Thalidomide currently is used to treat relapsed and refractory multiple myeloma. The drug also is being actively investigated in patients newly diagnosed with multiple myeloma. The therapeutic applications of thalidomide are expected to grow as clinical trials document its activity in treating other neoplastic disorders and diseases. Healthcare providers need to be well informed of its toxicities and able to identify their signs and symptoms immediately. They must have adequate knowledge about thalidomide administration and provide comprehensive patient education regarding thalidomide and its toxicities to ensure patient safety and compliance.

**Key Words:** angiogenesis inhibitors, teratogens, thalidomide, constipation, fatigue

**Mechanism of Action**

The exact mechanism of the antineoplastic action of thalidomide is unclear. Researchers believe that angiogenesis inhibition, immunomodulation, and cytokine modulation, individually or in combination, likely underlie the drug’s antitumor activity (see Figure 1) (Haslett, Corral, Albert, & Kaplan, 1998; McHugh et al., 1995; Moreira, Friedlander, Shif, Kaplan, & Zaggag, 1999; Moreira et al., 1993; Rowland et al., 2001; Singhal et al., 1999). In multiple myeloma, aside from its antiangiogenic properties (D’Amato, Lenz, Anderson, & Rogers, 2001; Kenyon, Browne, & D’Amato, 1997), thalidomide has several other properties that contribute to its activity, such as immunomodulation (including stimulation of cytotoxic T cell proliferation and induction of interferon-γ and interleukin-12 secretion (Haslet et al.); modulation of cell surface adhesion molecule expression (Geitz, Handt, & Zwingenberger, 1996), direct inhibition of myeloma cell growth and survival via free radical mediated oxidative DNA damage (Parman, 1999), and cytokine modulation, which includes inhibition of production of IL-6, IL-1β, IL-10, and tumor necrosis factor alpha (Moreira et al., 1993).

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