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Precision Medicine and Targeted Therapy in Pediatric Oncology: Current Status and Perspectives in Mexico

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Abstract

Precision medicine is transforming pediatric oncology by tailoring treatment to the genomic profile of each tumor, moving beyond the traditional one-size-fits-all approach. This strategy is especially relevant in children, whose tumors are rare and have a low mutational burden—which limits therapeutic targets but makes accurate detection of actionable alterations essential. This review describes the current status of targeted therapy and precision medicine in Mexican pediatric oncology, highlighting recent advances, structural challenges, and strategies for future consolidation. International programs such as Australia's ZERO Childhood Cancer demonstrate that molecular tumor boards and genomic profiling improve outcomes in high-risk disease. In Mexico, progress is incipient but promising: in acute leukemias, imatinib and the bispecific antibody blinatumomab—followed by hematopoietic stem-cell transplantation—have shown clinical and cost-effectiveness benefits, and an academic anti-CD19 CAR-T platform is under preclinical development; in lymphomas, dose-optimized immunotherapy (low-dose nivolumab, brentuximab vedotin) offers equitable, sustainable access; and in central nervous system tumors, neuroblastoma, and bone tumors, molecular characterization is increasingly feasible despite logistic barriers. Persistent challenges include limited access to next-generation sequencing, geographic inequity, high costs, fragmented infrastructure, and a shortage of trained personnel.

Consolidating regional molecular tumor boards, locally validated genomic panels, defined drug-access pathways, and collaborative research is essential to make precision pediatric oncology accessible, sustainable, and equitable in Mexico.

Keywords

Precision medicine; Targeted therapy; Pediatric oncology; Next-generation sequencing; Immunotherapy; Mexico.

Abbreviations

NGS: Next-Generation Sequencing; ALL: Acute Lymphoblastic Leukemia; Ph+: Philadelphia Chromosome-Positive; CML: Chronic Myeloid Leukemia; TKI: Tyrosine Kinase Inhibitor; COG: Children's Oncology Group; MRD: Minimal Residual Disease; HSCT: Hematopoietic Stem-Cell Transplantation; CRS: Cytokine Release Syndrome; CAR-T: Chimeric Antigen Receptor T-cell; R/R: Relapsed/Refractory; BiTE: Bispecific T-cell Engager; ADC: Antibody–Drug Conjugate; ICI: Immune Checkpoint Inhibitor; CNS: Central Nervous System; DIPG: Diffuse Intrinsic Pontine Glioma; IMSS: Mexican Institute of Social Security; HIMFG: Hospital Infantil de México Federico Gómez; INMEGEN: National Institute of Genomic Medicine; HITO: Hospital Infantil Teletón de Oncología; FDA: US Food and Drug Administration; EMA: European Medicines Agency; SNP: Single-Nucleotide Polymorphism; GM-CSF: Granulocyte-Macrophage Colony-Stimulating Factor.

Introduction

Pediatric oncology is undergoing a transformation driven by precision medicine, which makes it possible to tailor treatments to the genomic profile of each tumor, in contrast to the traditional one-size-fits-all approach. This strategy is especially relevant in pediatrics, where tumors are rare and have a low mutational burden—which limits therapeutic targets but makes accurate detection of actionable alterations vital [1]. In Mexico—where overall survival reaches approximately 75%, albeit with high rates of chronic sequelae—advancing toward more specific and less toxic therapies represents a clinical and public-health priority [2].

The objective of this review is to describe the current status of targeted therapy and precision medicine in Mexican pediatric oncology, highlighting recent advances and structural challenges and proposing strategies for its future consolidation.

International Landscape and Recent Clinical Evidence

Precision-medicine programs, such as Australia's ZERO Childhood Cancer, are generating solid clinical evidence of their utility. In its PRISM trial, which included 384 children with high-risk cancer, tumor genomic profiling allowed a targeted therapy to be recommended for 67% of patients; of these, 29% were able to receive the recommended treatment [3]. Results in these patients were encouraging: an objective response rate of 36% was observed, and two-year progression-free survival was significantly higher (26%) than in those who received only standard treatment (12%) or a targeted therapy not based on their molecular profile (5.2%). These findings support the view that early target identification and consensus decision-making within a molecular tumor board can improve outcomes, even in cases with a guarded prognosis [4].

