factors inside and outside the tumour affect how well immunotherapy works. The tumour microenvironment (TME) sits at the center. It varies in immune cell entry and suppressive parts. Tumour types and gene subtypes set mutation load and neoantigen output. Cancers with high TMB, like melanoma and NSCLC, respond better to ICIs. Neoantigens come from body cell mutations. They serve as prime targets for T cells against tumors. The gut microbiome shapes whole-body immunity. It impacts results through germ variety and waste products. Changes in genes for antigen display, checkpoints, and growth paths drive immune escape and drug resistance. Tumor microenvironment variety and escape tricks, such as high PD-L1 or low MHC, cause mixed outcomes. Studies test drug mixes to hit these issues. This boosts success against resistance. The image shows how complex immunotherapy replies are. It calls for custom treatments.

6.Small Molecules and Synthetic Drugs

New studies show small-molecule blockers can change cancer immunotherapy. They hit tumor escape and resistance paths. One way tweaks the suppressive TME. Toosendanin shifts macrophages to fight glioblastoma tumors [27]. Other drugs curb tumor growth and lift current immunotherapy power, like MYC blockers [28]. Drugs hit checkpoints too. BAY 2416964 targets AhR [29]. Small blockers hit Tim-3 [30]. This ramp up T cell attacks on tumors. Nano-tools deliver blockers for calcium channels and CD47 together. This aids immunotherapy in lung cancer [31]. Blockers that break PD-1/PD-L1 links offer checkpoint control in NSCLC and melanoma [32].

These varied tactics show how flexible small-molecule drugs are. They fix gaps in current treatments and better results across cancers. Work on these drugs holds great promise for new immunotherapy boosts. For instance, a small-molecule trigger raises gasdermin D (GSDMD) in tumor cells. This sparks pyroptosis, a fiery cell death that aids immunity with low body-wide harm [33]. A small drug that wakes the P2X7 receptor boosts immune attacks and immunotherapy in lung cancer [34]. Small drugs that spur MHC-II make tumors easier for immunity to spot. They also shift tumor fuel use to help anti-tumor effects [35]. In multiple myeloma, HDAC and Akt blockers together curb growth and lift immunotherapy [36]. PTPN2 blockers make tough melanomas open to anti-PD-1 drugs [37]. A new blocker for hematopoietic progenitor kinase 1 boosts anti-tumor immunity [38]. Degraders for protein tyrosine phosphatases like PTP1B and TCPTP tweak T-cell work. This improves cancer immunotherapy results [39].

7.Clinical-Importance

Small-molecule inhibitors hold promise. They boost anti-tumor immune responses. They help beat treatment resistance in various cancers. Many such immunomodulators exist. Several have advanced well in clinical trials. One review sort them by molecular targets [40].

Pairing these inhibitors with other treatments like ICIs or T-cell therapy creates synergy. It strengthens immune attacks and counters resistance [41]. Combo therapy hits multiple paths or checkpoints at once. This widens and lengthens anti-tumor responses for

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better results. Small-molecule inhibitors also pair with chemo or radiotherapy. Together, they strike tumors from many angles for stronger, lasting effects [42]. Smart combos draw on varied treatments. They push cancer immunotherapy forward. They tackle immune escape and tumor variety [43].
Targets of small molecules in cancer immunotherapy. Tumor cells sit amid immune cells like Tregs, Teffs, MDSCs, TAMs, and DCs. Researchers eye proteins and receptors on tumors and immune cells. Key one's tie to adaptive immunity: PD-1/PD-L1, RORγt, chemokine receptors, TGF-β. Innate immunity links: Sting, TLR. Tumor microenvironment involves IDO, arginase, A2A adenosine receptor. Top targets in trials include checkpoints like PD-L1 on tumors, PD-1 on T effector cells, CTLA4 on Tregs. Other immuno-oncology aims: IDO/TDO, arginases in cancer processes [44].

