Review article

Re-engineering Natural Killer cells: A new frontier in next generation cancer therapy

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Abstract

Natural Killer (NK) cells, long overshadowed by other immune cell–based immunotherapies, are emerging as potent anticancer effectors with distinct advantages and therapeutic challenges. NK cells act rapidly on cancer cells without prior sensitization, and by balancing between inhibitory and activating signals, they eliminate transformed cells in circulation and participate in early metastasis control. But most often, the tumour cells intelligently overcome it by inhibiting NK Cell activities or by forming protective clusters. NK cells on the other hand, act as "field commanders," on solid tumours, attracting Conventional Dendritic Cells or cDC1 and cytotoxic T cells for tumour defence by releasing chemokines. But again, the tumour microenvironment imposes profound Tumour Growth Factor-Beta (TGF-β)-driven suppression and reprogramming of NK cells which make them disarmed or convert them to exhausted Type 1 Innate Lymphoid Cells (ILC1s) which by producing pro-inflammatory cytokines contribute to tumour progression. Therefore, the future immunotherapy aims to develop new immuno-therapeutic strategies to energize and boost NK cell activity in combination with widely acclaimed T cell based therapeutic methods. This is supposed to achieve through engineered NK cell engagers that combine tumour targeting with NK activation using cytokines and adoptive NK cell transfer. The immune regulatory and tumour defending potentials of NK cells make them ideal candidates to rewrite the immune strategies against cancer. The advances in NK-cell specific therapeutic frameworks in combination with T-cell–based strategies are very promising, empowering the body's built-in assassins to programmed soldiers of next generation cancer therapy.

Keywords: Anti-tumour therapy, immune defence, Natural Killer cells, NK cell adoptive transfer, NK Cell engagers, PD-1 therapy.

Introduction

If the immune system were considered as an army, T cells would be the elite commandos everyone talks about. They have already inspired the development of blockbuster cancer therapeutics and captured global attention. But there is another quieter force on the battlefield: Natural Killer (NK) cells – the body's fast-acting assassins. Instead of waiting for orders, these cells patrol, sense danger and, when activated, can destroy transformed cells in hours. So, the pertinent question is: "*Can we deliberately harness NK cells to fight against tumour?"* This review explores the mechanisms by which NK cells exert anti-tumour activity, primarily through immune surveillance and the direct killing of abnormal cells and clearance of Circulating Tumour Cells (CTCs) to prevent metastasis. It also describes tumour immune-evasion strategies, including the expression of inhibitory ligands and the formation of

protective cellular clusters, and discusses the subsequent revitalization of the tumour microenvironment through the recruitment of Conventional Dendritic Cells (cDC1s) and cytotoxic T cells mediated by chemokine release. In addition, the review examines the challenges posed by Tumour Growth Factor-Beta (TGF-β)-driven immunosuppression by tumour cells and the reprogramming of NK cells into non-cytotoxic Type 1 Innate Lymphoid Cell-like cells (ILC1s). Finally, it provides an overview of emerging combination immunotherapeutic strategies, such as NK cell engagers, cytokine support, adoptive NK cell transfer, and integration with T-cell based therapies, that aim to overcome these barriers. These concepts are summarized in Figure 1.

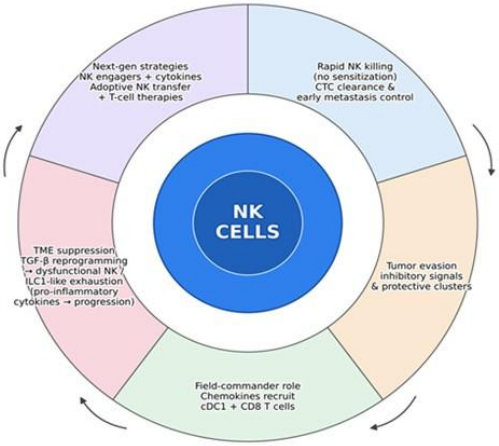


Figure 1: NK cells in cancer**:** Schematic presentation of NK-cell functions, barriers, and therapeutic opportunities in cancer