These initiatives rest on three fundamental pillars: (1) the use of robust genomic sequencing (whole-

genome, transcriptomic, and methylation); (2) discussion in specialized multidisciplinary committees; and (3) the creation of drug-access pathways through clinical trials, compassionate use, or institutional funding [5]. Although pediatric tumors have fewer actionable alterations than adult tumors, next-generation sequencing (NGS) with multigene panels has identified new therapeutic opportunities in both hematologic malignancies and solid tumors [2,6].

Targeted Therapies: Global Evidence and the Mexican Reality

The landscape of targeted therapy in Mexico is incipient but promising, characterized by pioneering institutional efforts, a growing base of local genomic evidence, and a palpable disparity in access to these cutting-edge technologies. Unlike countries with consolidated programs, implementation in Mexico faces unique challenges of infrastructure, financing, and equity, although it is beginning to chart its own course [7].

Acute leukemias

Acute leukemias are the most frequent type of childhood cancer. Their pathophysiology lies in the clonal proliferation and accumulation of malignant lymphoid or myeloid precursors in the bone marrow, which suppress normal hematopoiesis. They are the paradigm of success in pediatric oncology, with cure rates exceeding 80% worldwide thanks to intensive chemotherapy protocols and risk stratification.

Among the wide repertoire of available therapies, imatinib stands out as the pioneer of targeted therapies. This tyrosine kinase inhibitor was developed to specifically block the oncogenic activity of the BCR-ABL fusion protein resulting from the t(9;22) translocation, or Philadelphia chromosome (Ph), characteristic of chronic myeloid leukemia (CML) and of a subtype of acute lymphoblastic leukemia (Ph+ ALL). Although patients with Ph+ ALL represent less than 3% of pediatric cases, the clinical relevance of this subgroup is enormous: before targeted therapies, its prognosis was unfavorable, and the only possibility of complete remission and long-term cure was allogeneic bone-marrow transplantation—a procedure with significant morbidity and mortality and limited access. This picture changed radically from the year 2000, when the Children's Oncology Group (COG) established the use of imatinib combined with chemotherapy for Ph+ ALL, achieving 5-year event-free survival rates of approximately 70%—not only comparable to those observed in adults but also allowing transplantation to be avoided in a significant subset of patients [8,9].

In Mexico, implementation in clinical practice outside controlled trials is often characterized by significant heterogeneity; experience at national centers, while valuable, often does not translate into robust clinical studies or publications that can inform local practice guidelines. The difficulty of generating scientific evidence is compounded by the low incidence of some molecular subgroups. For example, a longitudinal study that reviewed records over two decades at a Mexican referral center identified only three cases of Ph+ ALL, illustrating the pressing need for national multicenter consortia to assemble cohorts of sufficient size to yield statistically significant results [10].

Considering that 3 of every 10 patients will require a second or third line of treatment, the incorporation of other therapies such as immunotherapy constitutes a strategy superior to conventional chemotherapy for achieving negative minimal residual disease and proceeding to bone-marrow transplantation.

A critical challenge in implementing innovative therapies such as blinatumomab is their high initial cost.

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However, pharmacoeconomic analyses are crucial to demonstrate their long-term value. A model based on the Mexican setting demonstrated that blinatumomab not only improves clinical outcomes but also represents an efficient use of health resources, with a 99.5% probability of being cost-effective according to local thresholds [11]. This type of evidence is fundamental for advocating its inclusion in the essential-medicines formularies of public institutions.

Fortunately, this economic soundness is supported by robust clinical evidence generated in the national context. A Mexican multicenter study in 76 children with relapsed/refractory (R/R) CD19+ pre-B ALL demonstrated that administration of blinatumomab as bridging therapy allowed 74% of patients to achieve molecular remission (MRD <0.002). More importantly, this study confirmed the transformative impact of the sequential strategy of blinatumomab followed by hematopoietic stem-cell transplantation (HSCT): patients who received a haploidentical transplant after remission with blinatumomab achieved 83% survival, in marked contrast to 0% among those who received blinatumomab alone. The observed safety profile—dominated by low-grade cytokine release syndrome (grade I–II in 90% of cases) and low treatment-related mortality (1.3%)—aligns with international reports and reinforces the feasibility of implementing this strategy in centers experienced in managing immunotherapies [12].