8.Antibodies

Antibodies drive cancer immunotherapy. They spark targeted immune attacks on tumors. PPAB001, a bispecific fusion, hits CD24 and CD47. It blocks CD47/SIRPα and CD24/Siglec-10. This boosts macrophage eating of cancer cells [45]. Antibody agonists target RPTPs like CD45. They deplete these phosphatases locally. RPTPs control immune signals, so antibodies may block their ties to receptors during activation [46]. Trispecific antibodies hit CD19, CD3, CD28. They link T cells to CD19-positive tumors. CD3 and CD28 binding amps T-cell action. It beats CD3-only antibodies thanks to CD28 co-stimulation [47]. IMM2902, another bispecific, targets CD47 and HER2. It cuts the "do not eat" signal via CD47 for macrophage phagocytosis. It also curbs HER2 signals like trastuzumab [48].NILK-2301 links CEACAM5 and CD3. It pulls T cells to CEACAM5 tumors. This sharpens recognition, cytokine release, and tumor kill [49]. Preclinical work backs antibodies in immunotherapy [50,51]. Some antibodies shine in clinics. Daratumumab (Darzalex) hits CD38 on myeloma and blood cancers. It fires up complement, NK cells, macrophage phagocytosis, and apoptosis. It clears tumors even near bone marrow stroma, which shields them. Low doses work with few side effects [52]. Sabatolimab blocks TIM-3 on worn-out T cells. A Phase Ib trial found it safe and effective with decitabine or azacytidine. It set best doses [53]. Ivonescimab, a bispecific, tests in Phase 1b for advanced NSCLC sans prior immunotherapy. It hits PD-1 and VEGF to boost immunity and cut tumor blood supply. Main goal: safety and tolerance. Secondary: early efficacy [54].

9.Peptides

Peptide-based nanoparticles known as PT-NPs break down PD-L1. This protein stops T cells from working. PT-NPs help T cells destroy tumors by clearing PD-L1 [55]. Peptide-led small-molecule groups build up in tumor cells. They boost chemo and immunotherapy results. The groups carry drugs and wake immune action in the tumor zone [56]. Peptide nanotubes carry the STING booster c-di-GMP. They aid melanoma treatment. These tubes send c-di-GMP into cancer cells. They start the STING path and spark strong anti-tumor immunity [57]. A changed superantigen joined to iRGD peptide targets tumors better. It draws in T cells. The pair sticks to cancer cells and fires up T cells. This pulls T cells to tumors and builds anti-tumor strength [58]. A new ring-shaped peptide hits LAG-3. This checkpoint curbs immunity. The peptide lifts CD8+ T cell attacks on tumors. It revives tired T cells and clears tumors by blocking LAG-3 [59]. A polymer mix joins a fixed cancer-killing peptide to an anti-PD-L1 peptide. It blends cell death from the killer peptide with PD-L1 block. In bowel cancer, it boosts immune fight through cell death signals and less PD-L1 block [60]. Peptide nano-boosters turn on the cGAS-STING path in cancer cells. They send STING drugs there and start strong innate immunity. This aids checkpoint block therapy [61]. A fresh ring peptide stops PD-1/PD-L1 links. It frees T cells to fight cancer [62]. Nano-groups blend a peptide neoantigen (Adpgk) and TLR9 booster (CpG ODN) for bowel cancer care. They send both to immune cells. This lifts antigen shows and strong anti-tumor reply [63].

10.Vaccines

Cancer vaccines play key roles in lab and clinic work. They train the body to spot and kill cancer cells. They create long-term immune memory. Teams spot TAAs and neoantigens to build them. Tumor immune clues help study DC and NK cell links. Platforms like peptide shots, RNA/DNA shots, and virus shells lift vaccine power. Boosters raise immune kick. TME studies fight blocks to vaccine work. In clinics, HPV and HBV shots cut cervical and liver cancer risk. Treatment shots hit TAAs to slow tumor growth or return. Sipuleucel-T treats prostate cancer. Neoantigen shots target melanoma and lung cancer. Pairing vaccines with checkpoint blocks, chemo, or radiation extends life. Gene and protein tools make custom shots for each tumor. Many trials test

vaccines across cancers like bile duct [64], melanoma [65], prostate [66], bowel [67], ovary [68], bladder [69], HPV-linked [70], and liver [71].

