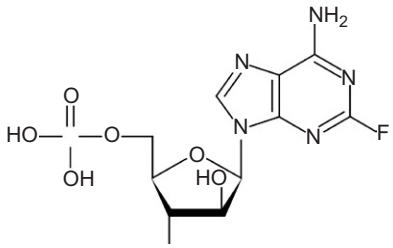
Parameters used for assessing the course of CLL and for assessing response include complete remission, partial remission, stable disease, and treatment failure. These parameters are determined by lymphocyte counts, lymph node size, neutrophil counts, platelet counts, and bone marrow cytology. Surrogate endpoints coupled with these parameters include time to progression (TTP) and progression-free survival (PFS), while the clinical endpoint is overall survival.

The following concerns chemotherapy and the mechanism of drug action. CLL is treated with fludarabine, where greatest efficacy occurs where treatment is with the combination of fludarabine and cyclophosphamide, or fludarabine with rituximab (70). Fludarabine is a purine analogue. After entering the cell, it is phosphorylated to produce fludarabine 5′-triphosphate (71). After phosphorylation, the drug causes damage to the chromosome, that is, cross-linking of DNA (72). In turn, the cross-linking of DNA induces apoptosis. The structure of fludarabine is shown below.



Rituximab is an antibody that binds to CD20. CD20 is a membrane-bound protein of B cells. When this antibody binds to the CD20 of malignant B cells, the result is depletion of these cells (73). The mechanisms of action of rituximab in killing cancer cells include the mechanism of antibody-dependent cell cytotoxicity (ADCC) (74). In a nutshell, in ADCC the antibody serves as an adhesive that positions an NK cell in close proximity with a cancer cell, thus ensuring that the NK cell will kill the cancer cell.

3. *Hairy cell leukemia*

Hairy cell leukemia (HCL) is a rare type of cancer, accounting for about 2% of lymphoid leukemias (75). There are only 500–800 new cases in the United States per year

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