Although few studies on the national experience have been published, a retrospective study from the Hospital Infantil Teletón de Oncología (HITO) in nine patients with relapsed/refractory ALL mirrored the international experience: 88% of patients required two or more cycles to achieve negative MRD, reinforcing the importance of prolonged therapeutic exposure to maximize response. Although the cohort was small, survival rates were encouraging (3-year overall survival of 65%), demonstrating the usefulness of the strategy [13].

Blinatumomab has unquestionably established a new paradigm in the treatment of relapsed/refractory ALL by demonstrating that the patient's immune system can be redirected against the malignancy with favorable efficacy and toxicity in high-risk settings [14]. Its success laid the conceptual and clinical foundations for the development of an even more personalized and potent immunotherapy: chimeric antigen receptor T-cell (CAR-T) therapy. If blinatumomab is an ephemeral molecular bridge requiring continuous administration, CAR-T cells represent the construction of a personalized, durable “army” within the patient. This transition from bispecific therapy to advanced cell therapy marks the natural evolution of immuno-oncology [15]. However, access to these innovative therapies is profoundly unequal worldwide. While high-income countries are rapidly advancing in the implementation of commercial CAR-T products, Mexico and other middle-income nations face formidable barriers: high costs, complex infrastructure, logistic challenges, and technological dependence on other countries. Against this backdrop, a critical and hopeful question arises: is it possible for a middle-income country such as Mexico not only to access but to endogenously develop its own CAR-T platform? [16]. The answer appears to be germinating in the national academic sphere. Initiatives such as the academic development of an anti-CD19 lentiviral vector for the transduction of chimeric T lymphocytes (at the preclinical stage) show that the answer is a resounding yes. Such projects are not a mere replication exercise; they are an act of scientific sovereignty and health equity. The governmental and institutional momentum toward this new therapy, far from being a fashion, is a necessary and visionary strategy: it represents the recognition that true sustainability in cancer treatment comes not only from purchasing foreign therapies but from

internalizing research, development, and innovation capabilities. This allows technologies to be adapted to the local epidemiologic and economic reality, reduces long-term costs, and trains the specialized human talent the country needs so as not to remain perpetually behind [17].

Local preclinical development of CAR-T is; therefore, the logical consequence of the lessons learned with therapies such as blinatumomab and the indispensable first step toward building a robust precision-medicine ecosystem that is accessible, sustainable, and, above all, Mexican.

Lymphomas

Lymphomas are a heterogeneous group of malignant neoplasms arising from the clonal transformation of lymphocytes (B or T) at any point in the lymphatic system (lymph nodes, spleen, mucosa-associated lymphoid tissue [MALT]), characterized by high potential for growth and dissemination. They are the third most frequent cancer in children, after central nervous system tumors. Broadly, they are divided into two groups—Hodgkin and non-Hodgkin—the latter being the most frequent in pediatrics overall, while the former is more common in adolescents and young adults [18].

In Mexico, since late 2021 the Mexican Institute of Social Security (IMSS) has undertaken strategic alliances to lay the foundations of precision medicine in pediatric oncology. During 2022, pilot protocols were introduced that included in vitro cultures and the integration of clinical, molecular, and immunologic data, with the fundamental aim of identifying subgroups of patients who might benefit from targeted treatments. This strategy currently operates through a network of 160 telemedicine meetings connecting 35 national experts, a system for regionalizing sample shipment across eight strategic areas of the country, and plans to enable state ONCOCREAN centers as references for anatomic-molecular diagnosis [19].

The fruit of these systemic efforts is beginning to be reflected in the generation of local clinical evidence. A paradigmatic example is a multicenter retrospective study in 23 adult patients with relapsed/refractory Hodgkin lymphoma that evaluated low-dose nivolumab (LD-Nivo). Although the population was adult, its findings are extremely relevant to pediatrics and to the Mexican context: a dose approximately four times lower than the standard (0.78 mg/kg vs. 3 mg/kg) achieved an overall response rate of 73% and one-year overall survival of 94.4%, with a 77–83% reduction in cost per cycle. This dose-optimization model—evaluated in a collaboration among institutions in Nuevo León, Sonora, and Mexico City—represents not only a therapeutic advance but a paradigm of equitable access and financial sustainability for implementing immunotherapies in the Mexican health system, and sets a precedent for exploring similar strategies in the pediatric population [20].