"Armed, but not dangerous until the alarm rings" - the NK cell mechanism

Natural killer cells act as circulating security guards in healthy adults. Equipped with cytotoxic granules and capable of releasing potent pro-inflammatory molecules, they can directly kill abnormal cells and recruit and activate other immune cells.[1] They selectively attack cells that display "kill-me" signals that appear when cells are infected, damaged, or transformed, under normal conditions; meanwhile never get triggered against normal cells as the healthy cells display "don't-kill-me" signals (like MHC-I) that bind to inhibitory receptors on NK cells, thus preventing their activation.[2] NK cells are kept in check by a powerful safety switch.[3] When tissues are damaged or infected, dendritic cells and macrophages release inflammatory cytokines such as Interleukins IL-2 and IL-18. Simultaneously, damaged or transformed cells start displaying stress ligands that activate NK cells by binding to NK cell activating receptors like NKG2D, NKp30 and NKp46. Flipping the action switches, the NK cell now changes into a fully armed effector, releasing cytotoxic granules that kill target cells. The immune Interferon -IFNγ released by T cells and NK cells at this stage further amplifies the immune response by rapidly initiating an immune effector cascade.[4-6] Even though this system is very much effective against infections, cancer is tricky and being an intelligent opponent, it designs strategies to escape the NK cell defence system and may even hijack it.

Metastatic cells-the lone travellers of cancer and NK cell encounters

The liver is a site of dominant NK cell activity and tumour cell elimination (Figure 2). It is found in a recent study that intravenously administered B16 metastatic melanoma cells are cleared by NK cells during their first pass through the liver.[7] This first-pass hepatic elimination of CTCs reduces the number of viable tumour cells reaching the lung, thereby lowering the pulmonary metastatic burden. Studies have also shown that when the injection route is modified to bypass the first hepatic passage, substantially more tumour cells reach the lung, resulting in a marked increase in lung metastasis. These studies underscore the critical importance of NK cell-mediated CTC clearance in liver in suppressing metastatic spread. The crucial role, played by the NK cells in preventing cancer from spreading, stems from their powerful and targeted spotting and killing of the 'CTCs'

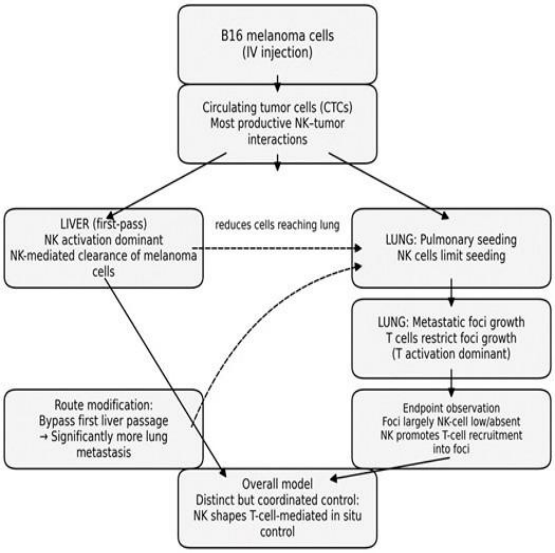


Figure 2: Mechanism of NK cell–mediated tumour cell clearance in the liver and immune responses in the lung

or tiny 'transformed cell clusters' before these cells can colonize distant organs. Animal studies have also shown that NK cells are capable enough to eliminate pulmonary metastases, within the first 24 hours after tumour cells reach lung tissue; but intriguingly, by about one day later, the tumour cell defence triggered by the NK cells will be tapered, leading to tumour cell survival. This observation indicates that the effective window of NK cell action against transformed cells is extremely short.[7]

After dissemination, the lung represents the next critical site of metastatic control (Figure 2). Once tumour cell foci are established within lung tissue, T cells become the dominant effectors, restricting the growth of metastatic foci. Although NK cell numbers are very low within the pulmonary metastatic foci, NK cells continue to contribute indirectly to the control mechanism by promoting T-cell recruitment into them. Observations from experimental models support the existence of a coordinated immune response in which NK cells provide early systemic protection through CTC elimination in the liver, thereby reducing pulmonary seeding, while also shaping subsequent T-cell–mediated, *in situ* control of metastatic outgrowth in the lung.[7]

