A pediatric oncologist and cancer biologist, Dr. Kastan has concentrated his research on DNA damage and repair, tumor suppressor genes, and causes of cancer related to genetic predisposition and environmental exposures. His discoveries have greatly increased understanding of how cancers develop and how they respond to chemotherapy and radiation therapy. His publications on the role of p53 and ATM in DNA damage signaling are among the most highly cited in the literature of the past two decades. Dr. Kastan was director of the cancer center at St. Jude Children’s Research Hospital and currently serves as the director of the Duke Cancer Institute.

**FIGURE 4A.** Flow cytometry cell sorting dot plot of HCC cell line PLC8024 dually stained for CD133 expression and ALDH activity. With reference to the negative diethylaminobenzaldehyde control as shown in Fig. 3A, the dot plot is divided into four quadrants for CD133+ALDH+, CD133+ALDH−, CD133−ALDH+, and CD133−ALDH−. Cells were collected for each quadrant as gated in the blue boxes. Note that the CD133−ALDH+ subpopulation does not exist because most, if not all, ALDH+ cells are also CD133+.

**FIGURE 2.** Accumulation of miRNAs in colorectal tissues. Northern blot analyses of the (≈21 nt) RNase-processed miRNAs, miR-143 and miR-145, are depicted (A). Northern blots of total RNA from matched normal mucosa (N), adenocarcinoma (T), or adenomatous polyps (P) were probed for the indicated miRNA. 5.8S rRNA is included as a loading control. Relative signal intensities in the RNA blots (B), normalized to the 5.8S rRNA loading control, allow comparison of mature miRNA levels.
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