For Mexican children with cancer, direct evidence already exists. An international multicenter phase 1/2 study—which included centers in our country—evaluated brentuximab vedotin (1.8 mg/kg) in children with relapsed/refractory Hodgkin lymphoma or anaplastic large-cell lymphoma. The treatment demonstrated an overall response rate of 46% and served as an effective bridge to stem-cell transplantation in 47% of patients, with a manageable safety profile in which the most relevant toxicity, peripheral neuropathy, was mostly low-grade and reversible [21].

Taken together, these studies illustrate the way forward: the implementation of systemic strategies by large institutions (IMSS) must go hand in hand with the generation of robust, pragmatic clinical evidence that validates the use of targeted therapies at optimized doses and demonstrates their value—not only

clinical but also in terms of access and cost-effectiveness—for the reality of Mexico.

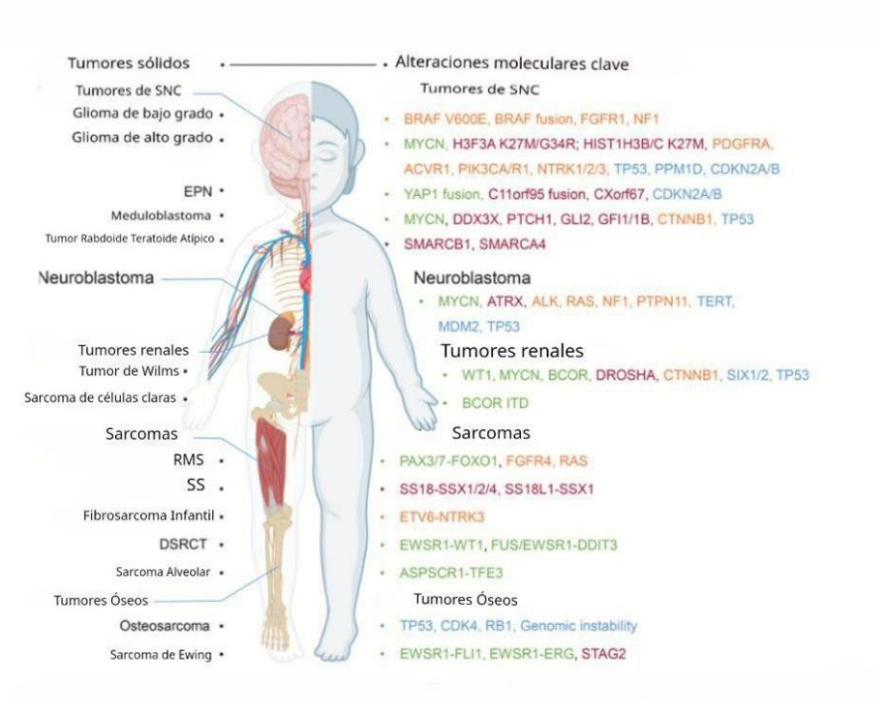

Figure 1: Overview of characteristic molecular alterations in pediatric solid tumors. The most frequent pediatric solid tumors and their most frequent molecular alterations are grouped into functional categories indicated by color (green = transcription factor; red = epigenetic/transcriptional regulator; orange = kinase/signaling pathway; blue = cell cycle/stress response). EPN: ependymoma; RMS: rhabdomyosarcoma; SS: synovial sarcoma; DSRCT: desmoplastic small round cell tumor [22].

Central nervous system tumors

These represent the second most frequent neoplasm in pediatrics, accounting for 25% of childhood neoplasms. Although current survival exceeds 75%, they are among the leading causes of cancer death in those under 20 years of age and are also associated with substantial morbidity and neuroendocrine sequelae [18].

Gliomas are the most frequent in pediatrics, with heterogeneous biological behavior. Pediatric oncology is currently moving from a histological model to a molecular one. This paradigm is particularly relevant in tumors with a poor prognosis, where the identification of actionable alterations can redefine management. An emblematic example is diffuse intrinsic pontine glioma (DIPG), for decades an entity without therapeutic options. Implementing precision medicine for DIPG in Mexico faces a complex reality. A retrospective study at a tertiary hospital [23] illustrates these barriers: of 29 patients diagnosed, only 37% could undergo a biopsy, and of these, only 5 cases (17% of the total cohort) yielded a sample sufficient for immunohistochemical studies. This logistic and technical limitation partly explains why the median overall survival reported in national series (4.2–5 months) [23,24] is significantly lower than that reported in countries with systematic access to biopsy and targeted therapies (9–12 months) [18].