The malignant cells on the other hand, evolve alternate strategies to escape from NK cells, as it has been observed that some circulating tumour cells cluster together which may help shield some of the malignant cells from NK cell attack, thereby increasing the metastatic possibility to 20–100 times than that for single cells. Another defence strategy adopted by the tumour cells involves displaying molecules like HLA-E that convey "I'm safe, don't kill me" signal, activating inhibitory receptors on NK cells, such as the CD94/NKG2A pathway, allowing the target cell to bypass NK-cell–mediated attack.[8]

NK cells -lone assassins to field commanders of the immune army

In solid tumours, NK cells aren't just lone assassins, but are field commanders which produce distress flares, XCL1 and CCL3/4/5, the chemokines. This is a part of attracting other immune cells to tumour site and activating the effector cells. These signalling molecules attract cDC1, a special type of dendritic cells, which are key players in presenting tumour antigens to cytotoxic T lymphocytes or CD8+ T cells. But tumours produce high levels of prostaglandin E2 (PGE2) which enhance tumour cell proliferation and angiogenesis. They also promote invasion and inhibit apoptosis (programmed cell death). Further they supress the anti-tumour immune response by several mechanisms; one among them involves weakening NK cells, by reducing their chemokine production. This negatively influences the NK - DC1 - CD 8+T Cell (Cytotoxic T cell) axis making the tumours harder to get infiltrated by the immune cells.[9,10]

Researchers have designed and developed several drug combinations that indirectly "wake up" NK cells in tumours with poor immune infiltration. Examples include boosting NK cell and cDC1 infiltration into tumours, inhibiting tumour cell pathways like WEE1 and AKT, that regulate cell

growth, survival and division and/or increasing the expression of "danger" signals (NKG2D ligands), by which tumour cells become more susceptible to NK cells.[11] Combining the NK Cell reactivating strategy with anti-PD1 therapy (PD1 – Programmed Cell Death Protein 1), a successful therapy of reactivation of exhausted T cells has been developed which is found to be highly effective in animal models with highly resistant tumour growth.[12] In some breast cancer models, increasing NK cell activation by IL-15/ IL-12 conditioning (cytokines that activate NK cells) after surgery, reduces metastases to a significant level. It also helps to develop long-lasting CD8+ T-cell memory in the host. Similar promising results have also been reported in early clinical attempts that infused a patient's own activated NK cells after tumour removal.[13,14] To summarise these observations, activated NK cells don't just fight alone, but they also naturally help build long-lasting, T-cell–driven immune defences against tumours.

NK cell therapies - The challenges

Why can't we design multiple drugs, exploiting NK Cell potentials? The answer is, we have to face "big challenges" as NK cells being innate cells, unlike T cells, respond in different ways while applying 'therapeutic tricks'. This is because, NK cells depend on IL-15 and IL-2 for proliferation and survival. However, unlike T cells, they don't produce their own IL-15 and IL-2; instead depend on external cytokines. Consequently, in tumours, the lack of these cytokines may render the NK cells dysfunctional, exhausted, or at times cause them to be converted into a reprogrammed or phenotypically altered cell type. Hence, T-cell–based strategies engaging NK cells through bispecific antibodies may not function properly.[9,10,12]

NK cell engagers: Point- and- shoot guidance with extra fuel

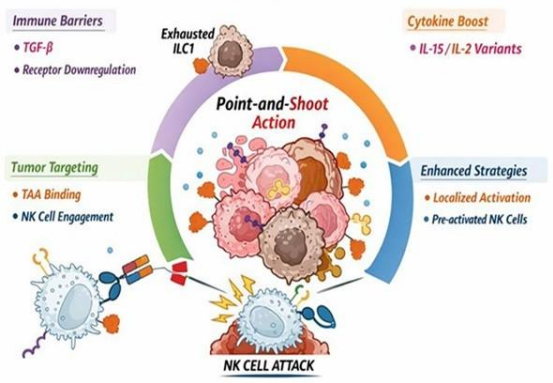
Considering all the above mentioned "challenges", new drugs are under trial, called NK cell engagers, to bridge the gap. These drugs are designed to bind a tumour antigen, and then to the NK cell through receptors like NKG2D, CD16, NKp46 or NKp30 simultaneously, forcing close contact equivalent to "point-and-shoot guidance". This is often assisted with a cytokine "boost", modified IL-2 or IL-15 variant, to give NK cells the extra power to fight tumour cells.[10]

