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Perspective

Therapy for the Treatment of Fms-Related Tyrosine Kinase 4 (FLT4)-Targeting Peptide in Blood Cancer

John Jennifer*

Department of Parasitology, Institute of Mexico Federico, Cancún, Mexico

DESCRIPTION

A rise in myeloid lineage cells with faulty genetic alterations that lead to dysfunctional hematological and immunological processes is referred to as Acute Myeloid Leukemia (AML). Immune checkpoint inhibitors, small compounds, therapeutic antibodies, blocking peptides, and chimeric antigen receptor-T cells have all undergone extensive clinical trials to treat it. Therapeutic peptides, which have great selectivity and strong solubility, are seen as attractive alternatives for clinical application in treating both leukemia and solid tumors, because most small compounds have a limited therapeutic window and severely harm normal tissues. Peptide-based combinational therapy is developing as a key technique for treating cancer. Peptides offer numerous benefits, including their small size, ease of synthesis, and access to the tumors, but big proteins, like monoclonal antibodies, have certain disadvantages, such as difficulty being transported into the tumors and non-specific absorption into the reticuloendothelial system. Peptide's ability to be used directly in clinical trials with a variety of combination regimens is their main advantage. Numerous peptide therapeutics, such as different Fc-fusion peptides, mutated peptides, and extracellular domain-targeting peptides, have been reported to have benefits and drawbacks in terms of their pathophysiologic function and present cutting-edge opportunities to fine-tune their activity spectrum. Synthetic peptides are particularly effective at controlling molecular movements because they are small, with a 12/18-mer shape that allows them to enter cells and operate as bioactive peptides that target internalized signaling pathways. Several domains are selected as the targeted binding sites using the crystal structure data, including the Vascular Endothelial Growth Factor C (VEGF-C) binding sites into the FLT4 extracellular domain (D1-2) and FLT4 intracellular domain (D4-5). The extracellular domain or homodimer structure of the intracellular kinase domain should be suppressed in order to prevent FLT4 kinase from binding to its ligand. Inhibiting FLT4 activity by preventing its phosphorylation boosted the production of IFN-γ in immune cells, suggesting that FLT4 inhibition offers the potential advantage of cancer regression in tumor microenvironments. Studies about the signal domains for targeting peptides are still lacking in comparison to studies about the extracellular domains for targeting peptides, despite the biological significance of the intracellular kinase domain in immune cell activation. Through its ligands VEGF-C and -D, FLT4 (also known as VEGFR-3) is a typical mediator of lymph angiogenesis. Both hematological malignancies and solid tumors can initiate lymph angiogenesis through the VEGF-C/FLT4 axis as a gatekeeper signaling mechanism. The first kinases to be taken into account in cancer research were Receptor Tyrosine Kinases (RTKs), and FLT4, an RTK belonging to the VEGF family, is a well-known conventional target for the treatment of leukemia. Our team previously concentrated on the role of the FLT4 inhibitor MAZ51 in restoring NK cell functionality because it is involved in the high rate of growth and survival of leukemia cells as well as with dysfunctional Natural Killer (NK) cells. This study demonstrates the therapeutic effects of blocking FLT4 by showing that it increased IFN- γ in NK cells in AML.

Hence, these Fc targeting peptides discovered in tumor microenvironments are emerging as therapeutic possibilities for treating tumor's directly or indirectly *via* access to immune cells. The investigations verified the antagonistic action of various peptides as an antibody concept that can decrease FLT4 activity by binding soluble FLT4, and they indicated the therapeutic potential of peptides by decreasing FLT4 kinase activity in tumor growth. This field has to be researched as there are currently no treatment recommendations for a peptide that inhibits FLT4 activity in the defective immune cells in tumor microenvironments.

Correspondence to: John Jennifer, Department of Parasitology, Institute of Mexico Federico, Cancún, Mexico, E-mail: jennifer@gmail.com

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