Despite these barriers, molecular characterization efforts have borne fruit. One dedicated study [23] determined the presence of the H3K27M mutation in 72.6% of biopsied patients, a finding corroborated

in the small subsample of the second study (80%, 4/5) [24]. This not only confirms that the prevalence of the alteration is comparable to the global figure but also demonstrates the technical feasibility of implementing molecular diagnosis in our setting—an indispensable first step toward accessing targeted therapies.

Neuroblastoma

Neuroblastoma is a malignant neoplasm arising from neural crest cells, the precursors of the sympathetic nervous system. It is the most frequent extracranial solid tumor in the pediatric population, accounting for approximately 6% of all cancers in this age group. Clinical presentation is most frequent in children under 2 years of age, a group that comprises nearly 50% of cases [18].

Although its clinical spectrum is diverse—owing to its ability to manifest along the entire sympathetic nervous system chain—presentation can range from well-differentiated tumors with relatively indolent biological behavior to highly aggressive clinical forms. Currently, within the approach proposed by one of the country's most important institutions, the Hospital Infantil de México Federico Gómez (HIMFG), therapeutic management is established according to risk stratification (low or high), based fundamentally on clinical and histopathologic criteria.

However, the development of precision pediatric oncology in Mexico cannot consist solely of importing high-cost treatments. It must be grounded in combining such therapies with a deep understanding of the biology and genetics of our own population. HIMFG studies of polymorphisms in cytokine genes (IL-6, IL-8, TNF-α) represent a seminal first step toward that goal, identifying a particular immunogenetic profile that could explain the greater aggressiveness reported in national cohorts. Routine implementation of these findings, integrated with the indispensable improvement in molecular diagnosis (such as determination of MYCN and 11q), could revolutionize neuroblastoma care in the country, enabling more precise, equitable, and effective risk stratification and, ultimately, more rational use of targeted therapies [25,26,27].

Therapeutic management is established according to risk stratification (low or high). The initial approach should consider complete surgical resection when feasible; however, in cases of unresectable tumors, neoadjuvant chemotherapy should be chosen to reduce tumor burden and facilitate surgery at a later stage. The main therapeutic challenge is posed by patients classified as high-risk, in whom survival rates do not exceed 50%. Among the new treatments aimed at improving survival in this subgroup are monoclonal antibodies directed against gangliosides, antigens characteristically expressed on the surface of these tumor cells [18].

Bone tumors

Bone tumors account for 5% of pediatric malignancies, with osteosarcoma and Ewing sarcoma being the most frequent entities in oncologic practice. Their treatment is a complex multidisciplinary challenge because of their local aggressiveness and high metastatic potential. In pediatrics, both have a higher incidence in adolescence (10 to 15 years). Osteosarcoma presents mainly in the metaphysis of long bones, whereas Ewing sarcoma arises most frequently in the diaphysis of long bones [18].

The landscape of targeted therapies in pediatric osteosarcoma remains limited, with mifamurtide one of the few approved options; however, its global adoption is not uniform, and barriers such as cost and

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logistics exist. A recent, preliminary retrospective study conducted in Mexico evaluated the incorporation of mifamurtide into a regimen using cyclophosphamide instead of ifosfamide. In this small cohort of 7 patients with localized osteosarcoma, a trend toward 85.7% one-year survival was observed, aligning with data reported in the international literature. Although the small sample size precludes definitive conclusions, this work serves as an important pilot study suggesting that this combination could be viable and merits exploration in a larger cohort [28].

