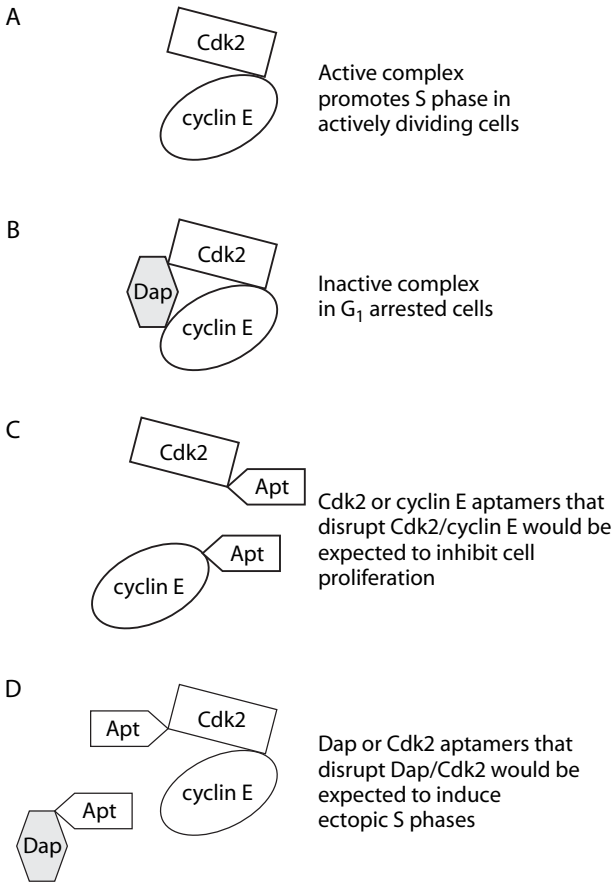

FIGURE 1. Analysis of protein interactions with aptamers. Cdk2, cyclin E, and Dap are well-studied *Drosophila* cell cycle regulatory proteins that have homologs in many other organisms, including vertebrates (de Nooij et al. 1996; Edgar and Lehner 1996; Lane et al. 1996). Cdk2 is a kinase that must be active for cells to enter S phase. Cdk2 must bind to the positive regulatory subunit cyclin E in order for the kinase to be active (*A*). Dap binds to both Cdk2 and cyclin E in G_1-arrested cells and inhibits Cdk2 activity; genetic inactivation of Dap results in ectopic S phases (*B*). Peptide aptamers that bind to Cdk2 or cyclin E and that inhibit the Cdk2–cyclin E interaction would be predicted to inhibit cell proliferation (*C*). In contrast, peptide aptamers that bind to cyclin E or Cdk2 and block their ability to interact with Dap would be predicted to cause ectopic S phases (*D*). This example illustrates the potential for different dominant-acting aptamers that bind to the same protein (e.g., Cdk2) to produce distinct phenotypes and reveal the functions of specific protein–protein interactions.

Protein–Protein Interactions: A Molecular Cloning *Manual*, 2nd Ed., © 2005 by Cold Spring Harbor Laboratory Press, Chapter 30, Figure 1.