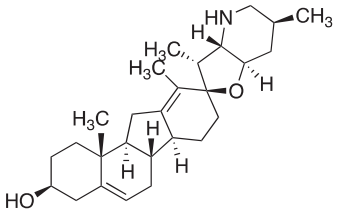
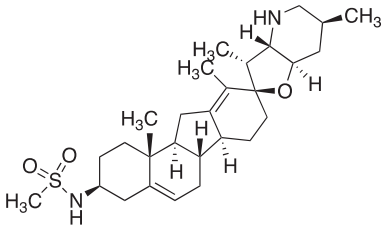
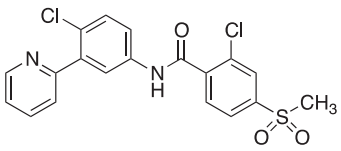
of residual tumors following chemotherapy. NVP-LEQ-506 is being investigated in a phase I trial of patients with advanced solid tumors.[138]



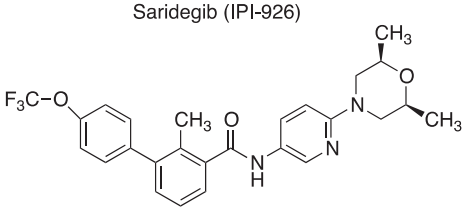
Cyclopamine



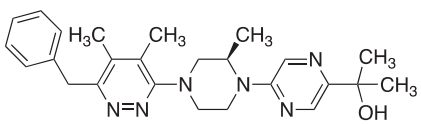
Saridegib (IPI-926)



Vismodegib (GDC-0449, Erivedge®)



Erismodegib (LDE225, NVP-LDE225)



NVP-LEQ506

7.4 MESENCHYMAL STEM CELL-MEDIATED GENE THERAPY FOR CANCER

Mesenchymal stem cells (MSCs) are multipotent stromal cells that can differentiate into a variety of cell types, including osteoblasts, chondrocytes, and adipocytes. After their systemic delivery, MSCs are integrated into the tumor sites mostly by the chemokine receptor CXCR4. This property allows for engineered MSCs targeting a given tumor to potentially be used for *in situ* delivery of therapeutic proteins, genes, or replicating oncolytic viruses.[139]

8 INHIBITORS OF ONCOGENIC PROTEIN–PROTEIN INTERACTIONS

Although drug development has been mainly focused on enzymes and receptors, these targets represent only approximately 1% of the proteins that comprise the human proteome.[140] Furthermore, only a few of the oncogenes encode proteins that are receptors or have enzymatic activities, whereas many others participate in protein–protein interactions (PPIs) that are different from those of noncancer proteins[141] and play essential roles in linking networks that relay oncogenic signals in cancer cells. For instance, mdm2–p53 and CDK4–Rb interactions are involved in neutralizing tumor-acquired mechanisms to evade growth suppression,[142] human papillomaviruses induce tumors through the binding of their