11.Clinical-Applications

A custom neoantigen DC shot treats lung cancer. It uses patient neoantigens to load DCs for injection. Side effects stayed mild. Response rate hit 25%. Control rate reached 75%. Median progress-free time was 5.5 months. Overall survival hit 7.9 months [72]. Small group size limits long-term views. A like vaccine for melanoma hits patient tumor marks. It sparks T cells with strong immune kick. Four of six patients stayed clear at 25 months. Two with return got anti-PD-1 and cleared tumors [73]. Custom neoantigen shots work well in melanoma. Pairing with anti-PD-1 helps. Long-term data needs more work. FixVac RNA shot BNT111 hits four melanoma TAAs. It treats advanced cases after checkpoint fail. It trains immunity to kill melanoma cells. Some saw lasting tumor shrink or loss. It works alone or with PD-1 blocks [74]. A phase I trial tested mRNA shots for gut cancer spread. They make neoantigens to fire T cells. All four patients took it well with T cell replies. No tumors shrank. Bigger trials with pairs are next [75].

HPV vaccines draw much focus for stopping cervical cancer and other HPV-linked cancers [76]. A Phase II study before surgery checked tecemotide (L-BLP25). This vaccine uses MUC1 to treat early-stage HER2-negative breast cancer. It sparks immunity against excess MUC1 in breast tumors. Patients got shots prior to surgery. This tested effects on tumor reply to standard care. The vaccine proved safe with no added toxicity. Yet it failed to raise full pathology response rates or lower leftover cancer load. Results point to hurdles in cancer vaccine creation. More tests on tecemotide in new settings or with other drugs seem vital [77].

Cancer vaccines hold promise but meet key barriers. Tumors vary widely across patients and inside single masses. Cancer cells show uneven antigen levels. This muddles vaccine plans. Tumours also dodge attacks. They cut antigen display and boost PD-L1 checkpoints. They form a suppressive TME that blocks immunity. Weak grasp of tumor antigens adds trouble. Few neoantigens trigger strong replies. Sound delivery to immune cells stays vital for better immunotherapy.

12.Gene Therapy

New cancer immunotherapy tools tweak gene output for clearer tumor immune views. Gene methods hit snags like off-target hits, poor drug flow, weak tumor reach, and RNase breakdown. Nanoparticles aid delivery here. Better nanoparticle use could lift cancer immunotherapy far.

One study used siRNA to build a method that blocks "self" signs and boosts "eat-me" signs on cancer cells. It cut CD47, a phagocytosis blocker, and raised calreticulin, a phagocytosis trigger. This sharpens immune spotting and tumor kill [78].Another looked at ultrasound-triggered carriers for siRNA and Fe_3O_4 nanoparticles. They retarget macrophages and curb M2 shift in NSCLC therapy. Carriers sent STAT3 siRNA to halt M2 drive plus Fe_3O_4 to spark IRF5 for M1 shift. This turns macrophages antitumor and aids NSCLC immunotherapy [79].A later study tested a nanodrug with PD-L1 siRNA and birinapant, a SMAC copy. It cut PD-L1 and sparked cell death in tumors. This boosts immunity and kills cancer cells at once [80].

One team tried lipid nanoparticles with siRNA to shift TAM roles in immunotherapy. These hit TAM polarisation genes. They push TAMs to antitumor work and strengthen anti-tumor immunity [81].A new siRNA tool crossed blood-brain and tumor barriers for glioma therapy. It reached glioma cells to block growth and evasion genes. This builds brain antitumor immunity [82].

Researchers built a DOX-linked polyphosphoester-siRNA nano assembly. It boosts macrophage and T cell replies against cancer. DOX sparks immunogenic cell death. siRNA blocks CD47 evasion. This lifts phagocytosis and T cell drive for stronger antitumor action [83].A PEI-EGFR-PD-L1-siRNA nano vaccine targets lung cancer. PEI carries EGFR and PD-L1 siRNAs to silence them. EGFR guides to tumors. This amps antitumor immunity [84].A twin siRNA nano adjuvant fires RIG-I/MDA5 paths and blocks CD47-SIRPα. It sends CD47 and LGP2 siRNAs. This curbs evasion and wakes innate immunity for full antitumor defense [85].Supramolecular carriers' pair TLR7/8 agonist with anti-CD47 siRNA for better therapy. They spark innate replies and cut "do not eat" via CD47. This grows innate and adaptive anti-tumor power [86].Ultrasound carriers with siRNA and Fe_3O_4 rework macrophage shift in lung cancer care. They hit STAT3 for M2 block and use Fe_3O_4 for M1 push. This reprograms macrophages antitumor and lifts replies [87].One study sent celastrol and PD-L1 siRNA to cell ER for immunogenic death and therapy gain. Nanoparticles carry celastrol for immune boost and siRNA for better tumor spot. This firms anti-tumor action [87].

