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# A Review of Neuroblastoma Treatment with a Focus on CAR T-Cell Therapy

**By Sunny Ma**

## AUTHOR BIOGRAPHY

Sunny Ma is a sophomore at Groton School in Massachusetts, US. Intrigued about the diverse field of biology, she is especially passionate about the human brain and nervous system. She focuses on neuroblastoma, a solid tumor cancer arising in the sympathetic nervous system. In the future, she hopes to conduct research on the brain and various neurodegenerative diseases, such as Alzheimer's.

## ABSTRACT

Neuroblastoma (NB) is a deadly disease that afflicts at least 700 U.S. children every year, most of which are under five years old. Complex and difficult to detect, NB treatment presents a number of challenges. The immunosuppressive tumor microenvironment (TME), difficulty in trafficking to tumor sites, exhaustion of CAR T cells, and adverse effects such as Cytokine Release Syndrome (CRS) and on-target off-tumor toxicity are all major issues that scientists face when applying CAR T-cell therapy. With current limitations, CAR T therapies for NB need further exploration. For example, the abilities of different antigens need to be compared. Also, differences between immune cells such as cytotoxic T cells versus NKT cells should be explored. Additionally, relatively unexplored forms of treatment such as autophagy and YAP inhibition show promise. This review discusses the origin and background of neuroblastoma, a solid tumor cancer arising from the sympathetic nervous system. Treatment of this disease is still relatively novel and needs further development. This paper describes the work done in this field thus far and also discusses potential future treatment strategies. With future study, the foundation of cancer immunotherapy research that has been laid will undoubtedly lead to improvement in CAR T-cell therapy and NB treatment.

***Keywords:*** *Neuroblastoma, Solid Tumor, CAR T-cell Therapy, Immunotherapy, Childhood Cancer, Cytokine Release Syndrome, On-target Off-tumor Toxicity, Autophagy*

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## INTRODUCTION

Neuroblastoma (NB) is the most widespread extracranial pediatric solid tumor (Yu et al., 2022), characterized by a deadly occurrence in early childhood, particularly in children under five years old. Approximately 700 cases of NB are diagnosed each year in the US (Chung et al., 2021), and 45% of patients at diagnosis have high-risk NB (Heczey et al., 2023). The five-year event-free survival (EFS) rate of high-risk NB patients is only 50% (Yang et al., 2022). Furthermore, NB accounts for more than 15% of childhood cancer deaths, although it only makes up 8-10% of overall pediatric malignancies (Grote et al., 2021).

NB occurs in the peripheral sympathetic nervous system, deriving from undifferentiated neural crest progenitor cells and often originating in the adrenal glands (Khelifa et al. 2025). Typically, neural crest cells divide and then migrate from the dorsal neural tube to their embryonic sites, where they differentiate into their final form of sympathetic nervous system tissue (Mora et al., 2021). However, cells that fail to complete differentiation are unable to finish developing into normal sympathetic neurons and can form NB tumors. Undifferentiated neuroblastic cells do not show distinguishable neuropil or cytoplasm, and are small in shape (Mora et al., 2021).

NB is associated with high clinical heterogeneity and recurrent metastasis. NB also expresses low levels of neoantigens and MHC-I protein, which assist the immune system in tumor detection; as a result, NB shows poor immunogenicity. Tumor size, rapid proliferation, and an advanced immunosuppressive microenvironment further contribute to difficulty in effective NB treatment. Past clinical treatments of NB demonstrated unsatisfactory results, reinforcing the complex and extensive obstacles associated with neuroblastoma (Pilgrim et al., 2023). A study found that despite multimodal treatment, over half of patients with high-risk NB showed relapse and a reduced possibility for successful cure (Pilgrim et al., 2023).

A prominent characteristic of neuroblastoma is the high expression levels of the GD2 disialoganglioside, a tumor-associated antigen (TAA) and sialic acid-containing glycosphingolipid found on the surface of cells (Yang et al., 2022). GD2 is involved in both signal transduction and cell adhesion, and along with other gangliosides, it plays roles in tumor immune evasion and immune suppression within the TME (Yang et al., 2022). Tumor cells expressing abundant GD2 have been shown to display strong cytotoxicity (Mora et al., 2021). As a result, GD2 is acknowledged as a promising immunotarget for CAR T-cell therapy.

NB treatment has seen great development, but is still a novel field of study and requires further research. This paper reviews the characteristics of NB cancer: its origin, challenges of treating it, the current stage of advancement, and future perspectives, with a focus on CAR T-cell therapy. Despite NB's prominence as a deadly cancer, promising methods of treatments remain relatively new and must be more thoroughly explored, which further substantiates the objectives of this review article.