The foremost challenge in cancer treatment is that NK-cell–activating receptors are often downregulated by factors like TGF-β or shed by tumour-associated enzymes. The receptors trigger NK-cell degranulation and tumour killing through cross-linking, but this often requires additional cytokines to function effectively within the tumour microenvironment. This limitation can be partly overcome by including IL-2 or IL-15 in the drug combination to promote NK-cell proliferation and sustain their activity; but this strategy is frequently hindered by cytokine-related toxicity. To address the issue, newer engagers are being developed with local delivery strategies or designs that become fully active only inside tumours, thereby reducing damage to the rest of the body. Another approach, which is largely in early trials and preclinical models, is combining these engagers with pre-activated donor NK cells to improve both efficacy and safety.[15-17]

Keeping the NK army fit: Metabolism, exhaustion and the TGF-β "minefield"

Most often, in the harsh tumour environments, NK cells lose the immune effector functions against tumours, may get re-programmed, losing their sharp killer edge, and become a less active, less aggressive tumour resident cell, ILC1s with upregulated inhibitory receptors like NKG2A, CD96 and LAG3. This is mainly caused by the presence of the high levels of TGF-β, a molecule that tumours use to create an immunosuppressive niche. In this situation, protecting the NK cells from "tumour programming" and keeping them

metabolically active will be a problem which can be addressed by providing IL-15 to help preserve NK-cell function and slow tumour growth. The kind of insights gained from strategies blocking TGF-β or combining its effects with PDL1 blockade have given promising outcomes in preclinical models.[18-20]



TGF- β=Tumour Growth Factor-Beta , TAA=Tumour-Associated Antigens, NK=Natural Killer

Figure 3: Schematic representation of strategies using NK cell engagers and cytokines to overcome tumour microenvironment barriers in NK cell therapy

The above-mentioned strategies using NK cell engagers and cytokines to overcome tumour microenvironment barriers in NK cell therapy are outlined in Figure 3. The mechanism of tumour targeting by NK cells and immune barriers, are depicted on the left side of the figure. Tumour cell targeting is achieved by cross-linking between NK-cell activating receptors (e.g., CD16/NKG2D/NKp46/NKp30) with tumour-associated antigens (TAA) on cancer cells that establish forced close contact, triggering NK-cell mediated tumour cell death. Similarly, the tumour microenvironment can limit NK cell efficiency and promote dysfunction through TGF-β–driven suppression and the downregulation or shedding of NK-activating receptors, ultimately leading to the conversion of active NK cells into exhausted ILC1s characterized by increased expression of inhibitory receptors (e.g., NKG2A, CD96, and LAG-3). The right side of the figure indicates methods to overcome these limitations and maintain NK-cell fitness and persistence. For this, engagers are incorporated or co-administered with cytokine support (modified IL-15/IL-2 variants) to enhance proliferation and sustain cytotoxic function, while acknowledging the challenge of systemic cytokine toxicity. Other enhanced strategies illustrated include localized or tumour-restricted activation (e.g., designs that become active primarily within tumours) and combination approaches using pre-activated donor NK cells, with the overall goal of improving efficacy and safety in solid tumours.

NK cells army vs cytokine fuel (Interleukins: IL-2, IL-10, IL-15 and IL-21)

In metastatic melanoma and kidney cancer, IL-2 was the first cytokine used for immunotherapy. It expands NK cells and T cells and produces effective responses in a small subset of patients. But in majority of cases high doses are required to achieve significant results, which may cause side effects. Another drawback of IL-2 is its ability to stimulate regulatory T cells (Tregs), which suppress anti-tumour immunity. Researchers later addressed this issue by engineering IL-2 variants fused to antibodies, such as anti-PD-1. This approach avoids Tregs and targets tumour-specific PD-1+ T cells and NK cells. [21] Another cytokine candidate is IL-10, which is produced by Tregs, NK cells and B cells, known for its role in dampening immune responses. It produces anti-inflammatory effects; in early trials high doses of IL-10 exhibit signs of anti-tumour activity; but its effects vary depending on context and the target cells it acts on. For this reason, the cytokine is considered as a ‘double-edged sword’ in cancer immunotherapy.[22,23]