Neoplasm	Molecular target	Drug	Therapy type	Mechanism of action	Approval (pediatric indication)
Ph+ ALL	BCR-ABL1	Imatinib	Tyrosine kinase inhibitor (TKI)	Inhibits the BCR-ABL1 tyrosine kinase.	FDA & EMA, with chemotherapy as first-line treatment.
	BCR-ABL1	Dasatinib	TKI	Inhibits BCR-ABL1 with greater potency and crosses the blood–brain barrier.	FDA & EMA, with chemotherapy as first-line treatment.
R/R CD19+ ALL	CD19	Blinatumomab	BiTE (bispecific antibody)	Links T cells (CD3+) to leukemic B cells (CD19+), activating T-cell–mediated cytotoxicity.	FDA & EMA (relapsed/refractory ALL).
	CD19	Tisagenlecleucel	CAR-T therapy	Patient T cells modified to express an anti-CD19 chimeric receptor.	FDA & EMA (R/R ALL, <25 years).
R/R CD30+ Hodgkin lymphoma (HL)	CD30	Brentuximab vedotin	Antibody–drug conjugate (ADC)	Anti-CD30 monoclonal antibody linked to an antimitotic agent (MMAE); internalization and toxin release.	FDA & EMA (pediatric relapsed/refractory HL).
Non-Hodgkin lymphoma (NHL)	CD20	Rituximab	Monoclonal antibody (anti-CD20)	Depletion of CD20+ B cells via ADCC and complement (CDC).	FDA & EMA (pediatric B-cell lymphoma, with chemotherapy).
	PD-1	Nivolumab	Immune checkpoint inhibitor (ICI)	Blocks the PD-1/PD-L1 interaction, reactivating the antitumor immune response.	FDA (pediatric classic HL, R/R after transplant or post-brentuximab).
	PD-1	Pembrolizumab	ICI	Blocks the PD-1/PD-L1 interaction.	FDA (pediatric R/R classic HL).
Neuroblastoma	GD2	Dinutuximab	Chimeric mAb (anti-GD2)	Binds GD2, mediating CDC and ADCC.	FDA (high-risk neuroblastoma in first remission) – withdrawn in Europe.
	GD2	Dinutuximab beta	Chimeric mAb (anti-GD2)	Same mechanism as dinutuximab but a different production process (cell culture).	EMA (high-risk neuroblastoma, patients >12 months).

Neoplasm	Molecular target	Drug	Therapy type	Mechanism of action	Approval (pediatric indication)
	GD2	Naxitamab	Humanized mAb (anti-GD2)	Binds GD2 with high affinity, mediating ADCC.	FDA (high-risk neuroblastoma in bone marrow/bone, R/R, with GM-CSF).
Solid tumors with BRAF V600E mutation	BRAF V600E	Dabrafenib	BRAF inhibitor	Selectively inhibits mutated BRAF V600E kinase.	FDA & EMA (with trametinib for BRAF V600E-mutant pediatric solid tumors).
	MEK	Trametinib	MEK inhibitor	Inhibits MEK1/2 kinases, downstream targets of BRAF.	FDA & EMA (with dabrafenib for BRAF V600E-mutant pediatric solid tumors).
Low-grade glioma and others with MAPK-pathway alterations	BRAF V600E	Dabrafenib + trametinib	Combined inhibitors (BRAF+MEK)	Dual MAPK-pathway inhibition to prevent escape mechanisms.	FDA & EMA (pediatric glioma with BRAF V600E mutation).
	BRAF fusions	Selumetinib	MEK1/2 inhibitor	Inhibits MEK1/2 signaling, key in gliomas with BRAF fusions or NF1.	FDA (NF1 with symptomatic, inoperable plexiform neurofibromas).
Osteosarcoma	Macrophage activation	Mifamurtide	Immunomodulator (L-MTP-PE)	Activates macrophages and monocytes, stimulating the immune response against micrometastases.	EMA (adjuvant in resected osteosarcoma, with chemotherapy) – not FDA-approved.
Solid tumors with NTRK fusion	NTRK (1/2/3)	Larotrectinib	Pan-TRK TKI	Pan-TRK tyrosine kinase inhibitor.	FDA & EMA (NTRK-fusion pediatric/adult solid tumors, no alternative).
	NTRK (1/2/3)	Entrectinib	Pan-TRK TKI	Inhibits TRK, ROS1, and ALK; designed to cross the blood–brain barrier.	FDA & EMA (NTRK-fusion solid tumors >12 years/adults, or ROS1+ NSCLC).
Alveolar rhabdomyosarcoma (PAX3/7-FOXO1 fusion)	—	(Clinical trials)	—	—	No specifically approved targeted therapy; standard is chemotherapy, radiotherapy, surgery.
Ewing sarcoma	—	(Clinical	—	—	No specifically