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## CHALLENGES TO CURRENT METHODS

Current NB treatment includes methods such as surgical removal, chemotherapy, and immunotherapy with monoclonal antibodies (mAbs) (Grote et al., 2021). However, as Grote et al. (2021) states, events such as disease relapse, drug resistance, and late effects remain prevalent challenges to successful treatment. According to Sun et al. (2023), about 50% of patients treated with multimodal therapy will experience relapse, despite initial remission. Additionally, due to lack of GD2 on tumor cell surfaces, patients do not always respond to mAb treatments (Pilgrim et al., 2023). According to Yankelevich et al. ( 2024), even after treatment of anti-GD2 mAbs, 10%-20% of high-risk NB patients do not see improvement; following stem cell transplants, 50%-60% see complete relapse. As a result, the need for more advanced and effective methods of treatment is prominent.

CAR T-cell therapy is a method of solid tumor treatment that shows great promise. In this mode of treatment, T lymphocytes are taken from the bodies of patients and genetically modified to recognize antigens on the surface of tumor cells. For example, the GD2 antigen is such an antigen that is overexpressed in NB, making it a promising target for CAR T-cell therapy. However, as Tian et al. (2022) has found, there are major obstacles to the efficacy of CAR T-cell therapy: heterogeneous expression of TAAs on tumor cells, identification of tumor-specific antigens, difficulty in trafficking to tumor lesions, lack of CAR T-cell endurance, and survival of the immunosuppressive tumor microenvironment (TME). Small tumors secrete chemokines that T cells often lack the right receptors for, thus preventing infiltration into tumor lesions (Tang et al., 2023). It is also noted that only one or few distinct antigens were used for current CAR T therapy (indicating that the identification of more tumor-specific antigens are warranted in future studies). However, tumor cells often express a variety of different antigens, which poses a challenge to the efficacy of CAR T cells. (Tian et al., 2022). In order for CAR T-cell therapy to be effective, chimeric antigen receptors (CARs) must be capable of high cytotoxic activity, CAR T cell expansion, tumor reduction, long-term persistence, and endurance when exposed to target antigens (Tian et al., 2022).

CAR T-cell therapy can also result in detrimental adverse effects. The two major challenges are on-target off-tumor toxicity and Cytokine Release Syndrome (CRS). Tumor-specific antigens are rare; healthy tissue cells often also express many of the antigens found on tumor cells (Grote et al., 2021). As a result, non-tumor tissue may be unintentionally attacked by CAR T cells, potentially leading to on-target off-tumor toxicity and damage to healthy cells. This occurrence is often seen as a result of targeting GD2, due to the expression of GD2 on regular mature tissues such as those of the nervous system (Pascual-Pasto et al., 2024). CRS, the second major adverse effect of CAR T-cell therapy, exhibits an excessive release of inflammatory cytokines, which can damage tissues and organs (Hou et al., 2019). Fever, chills, headache, and nausea are examples of symptoms often observed with CRS (Yankelevich et al., 2024). Additionally, symptoms of neurotoxicity, such as fatigue and dizziness, have been reported (Hou et al., 2019).

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## CURRENT STAGE OF CAR T-CELL THERAPY AND OTHER METHODS OF TREATMENT

The current stage of immunotherapy treatment for NB is a growing field of different methods and perspectives. The complexity and struggles currently associated with implementing CAR T-cell therapy require extensive development to overcome. Numerous methods of improving this immunotherapy have been studied, including research of different immunotargets. Currently, a few promising antigens have been identified, including GD2, B7-H3 (CD267), and Glypican-2 (GPC2) (Pascual-Pasto, 2024; Moghimi et al., 2021; Heczey et al., 2023). In addition, utilizing Bispecific Innate T-cell Engagers (BiCEs) to simultaneously target two antigens and exploring the abilities of Natural Killer-92 (NK-92) and Natural Killer T cells (NKT cells) are examples of other methods that have been explored (Grote et al. (2021; Heczey et al., 2023). Furthermore, restricting autophagy and the yes-associated protein (YAP) are both approaches that demonstrate high promise for enhancing CAR T-cell therapy (Pilgrim et al. 2023).