CRISPR-Cas9 screens found COX2, KRAS-triggered, drives lung cancer therapy resistance. COX2 block could aid immunotherapy in KRAS-altered lung tumors [89]. One study applied in vivo CRISPR screening. It used a lentiviral vector to remove targeted antigens. This probed immune needs in renal cell carcinoma. It stressed the role of tumor antigens in immune spotting and killing [90].In vivo epigenetic CRISPR screening picked out Asf1a. This histone chaperone shows promise as an immunotherapy target in KRAS-mutant lung adenocarcinoma. It helps control immune replies in the tumor microenvironment [91].Another study mixed in vitro models of CD8+ T cell exhaustion with CRISPR screening. It checked the transcription factor BHLHE40's role in T cell fatigue. This offers ways to reverse it and boost immunotherapy results [92].Multi-angle in vivo CRISPR screening found Lgals2. This galectin family member serves as a key immunotherapy target in triple-negative breast cancer. Lgals2 blocks anti-tumor immune action. Blocking it may lift immunotherapy power [93].A full genome CRISPR screen on CD8+ T cell function spotted proline metabolism. It acts as a target to boost CAR-T cell therapy. Metabolic tweaks could raise CAR-T cell strength [94].Researchers built a flexible CRISPR-Cas13d system. It handles multiplex transcriptome control and metabolic tweaks in main human T cells. This allows fine gene tweaks. It aids custom immunotherapies [95].

13.Nanomedicine

Nanomedicine holds promise for targeted drug delivery in many diseases, such as cancer. Experts focus on cancer cells due to their fast growth and resistance to old treatments. Lipid nanoparticles wrap and send drugs to set cells or tissues [96]. Teams run many tests to adjust size, makeup, and surface charge. This ensures good results and safety. They also check toxicity and fit with body tissues for clinic use [97]. The goal is drug systems that max treatment power, cut side effects, and hit the right spots. Chemists, biologists, and doctors team up to grasp body links and refine lipid nanoparticles [98]. Chemists build lipid types for better strength and body fit. Biologists study cell uptake and inside paths. Doctors test effects and risks in animal models or trials. This gives tips to improve designs. Still, lipid nanoparticles face issues like long-term strength and build-up in some organs [99]. Body factors like immune reactions and clearance shape their in-body work and safety.

Many nanoparticle types treat cancer. They send chemo drugs straight to cancer cells. This raises power and cuts side effects [100]. They can spark hyperthermia to heat and kill cancer cells. Nanoparticles also sharpen imaging for better tumor finds and tracking. In all, they offer big gains for cancer care and patient health [101]. Full checks on these factors must come before human trials. Ways to boost strength and cut organ build-up are key for clinic shift [102].

14.Types of Nanoparticles used in Cancer Therapy

Nanoparticle drug systems have changed cancer care. They bring targeted delivery, better drug uptake, and fewer side effects. Many types exist. Each has unique traits for set cancer types. Zhao et al. list liposomes, polymeric nanoparticles, metallic nanoparticles, dendrimers, carbon nanotubes, and exosomes as main ones [103].

14.1 Liposomes

Liposomes are round vesicles of one or more phospholipid layers. They hold water-loving and fat-loving drugs. Cancer care uses them for body fit, drug holding power, and passive targeting via EPR effect. Teams tweak surfaces with PEG or ligands for longer blood time and active targeting [104].

