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Leukemia Research Paper

The way drugs work on such a base level is truly fascinating. I never thought to look deeper than you take a couple Advil's and then your headache magically goes away. It's baffling to think that so many factors go into how a drug affects your body through your individual cells.

The chosen study on leukemia sought to test different drugs for their ability to affect specific proteins in the cell cycle involved in signaling the cell for apoptosis. The first checkpoint in the cell cycle screens for defective DNA and based on the results will lead to proliferation of the cell or apoptosis. The screening process is complicated and has multiple factors but mainly consists of the p53 protein getting signals from gamma H2AX after it is phosphorylated as "a reaction to DNA double-strand breaks (Wikipedia, 2018)." The p53 protein can also be triggered by being phosphorylated by KC1 alpha. This sets the cell on the path to apoptosis and prevents the damaged cell from replicating and further harming the individual.

The KC1 alpha protein is a kinase which means it adds phosphate groups. Kinase are known to "play an essential role in almost all cellular processes" which makes it no surprise that they play a role in leukemia (Schittek and Sinnberg, 2014). The study tested six inhibitors of CK1 alpha on mice with AML (acute myeloid leukemia). They were testing these inhibitors for their ability to bind to the CK1 alpha complex and their effectiveness at promoting apoptosis through the p35, beta-catenin, and gamma H2AX proteins and inhibiting proliferation through the CDK7/CDK9 proteins.

The results of this study indicated two drugs that showed promise in their ability to rectify leukemia. These two drugs were A51 and A86 both of which exhibited a strong binding affinity for the KC1 alpha complex suggesting that the drugs would show greater effects. This proved to be true in this case with both drugs having major inhibiting effects on CDK7 and CDK9 as shown in the western blot analysis of figure 4E. Inhibitors A51 and A86 also showed an increase in the number of p53, beta-catenin, and gamma H2AX proteins all of which are involved in cell apoptosis. This was emphasized in figure 2A with A51 and A86 having the most increase in apoptosis. The study further tested A51 and found it specifically targeted AML cell leaving healthy cells unaffected. Figures 2D and 2F exemplified the inhibitors overall effect on the health of the mice with pictures of organ tissues such as the spleen showing increased resemblance to that of a healthy mouse. The survival rate of mice with this treatment, however, was around 50 percent.

The researchers succeeded in finding an effective drug that decreases the effects of leukemia. The drug does what they designed it to do but the reliability of it is questionable with its 50 percent survival rate. I wouldn't say the drug cures AML more so that it decreases the severity of the symptoms. The drug is a good first step towards finding a cure but it definitely needs a little more refining before even considering it for human testing because of said unreliability.

Bibliography

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