Although GD2 is a commonly explored target antigen for CAR T-cell therapy, its disadvantages has led scientists to examine other possible immunotargets. Pascual-Pasto et al. (2024) determined GPC2, a cell-surface signaling receptor that enhances neuroblastoma tumor growth, to be a possible option. GPC2 is expressed on neuroblastoma tumor cells and not on crucial normal tissue, eliminating the risk of on-target off-tumor toxicity that targeting GD2 can induce. However, low GPC2 expression in NB can hinder antitumor activity, highlighting a drawback to the CAR (Sun et al., 2023). To explore the abilities of anti-GPC2 cells, Sun et al. (2023) conducted a study in which an anti-GPC2 single-chain variable fragment (scFV) CT3, a CD28 hinge, CD28 transmembrane, and 4-1BB co-stimulatory domain were fused together to create the CAR T-cell construct CT3.28H.BBζ. Sun et al. (2023) found that CT3.28H.BBζ demonstrated the greatest preclinical effect against NB when compared with alternative CAR backbones, such as K666.28H.BBζ CAR T, which targets GD2. Thus, Sun et al. (2023) determined CT3.28H.BBζ to be a promising GPC2-targeted CAR for future clinical treatments. Pascual-Pasto et al. (2024) similarly investigated the potential of CAR T-cells targeting GPC2. Through a dual-targeting approach, they engineered T-cells to target GPC2 through a GPC2.CAR while also targeting GD2 via a BiCE. The BiCE simultaneously binds to tumor cells and innate immune cells, enabling NK-cells or macrophages within the TME to attack and kill the tumor cells (Pascual-Pasto et al., 2024). Tian et al. (2022) reinforces the work of Pascual-Pasto et. al in a study exploring GPC2 and CD276 as immunotherapeutic targets for NB. CD276, or B7-H3, is a checkpoint molecule that contributes to both metastasis and tumor immune evasion (Tian et al., 2022). Using P-COCC, Tian et al. (2022) identified two top CARs to be used in CAR T-cell therapy: CT3 among anti-GPC2 CARs and MGB7H3-LH among anti-CD276 CAs. They developed a bicistronic CAR (BiCisCAR) with these two constructs. This strategy of dual-targeting demonstrated both effective killing of tumor cells expressing GPC2 and CD276 and longer therapeutic activity compared to previous methods that targeted only one antigen.

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Other studies reinforce B7-H3 (CD276) as a favorable approach for cancer immunotherapy. Belonging to the immunoglobulin superfamily and B7 family, B7-H3 is expressed on many solid cancers, including neuroblastoma. Additionally, B7-H3 shows limited expression on healthy tissues and is crucial to both the adaptive and innate immune systems, making it a good target for tumor treatment. In a study conducted by Grote et al. (2021), second generation CD276-CAR cells demonstrated the ability to kill NB cells. Using the NK-92 cell line, which is associated with effective solid tumor resistance, CD276-CAR NK-92 cells were engineered. Grote et al. (2021) found that these CD276-CAR NK-92 showed proficiency in infiltrating and killing NB spheroids. Additionally, a novel approach using SynNotch design and targeting B7-H3 as a target antigen was found to be highly effective in killing NB cells (Moghimi et al., 2021). When GD2-B7H3 T cells were constructed, they were found to be superior to regular B7-H3 CAR T cells in regards to anti-tumor efficacy, metabolic fitness, and exhaustion level (Moghimi et al., 2021).

Another prominent approach to CAR T-cell therapy is the usage of NKT cells in the form of CAR-NKTs. NKTs differ from conventional T cells in that they express receptors for chemokines released by NB cells and demonstrate superior ability to traffic to tumor sites. Additionally, NKTs are capable of killing tumor-associated macrophages and enhancing NK and T cell anti-tumor responses. The anti-tumor abilities NKTs possess are not always shared by current T cells used in CAR T-cell therapy; this has led scientists to consider NKT cells as an alternative to conventional T cells. Heczey et al. (2023) found that for patients who received previous treatment but relapsed, CAR-NKT cells were able to expand after infusion and traffic to secondary tumor sites, causing regression of the tumor. Furthermore, Heczey et al. (2023) found that engineering regular GD2-CAR-NKT cells to also secrete IL-15 resulted in enhanced CAR T cell infiltration, anti-tumor ability, and persistence. IL-15, a cytokine involved in antitumor activity and function of NK and T cells, is crucial to fighting diseases such as various cancers. Hence, utilizing cytokines to enhance anti-tumor activity is a valuable strategy (Tian et al., 2025). Correspondingly, NKT cells show promise in designing CAR T cells.