14.2 Polymeric nanoparticles

Biodegradable polymers such as chitosan, PLGA, and PCL create these nanoparticles. They enable long-term drug release. Design allows control over the release. Surface tweaks support targeted delivery. They carry drugs, proteins, or nucleic acids. PLGA nanoparticles hold chemo drugs for slow release. Research shows less harm and better uptake. Paclitaxel in PLGA boosts action and lowers toxicity [104].

14.3 Metallic nanoparticles

Metallic nanoparticles Gold, silver, and iron oxide make metallic nanoparticles. They offer optical traits, magnetic pull, and simple surface changes. Gold nanoparticles aid cancer care. Biocompatibility fits PTT and imaging [103]. With heat agents, they warm

and kill cancer cells. They strengthen radiation too. Iron oxide helps magnetic hyperthermia and MRI contrast. This spots and treats tumors [104].

14.4 Dendrimers

Tree-like synthetic polymers with branches form dendrimers. They carry drug molecules. Clear structure, multiple sites, and inner spaces hold drugs, genes, or imaging agents. Exact size control and target ligands aid delivery. Dendrimer systems send doxorubicin to cancer cells. This cuts body-wide harm. They mix genes and drugs to hit cancer growth paths [103].

Carbon nanotubes (CNTs) Rolled graphene sheets shape carbon nanotubes (CNTs) into cylinders. Strong mechanical, electric, and heat traits suit PTT, drug delivery, and imaging. Target ligands guide chemo drugs to cancer cells. In PTT, CNTs grab near-infrared light. They turn it to heat that kills cells. They also help image tumors. CNTs mix drug carry with heat kill for full cancer care [103].

14.5 Exosomes

Cells shed exosomes as natural vesicles; 30 to 150 nm wide. They move proteins, lipids, and nucleic acids for cell signals. Low immune risk, body fit, and barrier pass draw them as drug carriers [105]. For cancer, they haul immune agents, siRNA, miRNA, or chemo drugs. Still early, their built-in aim promises custom tumor treatments [104].

14.6 Mesoporous silica nanoparticles (MSNs)

Mesoporous silica nanoparticles (MSNs) boast big pore space, vast surface, and set pore sizes. Pores trap drugs for release by heat or pH shifts. Surface ligands sharpen targeting [104]. Tests probe MSNs with doxorubicin, paclitaxel, or cisplatin. In cancer care, they trim side effects, lift power, and meter drug flow [105].

mRNA delivery Nanoparticles send mRNA to dendritic cells. This lifts antigen show. It ramps immune fight on tumors.

Chemoimmunotherapy boost Immune cells take up tumor RNA nanoparticles (RNA-NPs) with ease. This step raises chemo power and sparks a stronger immune attack.

Nanoparticle aims at tumor Nanoparticle makeup and shape pick the cancers they hit. One key method links targeting parts to the surface. These parts latch onto tumor cell receptors [106]. This cuts damage to healthy cells. It helps nanoparticles pile up in the tumor [107]. Some nanoparticles also open their drug load when they sense heat or pH shifts [108]. These smart triggers make targeting sharper. Drugs hit the right spot. This cuts side effects from random drug spread. It lifts cancer treatment success [109]. The approach eases patient load and betters' cancer care. Smart nanoparticles guide drugs straight to the tumor. They spare nearby healthy cells and tissues [110]. This hikes treatment power. It trims chemo side effects. Patients gain better life quality [111]. pH-sensitive nanoparticles spot the tumor's acid. They then dump drugs to kill cancer cells. Healthy tissues stay safe [112]. This boosts treatment wins and cuts patient woes. Yet if nanoparticles meet acid outside tumors, they might hurt healthy parts. That risks surprise damage and undoes side-effect cuts [113]. Tailored systems must spark only in tumor acid. New work tunes nanoparticles for higher pickiness to shield healthy tissues [114].

15. How nanoparticles hit cancer cells?

Nanotech aids targeted drug drops, cancer scans, and test tools [112]. Ligands or antibodies stick to extra receptors on cancer cells. Nanoparticles gather in tumors. They skip healthy cells. pH-smart nanoparticles sense tumor acid. They release drugs only there. This lifts treatment safety and strength [82]. Nano-test gear like biosensors and chip labs spots cancer signs in body fluids. It aids early finds and custom plans. Nano sensors catch tiny biomarker traces in blood. These tools could change cancer hunts and care. Patients get better odds [114]. Drug systems aim at cancer cells to max chemo punch and cut side hits. But some biomarkers show in many cancers. This risks wrong calls or false alarms [108]. Nano-drug tools need deep checks for new risks like poison or immune flares before wide use.

