A

```
                IV                  V
JNK         NHKNIIGLLNVFTPQKSLEEFQDVYIVMELMDA----- 113
ERK2        RHENIIGINDIIRAP-TIEQMKDVYIVQDLMET----- 108
c-src       RHELKVQLYAVVSEE-------PIYIVTEYMSK----- 343
v-src       RHELKVQLYAVVSEE-------PIYIVIEYMSK----- 343
Cdk1        KDDNIVRLYDIVHSD-----AHKLYLVFEFLDLDLKR- 97
Kin28       QHPNVIELIDIFMAY------DNLNLVLEFLPTDLEVV 93
Srb10       HNKHLTTLVEIFLER------KCVHMVYEYAEHDLLQI 246
```

B

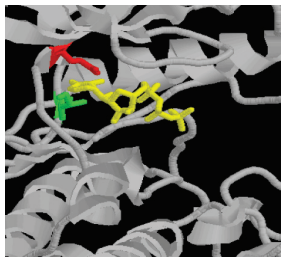

FIGURE 2. (*A*) Sequence alignment of the ATP-binding site of protein kinases that have been successfully engineered to accept ATP analogs. Initial studies (Shah et al. 1997) identified residues in kinase subdomains IV and V for mutation (residues in bold). Residues that provide most successful use of ATP analogs are in subdomain V (residues in bold and underlined). (*B*) Representation of the crystal structure of ERK2, bound to ATP (Zhang et al. 1994). This image was produced using the RasMol program. Bound ATP is shown in yellow. The two residues initially mutated, isoleucine 82 (*green*) and glutamine 103 (*red*), are highlighted. Only mutation of glutamine 103 to glycine allowed the efficient use of ATP analogs by ERK2.

Protein–Protein Interactions: A Molecular Cloning *Manual*, 2nd Ed., © 2005 by Cold Spring Harbor Laboratory Press, Chapter 24, Figure 2.