Neoplasm	Molecular target	Drug	Therapy type	Mechanism of action	Approval (pediatric indication)
		trials)			approved targeted therapy; standard is chemotherapy, radiotherapy, surgery.
Retinoblastoma	—	No targeted therapy	—	—	Standard of care for intraocular disease is an administration technique, not a molecular targeted therapy.
Tumors with ALK alteration	ALK	Crizotinib	ALK/ROS1/MET inhibitor	Inhibits ALK kinase, a target of fusions in ALL and lymphomas and of mutations in neuroblastoma.	FDA (pediatric R/R ALK+ anaplastic large-cell lymphoma); ALK-mutated R/R neuroblastoma (off-label).
	ALK	Alectinib	ALK inhibitor (2G)	Inhibits ALK with greater potency and CNS activity than crizotinib.	FDA (ALK-mutated R/R neuroblastoma).
	ALK	Lorlatinib	ALK inhibitor (3G)	Inhibits ALK resistant to other TKIs and crosses the blood–brain barrier.	FDA (ALK-mutated R/R neuroblastoma).

Table 1: Targeted therapies available in pediatrics approved by the FDA and/or EMA. FDA, US Food and Drug Administration; EMA, European Medicines Agency [18].

Institutional Initiatives and National Strategies

The National Institute of Genomic Medicine (INMEGEN), located in Mexico City, is a key center for research in oncogenomics, biomarkers, and pharmacogenomics. Although its focus is broad, its capabilities could be integrated into the pediatric effort, contributing infrastructure and technical expertise to drive national genomic panels [7,23].

In the Mexican literature, studies such as those on ARID5B single-nucleotide polymorphisms (SNPs) in childhood acute lymphoblastic leukemia exemplify how genetic markers can explain predisposition and open doors to more targeted treatments in the Mexican population [2].

Barriers and Opportunities in the Mexican Context

Barriers:

- Limited access to specialized pediatric NGS technologies, with high geographic inequality among states.
- Shortage of personnel trained in bioinformatics and multidisciplinary molecular tumor boards.
- High costs of tests and targeted drugs, and the absence of homogeneous coverage by health regulators or funders.
- Fragmented infrastructure and logistic barriers to the rapid shipment and processing of samples.

Opportunities:

- The ONCOCREAN centers offer a platform to centralize pediatric molecular diagnosis and generate synergies.
- The IMSS experience in telemedicine and national committees provides a replicable, scalable model.
- INMEGEN and academic centers can lead the development of panels and genomic-clinical databases, including pharmacogenomic analysis capability.
- Public–private collaborations and participation in international networks (such as ZERO, INFORM, MAPPYACTS) can broaden access to drugs and clinical trials.

Perspectives and Recommendations

To consolidate precision medicine in Mexican pediatric oncology, the following are suggested:

1. Consolidate regional and federal pediatric molecular tumor boards, with participation from oncology, genomics, bioinformatics, and pharmacology.
2. Develop locally validatable national genomic panels, accessible from regional centers and coordinated by INMEGEN and academic networks.
3. Ensure targeted-therapy access pathways through clinical trials, compassionate-use programs, and dialogue with COFEPRIS and institutional funders.
4. Foster collaborative research including pediatric genomic studies representative of the Mexican population, with evaluation of clinical impact and cost-effectiveness.
5. Train human capital (oncologists, pathologists, bioinformaticians), promote policies that include pediatric precision medicine in standard care, and strengthen logistic infrastructure (sample transport, digital platforms, clinical registries).

Conclusion

Precision medicine in pediatric oncology represents a therapeutic revolution capable of improving response and survival even in tumors with a critical prognosis. International studies and the growing momentum of the IMSS show that the model is viable, though still incipient in Mexico. Consolidating a coordinated national strategy—with diagnostic infrastructure, multidisciplinary committees, and equitable access to therapies—will transform the care of children with cancer in the country. That goal is not only clinically viable but indispensable for advancing toward a fairer and more effective future in pediatric oncologic health.

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None.

Conflict of interest

The authors declare that they have no conflict of interest.

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