Targeted nanoparticle drug drops pH-polymer coated nanoparticles fix cancer therapy issues. They ship drugs right to tumors. This raises drug power and spares healthy tissues [115]. New systems beat old pH polymers. They sense small pH shifts for spot-on drug release. Nanogels offer one smart path. These are linked polymer nets that hold drugs. They swell and release at set pH [116].

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Teams test other triggers too, like heat or enzymes. This sharpens drug pick. It could shift cancer care and patient wins. pH nanogels pack chemo drugs for IV shots. They hit cancer cells hard but spare healthy ones. This cuts side effects and lifts treatment bite [117]. Cancer cells may fight back with drug walls. Combo therapy with mixed drugs beats this. It breaks walls and boosts chemo [118]. Teams eye gene fixes and immune boosts too. These amp immune work or tweak cancer genes for drug fits. Nano progress hunts better drug ships that dodge walls and raise wins [119].

Cancer nano-drug drop hurdles Immunotherapies change cancer care. They wake the immune system to kill cancer cells. More study must check breast cancer risk in women [120]. Teams track long-term effects on risk factors. This shapes safer plans. Do these treatments raise breast cancer odds? Clear answers let doctors pick best care. It aids risk cuts and better results [121].

A full study would check how one immunotherapy drug changes breast cancer risk factors over time in patients. Al-Obaidi and Florence [122] note the trial would match patients to a control group without the drug. It would track their health for years and note shifts in risk factors. Patients on the drug who see risk factors drop, not rise, could prove a clear counterpoint. This might stem from drug response, lifestyle shifts, or gene differences. Researchers must weigh these factors before firm views on the drug's impact. More work should probe why some patients' risks fall after immunotherapy [123]. Examining personal drug reactions, gene types, and lifestyle changes helps grasp the drug's effects on risk factors. This lets researchers give sharper advice on using immunotherapy for breast cancer [124].

The immune system clears or traps nanoparticles in wrong tissues. This hampers cancer nanodrug delivery. Tumors block nanodrugs with dense outer layers and odd blood vessels [125]. Targeted systems add ligands to nanoparticle surfaces. These bind to tumor cell markers. The approach cuts side effects and boosts treatment power. Ligands like aptamers, peptides, and antibodies have been tested [126]. Antibody delivery uses lab-made antibodies that grab tumor cell tags. They link to nanoparticles with chemo drugs. This spares healthy tissue, aids patients, and hits cancer cells harder [127]. Yet it fails in mixed tumors where cells show uneven tag levels. Tough tumor settings also block antibody entry or nanoparticle reach.

16. Role of new body-wide treatments and therapy options for solid tumors

Spotting and treating cancer rank high in modern medicine. New ways aim to find and kill cancer with patient-matched plans. This starts precision medicine. Cancer theranostics needs early detection to build strong plans. New methods mix imaging, sensing, molecular tools, and omics for cell and tissue checks [128]. Sharp diagnosis aids lab and body tests. Real-time watch helps craft new therapies. Iravani and Varma [129] say MXenes suit nanodrugs and sensors. These 2D materials flex with physical and structure traits. High surface area, tunable electric traits, and bio-match make them fit for health uses. Late gains use MXenes alone or with nanoparticles to boost diagnosis and therapy [130]. MXenes shine in drug delivery and health roles due to small size and bio-match [131]. Types like Ti3C2, Nb2C, and Ti2C, linked to polymers, aid cancer theranostics.

