**Chapter Review**

**Chapter 19: Cell Renewal and Cell Death**

19.1

Most cells in adult animals are arrested in the G0 stage of the cell cycle. A few types of differentiated cells, including skin fibroblasts, endothelial cells, smooth muscle cells, and liver cells, are able to resume proliferation as required to replace cells that have been lost because of injury or cell death. However, most differentiated cells do not themselves proliferate but can be replaced via the proliferation of stem cells, which divide to produce one daughter cell that remains a stem cell and another that divides and differentiates. Adult stem cells are used clinically in hematopoietic stem cell transplantation. However, clinical applications of adult stem cells are limited by difficulties in isolating and culturing these cells.

19.2

Embryonic stem cells can be grown in the undifferentiated state while retaining the ability to differentiate into all of the cell types in an organism. Mammals have been cloned by somatic cell nuclear transfer, in which the nucleus of an adult somatic cell is transplanted into an enucleated egg. This opens the possibility of therapeutic cloning in which embryonic stem cells derived from a cloned embryo could be used for transplantation therapy. Alternatively, adult somatic cells can be converted to pluripotent stem cells in culture by four key transcription factors, bypassing the use of embryonic stem cells for transplantation therapy. Somatic cells can also be converted directly into other differentiated cell types by appropriate combinations of transcription factors.

19.3

Programmed cell death plays a key role in both the maintenance of adult tissues and embryonic development. In contrast with the accidental death of cells from an acute injury, most programmed cell death takes place by the active process of apoptosis. Apoptotic cells and cell fragments are then efficiently removed by phagocytosis. The effectors of apoptosis are a family of proteases called caspases, which cleave more than 100 cellular proteins. Members of the Bcl-2 family are central regulators of apoptosis, which either inhibit or promote caspase activation. In mammalian cells, proapoptotic Bcl-2 family members act at mitochondria, where they promote the release of cytochrome *c*, leading to caspase activation. A variety of signaling pathways regulate apoptosis by controlling the expression or activity of members of the Bcl-2 family.