IL-15 is considered the most promising cytokine candidate for NK cell mediated immune- therapy. It supports NK cell activation and effector functions without

triggering Tregs. Like other cytokines, systemic administration of IL-15, like IL-2, has dose-limiting toxicities.[24] To vanquish this, researchers have developed different methods that include conjugating it with antibodies like anti-PD-1 to target PD-1+ lymphocytes in tumours, designing pro-drugs which get activated only in the tumour microenvironment, and delivering it locally as exemplified by the FDA-approved intra-vesical delivery for the treatment of bladder cancer.[25,26]

Another strategic therapy is to inactivate a protein named CIS, encoded by the gene CISH. CIS restrains IL-15 receptor signalling, and its inactivation enhances NK-cell sensitivity to IL-15 and boosts their anti-tumour activity.[27,28] A second promising cytokine candidate is IL-21 which can redirect NK Cell metabolism, mainly *via* STAT3 rather than STAT5 (proteins belonging to Signal Transducer and Activator of Transcription family) and improve the cytotoxicity and cytokine production of NK cells, making them more efficient. It has been experimented that, NK cells engineered to produce their own IL-21 (such as anti-PD-1–IL-21 constructs) are stronger killers in pre-clinical models.[29-32] Alternative strategies to cytokine therapy, by exploring the "brakes" on cytokine signalling could induce stronger NK responses without flooding the body with cytokines and related issues.

The functional roles of NK-cell engagers in overcoming tumour microenvironment (TME) barriers are summarized in Figure 4. Tumour targeting is initiated through the binding of tumour antigens and engagement of NK-cell receptors—specifically CD16, NKG2D, NKp46, and NKp30—thereby enabling receptor cross-linking and triggering degranulation that results in direct tumour cell killing. To sustain NK-cell proliferation, metabolic fitness, and prolonged cytotoxic activity, 'extra fuel' is often integrated into these engagers *via* modified IL-2 or IL-15.

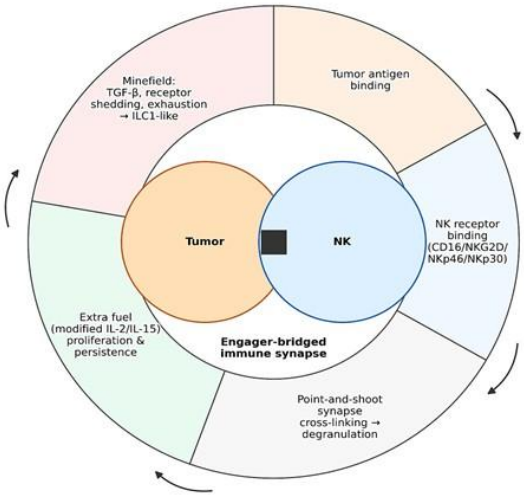


Figure 4: The mechanisms by which Natural Killer **(**NK) cell engagers overcome tumour microenvironment barriers

However, TME-imposed constraints, including TGF-β-mediated suppression, receptor downregulation or shedding, and ILC1-like reprogramming (functional exhaustion) of NK cells—collectively dampen NK-cell activity. These challenges necessitate strategies to restore NK-cell function and enhance antitumour efficacy, such as TGF-β blockade (with or without PD-L1 inhibition), localized or tumour-activated engager formats, and combination approaches including the use of pre-activated donor NK cells.

Conclusion

Our understanding of NK cell biology is currently enriched with functional genetic screens, single-cell RNA sequencing, proteomics, and detailed tumour models. New insights are emerging into how NK cells are regulated within the tumour microenvironment. Promising pathways and checkpoints have been established to enhance NK-cell anti-tumour activity while keeping them potent against malignant cells; yet safe for the patient's normal tissues. Even though T cells may be taking the 'centre stage' of immune-therapy, the NK cells are slowly 'stepping out' of the shadows. It is noteworthy that people with higher NK cell activity tend to have a lower incidence of cancer, and patients whose

tumours exhibit strong "NK cell signatures" (indications of NK cell infiltration and gene activity) have the chance of enjoying longer disease-free survival.[33] This is due to the fact that NK cells eliminate circulating 'transformed' or precancerous cells and prevent them from 'seeding' in new locations, thereby lowering the incidence of cancer and the risk of relapse. NK cells hold great promise as the next generation fighter/killer candidates of cancer immune-therapies, provided they are kept metabolically active and protected from the deceits of the tumour microenvironment.

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