17. Biosensors and their types

New diagnostics seek early signs of cancer markers in changed cells. They check amounts and types. Biosensors lead here. These systems pair sensors for markers with converters for readable data. Studies cover biosensors for scans, health checks, and treatment checks. Molecular markers reshape care. Detailed bio-data lets doctors tailor plans. This lifts standard treatment wins and survival [132]. Tech offers receptors like DNA, antibodies, enzymes, or cells from disease studies. They spot bugs, viruses, cancer, metabolism issues, or heart events [133]. Biosensors build from polymers with added receptors for proteins, DNA, signals, or hormones. Markers guide outlook, prevention, forecasts, and treatment replies. Converters turn signals to graphs or numbers. Binding shifts sample traits. Converters read these as electric, light, or weight changes. Types split by converter: electric, light, or mass [134]. Cancer checks grow with pathology tools like tissue stains or PCR for markers. No gold standard exists yet. Smart sensors at early stages could raise survival and cut costs for patients.

17.1 Optical biosensor

Optical biosensors aid clinic checks. They track reactions non-stop. They spot tagged or free targets and reaction products. Methods include absorption, glow, light-up, Raman, bend, and scatter checks [135].

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Recombinant antibodies or fragments on carbon nanotubes (CNTs) take special forms. They boost detection power and sensitivity in many optical biosensors. Designs match analyte traits and needed sensitivity [136]. Vertically aligned single-wall carbon nanotube (SWNT) forests aid electron flow. They link tubes to enzymes at tips. This setup detects human serum albumin via amperometric enzyme-linked methods. Sensitivity jumps high. Detection limit drops to 1 nM [137]. CNT-based immunosensors find alpha-fetoprotein, gonadotropin, interleukin, and other biomarkers [136].

17.2Immunobiosensor

New conventional and wearable immunosensors have appeared. Graphene-based ones show strong potential in biosensing. They spot microbes, chemicals, proteins, and biomarkers [108]. Wang's team made an impedimetric immunosensor. It uses freestanding graphene paper with gold nanoparticles [139]. Many CNT-based immunosensors target cancer biomarkers. SWNT forests with bound antibodies detect Interleukin-6 (IL-6). This cytokine controls inflammation and immunity. It marks several cancers. Detection proves ultra-sensitive, specific, and precise via electrochemistry [140]. SWNT sensor beats traditional ELISA. Its IL-6 limit sits at 0.5 pg/mL; 16 times lower. The approach works for other early cancer markers too. These include prostate-specific antigen (PSA), human epidermal growth factor receptor 2 (HER2), and IL-8.

17.3NucleicAcid-BasedBiosensors

Nucleic acid biosensors use DNA strands for natural target binding. Two single-strand DNA (ssDNA) chains form double-strand DNA (dsDNA) in a key reaction [141]. Sensors track and fix sequences that match target nucleic acids. Kang's group built a glucose oxidase/graphene/chitosan mix [142]. It stacks enzymes thick (1.12 × 10^29 mol/cm²). This gives top strength and enzyme movement for glucose detection. Biosensors show high response (37.93 μA/mM/cm²). Detection limit reaches 0.02 mM for exact glucose reads [143]. Graphene's high conductivity and surface area drive this. It aids chemical uptake and electron shifts from redox agents to electrode surfaces. Wu's team made a glucose sensor down to 0.6 μM [144]. A film of glucose oxidase, Pt, graphene sheets, and chitosan cuts ascorbic and uric acid interference. Graphene aids enzymatic electrochemistry for biomolecules like H_2O_2 and cholesterol. It pairs with other materials.

17.4WholeCell-BasedBiosensors

Cell-based biosensors use live cells as smart sensors. They check outer and inner cell conditions in varied states. Sensors draw from life forms like nucleic acids and membrane proteins [145]. Past decades focused on cell health, life span, purity, and device biocompatibility. Goals aid early checks for microbes and oral cancer. Guanine-rich DNA aptamers with 6-carboxyfluorescein (6-FAM) spot PSA in serum fast [146]. This chemiluminescence tool cuts prostate cancer diagnosis costs. KCHA10a aptamer with 11-mercaptoandecanoic acid (11-MUA) binds target cells well [147]. It shows fine sensitivity for colon cancer diagnosis. Advanced sensors catch cancer cells via markers and false negatives. They offer high accuracy and sensitivity for hidden or symptom-free cases [148].