Recently, autophagy has been another area of interest for advancing cancer immunotherapy. A self-degradative process involved in metabolic and genetic homeostasis, autophagy is key to stability within the TME and its immunosuppressive nature. According to De Mitri et al. (2025), autophagy contributes to activation, proliferation, and differentiation of the immune system, including NK cells, macrophages, and T lymphocytes. Autophagy is both a tumor suppressor and adaptive response: it degrades damaged organelles and thus cleans cells but also assists cancer cells in surviving diverse conditions. De Mitri et al. (2025) found that autophagy inhibition affects growth of NB spheroids, enhances MHC-I expression, increases immune cell infiltration of tumors, sensitizes NB cells to CAR T-cell therapy, and thus promotes the death of NB cells. MHC-I is a protein complex that plays a crucial role in the destruction of tumorous or infected cells. By demonstrating autophagy inhibition's ability to amplify the effects of CAR T cells , De Mitri et al. (2025)

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provide a valuable new perspective on improving the effects of CAR T-cell therapy by combining it with autophagy-targeted methods.

YAP is a transcriptional co-regulator highly expressed in NB cells, and is a key contributor to immune resistance within the solid TME. Immune cells that express YAP have demonstrated ability to support immunosuppression and diminish effects of immunotherapy. Furthermore, the YAP protein inhibits the GD3 synthase gene ST8SIA1, thus suppressing GD2 expression; correspondingly, NB cells with low levels of GD2 show high YAP gene. A study shows that YAP inhibition can make NB cells more vulnerable to anti-GD2 antibodies (Pilgrim et al., 2023). Accordingly, YAP inhibition can elevate ST8SIA1 expression, which can in turn promote GD2 expression in NB cells, suggesting that YAP inhibition can improve CAR T-cell therapy.

## FUTURE PERSPECTIVES IN CAR T-CELL THERAPY

CAR T-cell therapy is a relatively new technology in the field of cancer immunotherapy treatment, with many areas for improvement. When designing CAR T cells, many factors affect CAR structural plasticity: epitope specificity, scFv affinity, CAR hinge, costimulatory domain, and transmembrane (Sun et al., 2023). These components are crucial to CAR T cell design. Paying attention to and accounting for these components can improve the efficacy of CAR T-cell therapy, and is a valuable perspective for future studies. Additionally, Pascual-Pasto et al. (2024) examined the ability of their GPC2.CAR-GD2.BiCE T-cells with immunodeficient mice. However, due to the difference in mouse and human receptors and lack of human innate immune cell ability to effectively traffic in mouse tissue, limitations were identified in their system. As a result, alternatives such as using syngeneic murine neuroblastoma models or mice in which murine Fcy receptors have been replaced by human ones have been identified. Investigation of these alternate methods in future studies may address the limits to the current work of those such as Pascual-Pasto et al. (2024). Finally, Tian et al. (2025) describes the potential of synthetic regulatory circuits in future studies to both improve the therapeutic results and minimize the side effects of CAR T cells. These synthetic regulatory circuits are designed to manage both cytokine release and CAR T cell proliferation, ensuring that immune cells numbers remain effective but are kept within a safe range.

NB treatment is an expanding field that possesses numerous promising technologies, which once further developed, will be crucial to successfully overcoming the deadly neuroblastoma.

## CONCLUSION

Neuroblastoma, the most common solid tumor occurring in children, contributes to over 15% of childhood cancer deaths. NB is a solid tumor associated with poor prognosis due to high growth rate and frequent metastasis. Deriving from a block in proper neural crest cell differentiation, NB is a disease of the

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sympathetic nervous system. NB cells demonstrate low immunogenicity, obstructing the immune system's ability to detect and destroy them. Among established clinical treatments, although anti-GD2 mAbs are prevalent, such methodologies have failed to overcome high-risk NB. CAR T cell-based immunotherapy is recognized as a promising method of NB treatment. However, CAR T-cell therapy comes with its own challenges: mainly, a risk of inducing CRS and on-target off-tumor toxicity. Additionally, CAR T cells must overcome the immunosuppressive microenvironment of tumors. Currently, many pathways of improving CAR T cell efficacy are being explored. Various studies have investigated different types of immunotargets, including GD2, GPC2, and B7-H3 (CD276). CAR T cells targeting two different antigens at the same time, such as BiCEs and BiCisCARs, have also been examined. Research on using different types of T cells to engineer CAR T cells has also been conducted, including the NK-92 cell line and NKT cells. Furthermore, novel studies into the inhibition of autophagy and YAP (yes-associated protein) show great potential for improved CAR T cell therapy. It is conceivable that with these new explorations and methods, the treatment and prognosis of NB patients will be improved.

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