

(37,38). Examples of the CD terminology include CD3, CD4, and CD8. The same type of CD terminology that is used to designate CD4<sup>+</sup> T cells and CD8<sup>+</sup> T cells is also used to identify leukemic cells. Flow cytometry has a routine sensitivity of detection of 0.01%, that is, one leukemic cell (blast) in 10,000 normal cells (39).

The earlier oncology chapters in this book focused on endpoints. In these previous chapters, there was little reason to spend time convincing the reader—to give an example—that prostate cancer cells are different from ovarian cancer cells, that prostate cancer and ovarian cancer occupy different organs in the body, or that prostate cancer inflicts only men and that ovarian cancer inflicts only women. It is obvious that prostate cancer occurs in the prostate gland. And it is self-evident that prostate cancer affects males, and that ovarian cancer affects females.

But things are not as clear-cut with the hematological cancers.

Distinguishing between the various leukemias is a subtle task. Thus, the focus of the present chapter is to distinguish the various

leukemias, and between the various types of MDS. These distinctions are set forth below by the criteria used for diagnosing these cancers, the methods of treatment, and mechanisms of drug action.

The RECIST criteria are generally not used in clinical trials for leukemia or MDS, though Blum et al. (40) raised the potential utility of the RECIST criteria for monitoring lymph node dimensions, in the context of leukemia. Information on parameters, such as endpoints, inclusion/exclusion criteria, that are needed in Clinical Study Protocols for the hematological cancers, can easily be found on the world wide web at [www.clinicaltrials.gov](http://www.clinicaltrials.gov).

## b. Secondary Hematological Cancers

The secondary hematological cancers are those that arise from prior chemotherapy for an earlier-existing disease, such as an earlier-existing cancer. Secondary AML (41,42) and secondary ALL (43,44) are well-documented. Secondary CML also occurs, but only on rare

<sup>37</sup>Zola H, Swart B. The human leucocyte differentiation antigens (HLDA) workshops: the evolving role of antibodies in research, diagnosis and therapy. *Cell Res.* 2005;15:691–4.

<sup>38</sup>Lai L, Alaverdi N, Maltais L, Morse HC. Mouse cell surface antigens: nomenclature and immunophenotyping. *J. Immunol.* 1998;160:3861–8.

<sup>39</sup>Campana D. Minimal residual disease in acute lymphoblastic leukemia. *Hematol. Am. Soc. Hematol. Educ. Program* 2010;2010:7–12.

<sup>40</sup>Blum KA, Young D, Broering S, et al. Computed tomography scans do not improve the predictive power of 1996 national cancer institute sponsored working group chronic lymphocytic leukemia response criteria. *J. Clin. Oncol.* 2007;25:5624–9.

<sup>41</sup>Stone RM, Mazzola E, Neuberg D, et al. Phase III open-label randomized study of cytarabine in combination with amonafide L-malate or daunorubicin as induction therapy for patients with secondary acute myeloid leukemia. *J. Clin. Oncol.* 2015;33:1252–7.

<sup>42</sup>Kayser S, Döhner K, Krauter J, et al. The impact of therapy-related acute myeloid leukemia (AML) on outcome in 2853 adult patients with newly diagnosed AML. *Blood* 2011;117:2137–45.

<sup>43</sup>Kelleher N, Olga G, Gallardo D, et al. Incidence, clinical and biological characteristics and outcome of secondary acute lymphoblastic leukemia after solid organ or hematologic malignancy. *Leuk. Lymphoma* 2015;12:1–6.

<sup>44</sup>Shivakumar R, et al. Biologic features and treatment outcome of secondary acute lymphoblastic leukemia—a review of 101 cases. *Ann. Oncol.* 2008;19:1634–8.