**A Development in Leukemia Research**

Acute myeloid leukemia (AML) is a hematologic malignancy. This means that the disease begins in the cells of blood-forming tissue (e.g. bone marrow) or in the cells of the immune system. Specifically, AML is caused by a buildup of underdeveloped leukemic blasts in both the blood and in the bone marrow. While we have grown to understand the disease in greater depth since its discovery, there has been minimal progress in conventional therapy for AML. By epigenetic modification of the leukemia cells through targeting chromatin regulators (IDH, HDAC, and BRD4) there is a chance that therapy methods can be improved. Another approach to treatment is through the activation of the tumor suppressor p53. Tumor suppressor p53 codes for a protein that manages the cell cycle, thus working to suppress cancer. The study conducted by Minzel et. al. brought forth the conclusion that the removal of CKI𝛼 results in the activation of tumor suppressor p53 and the degradation of CKI𝛼 primes the therapeutic effect of the drug lenalidomide in a pre-leukemia syndrome.

To test for AML doctors will test blood and bone marrow. The blood test will be done first and will be followed by bone marrow aspiration and bone marrow biopsy. AML can also be tested for within the spinal fluid. This is because the disease can sometimes spread around the brain and the spinal cord. AML can be treated with drugs, chemotherapy, and targeted therapy. According to the article, a notable disadvantage of all available drug treatments for AML is quick relapse following a short remission and poor response to the drug itself (Minzel et. al., 2018). Molecules that target CKI𝛼, CDK7, and CDK9 (transcriptional kinases) have a healing effect according to mouse models. These mouse models were also used to test the effect of bone marrow transplants and what the procedure could do to eliminate AML. The mouse was given a lethal dose of radiation to kill off its living bone marrow and donor bone marrow was then introduced. Some were given sick bone marrow while others were given cured bone marrow. Those who were given sick bone marrow got sick and eventually died from AML while those with cured bone marrow survived. It is safe to say that bone marrow transplants could be the most effective way to ensure the survival of AML victims.

Through numerous other experiments it was made clear that there was no profoundly significant cure for the disease. Drugs that were used on the mice were unreliable, as some were cured, and others were not with the same dosage. When the mice were treated with A51 and A86, however, apoptosis of AML cells was significant in number. A51 and A86 also have the strongest effect on p53 according to Figure 1F within the article. Based on binding affinity, however, it was likely that A14 would have had the strongest effects.

While there is not yet a cure for AML, or for any type of cancer for that matter, we continue to work towards it. While we do have drugs and therapies that will help aid in remission, there is nothing yet that will cure the disease without a middle step. The research done by Minzel et. al. can aid in the discovery of a cure. It could also aid in the discovery of something to make the suffering of an AML patient less than what it is.

Works Cited

Minzel, W., Venkatachalam, A., Fink, A., Hung, E., Brachya, G., Burstain, I., . . . Ben-Neriah, Y. (2018). Small Molecules Co-targeting CKIα and the Transcriptional Kinases CDK7/9 Control AML in Preclinical Models. Cell, 175(1). doi:10.1016/j.cell.2018.07.045

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