17.5Nano-Polymers

Nanotech aids cancer theranostics in drug tweaks, delivery, and sensor boosts. Drugs link or trap in polymers without extra surface changes [149]. Synthetic and natural polymer carriers extend blood time, improve dosing, and block clearance [150]. Polymers release drugs via pH or heat breakdown in body shifts. Dendrimer surfaces hold groups like amides, acetyls, and methyls. They allow more changes [151]. Sub nanometer gaps trap drugs or imaging agents. PEG-coated dendrimer-gold nanoparticles boost CT imaging biocompatibility [152]. Yet its strict controls and high production costs might limit broad adoption.

I. Conclusion

Outlooks, and future hurdles Cancer immunotherapy brings fresh options for cancer care. Still, like other treatments, it meets key barriers. These must be fixed to lift its power and reach against tumors. Main issues boil down to a few points. Top of the list, it struggles to hit and curb solid tumors. Tools like ACAR-T cell therapy fail to enter them. They clash with the suppressive tumor microenvironment. Side effects hit hard too. Think CRS plus other immune harms. These grow tricky in mixed therapies. Mass-making and selling custom options like CAR-T stays tough. Cancer cells dodge attacks via tricks such as antigen drop, extra checkpoints, or changed escape routes. Biomarker work has come far. But room grows for sure guides to pick therapies and aid patients. Research must face tumor variety too. This blocks clinic rollout. Bright paths lie ahead for progress. Sharpen personal

plans. Match therapies to each patient's tumor type and immune state. Nano carriers stand out to boost immunotherapy. Yet weigh their power, lasting safety, and big-batch build. Gut microbes shape results. Shift their mix to lift responses. Swap cold tumors for hot ones to raise sensitivity. AI promises sharp forecasts and custom care. New hits on neoantigens and T cells aid tumor control. At last, better animal models mimic human tumor-immune ties. They sharpen early tests and fine-tune plans.

Abbreviations

HLA: Human leukocyte antigen

CAR-T: Chimeric Antigen Receptor T

TCR-T: T-cell therapy utilizes T-cell receptors

CTL: Cytotoxic T Lymphocyte

RAS: (rat sarcoma) proteins

TIL: tumor-infiltrating lymphocytes

DCs: Dendritic cells

PDL-1: Programmed Death-Ligand -1

MDCS: Myeloid-derived suppressor cells

PRRs: Pattern recognition receptors

FMT: Fecal Microbiota Transplantation

TLR: Toll-like receptors

SCF: Stem cell factor

Tregs: Regulatory T cells

TAMs: Tumor-associated macrophages

VEGF: Vascular Endothelial Growth Factor

HIF-1 alpha: Hypoxia inducible factor-1-alpha

Th17: type 17 immunity is an immune response against certain types of pathogens

LPS: are the major outer surface membrane

SCFAs: Short-chain fatty acids

HDACs: Histone deacetylases

CpG-ODNs: Cytosine-guanine oligodeoxynucleotides

CTLA-4 shots: are immune checkpoint inhibitors that boost the immune system by blocking the CTLA- 4 protein, which usually suppresses T-cell activity

FMT:Fecal microbiota transplantation

MSI-H: Microsatellite instability-high (MSI-H) tumors are highly immunogenic, characterized by a high tumor mutation burden (TMB) and dense immune cell infiltration

SNVs: Single Nucleotide Variants

NSCLC: Non-small cell lung cancer

MYC: acts as a "master regulator" of immune evasion, fostering immune-privileged environments in tumors

TAAs: Tumor-Associated Antigens

(PDAC): Pancreatic ductal adenocarcinoma

PTPN2: Protein Tyrosine Phosphatase Non-Receptor Type 2

IDO/TDO :IDO (Indoleamine 2,3-dioxygenase) and TDO (Tryptophan-2,3-dioxygenase)

(Teffs) :Effector T cells

CD47/SIRPα: Blocking the CD47/SIRPα interaction allows macrophages to "eat" the tumor cells

(RPTPs): are crucial regulators of immune cell activation, signaling, and function

STING: is an ER-anchored protein that acts as a sensor of cyclic GMP-AMP (cGAMP)

(AhR) :The Aryl Hydrocarbon Receptor

Pt-NPs: Platinum nanoparticles

LAG-3: Lymphocyte activation gene **3**

siRNA: short interfering RNA or silencing RNA

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