

# The Impact of Arsenic Trioxide on Acute Myeloid Leukemia

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## DESCRIPTION

Hematopoietic progenitor cells that control self-renewal, proliferation, and differentiation accumulate somatic cytogenetic changes in Acute Myeloid Leukemia (AML), a diverse clonal disease. With treatment, approximately 30% to 40% of AML patients would survive for longer than five years. Damage to the immune system and Tumor Microenvironment (TME) in favor of immunological escape is one factor contributing to the poor prognosis of AML. As leukemia proceeds, AML and NK cells can devise ways to evade immune system detection and enable AML cell multiplication in TME. According to another research, the immunosuppressive TME and the selective loss of an immature subset of NK cells in AML patients are likely to blame for the unfavorable prognosis, so it's critical to investigate methods for improving TME and NK cell anti-tumor immunity against AML. Gasdermins carry out pyroptosis, a type of inflammatory cell death. In order to relieve auto inhibition on the gasdermin-N fragment, which is essential to the pyroptosis process, the caspases cleave the gasdermins in their middle linker. This defines the gasdermin's pore-forming activity.

The external outflow caused by cell membrane holes, which triggers antitumor immunity, is what distinguishes pyroptosis from other processes. Consequently, pyroptosis-inducing treatment approaches may increase the effectiveness of immunotherapy for leukemia. Important cytotoxic and cytokine-producing elements of the innate immune system are NK cells. There is enough data to conclude that Natural Killer (NK) cells can eradicate both acute and chronic myeloid leukemia cells. Patients with AML who express NK-activating ligands on their blasts have a better prognosis. Pyroptosis and Natural Killer (NK)

cells have an interdependent interaction. On the other hand, it has been noted that pyroptosis stimulates NK cells. During pyroptosis, holes on the cell membrane surface open up and produce molecules known as Damage-Associated Molecular Pattern (DAMPs), such as HMGB1 and IL-18, which can activate Natural Killer (NK) cell's immune system against tumors. Immunoproteins, for instance, like HMGB1, can control the activation of NK cells and take role in lymphocyte development. According to reports, NK cell cytotoxicity can be strongly stimulated by gasdermin E (GSDME)-mediated pyroptosis, perhaps leading to an advantage in survival. Granzymes B is one of the cytokines that NK cells produce, which is how they carry out their cytotoxicity on cancer cells.

However, granzyme that is released during NK cell degranulation has the ability to directly cause pyroptosis that is not dependent on caspase. Granzyme B (GZMB) can cause GSDME-dependent pyroptosis in tumors by cleaving GSDME directly and activating caspase-3 indirectly. Granzyme A can also cause pyroptosis by cleaving GSDMB directly. Granzyme's activity enables NK cells to positively feedback on pyroptosis. Chemical medicines have been shown in earlier studies to improve antitumor immunity by the conversion of apoptosis to pyroptosis. One therapeutic medication that has proven to be useful in treating Acute Promyelocytic Leukemia (APL) is Arsenic Trioxide (ATO). ATO may be connected to pyroptosis since it has been proposed that it can reinstate the oncogenic activity of p53, which can trigger the transcription of GSDME, a member of the Gasdermin family. Furthermore, it has been previously demonstrated that ATO increases the *in vitro* NK cell cytotoxicity against APL. ATO has the ability to up-regulate the NK ligands on tumor cells, making cancer cells more vulnerable to NK cells.

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