

Effects of Curcumin on Tumor Growth and Muscle Mass in a Mouse Model of Cancer Cachexia

Donna McCarthy Beckett, PhD, RN, FAAN, Katrina Pycha, BS, RN,
and Thomas Berg, BS, RN

Purpose/Objectives: To determine whether a diet containing 1% curcumin, a highly colored botanical food containing compounds that suppress expression of proinflammatory mediators implicated in the pathobiology of tumor-induced muscle wasting, would preserve muscle mass in two animal models of cancer cachexia.

Design: Four-group experimental design.

Setting: A health sciences animal care unit in the midwestern United States.

Sample: 60 female C57Bl/6 mice.

Methods: Mice were subcutaneously inoculated with Lewis lung carcinoma or B16 melanoma tumor cells. Curcumin was mixed into ground standard rodent food. The animals were fed the standard food or food with 1% curcumin for 17 days. Body weight and food intake were monitored and hind limb muscles, spleen, and tumor mass were weighed on day 17. Data were analyzed using two-way (tumor, diet) analysis of variance.

Main Research Variables: Body weight; plantaris, soleus, and gastrocnemius muscles relative to body weight; spleen; and tumor.

Findings: Food intake was not affected by 1% curcumin in the food. Tumor growth caused significant wasting of skeletal muscles in both animal models. Curcumin did not reduce splenomegaly or preserve body weight or muscle mass in either model, but curcumin did significantly reduced tumor mass in mice with B16 melanoma.

Conclusions: Curcumin may not be beneficial in the treatment of cancer cachexia; however, controlled clinical trials of curcumin in patients with cancer are warranted based on evidence of its antitumor effects in animal models.

Implications for Nursing: Patients frequently self-prescribe complementary and alternative substances after diagnosis and during cancer therapy. Answers to patients' questions about purported effects of these substances must include research findings when possible.

Key Points . . .

- ▶ Cancer cachexia causes anorexia, weight loss, and significant wasting of skeletal muscles in patients with advanced or incurable diseases.
- ▶ Animal models are useful for exploring the biologic effects of nursing interventions.
- ▶ Curcumin, a botanical food with anti-inflammatory effects, slowed growth of melanoma tumors in mice.

some cancers (Harris, 2007; Wallace, 2002). Many botanical polyphenolic compounds that suppress COX-2 expression also have been shown to suppress tumor cell growth in vitro and carcinogenesis in laboratory animals (Nishino et al., 2004; Takada, Bhardwaj, Potdar, & Aggarwal, 2004). Several are believed to have chemopreventive potential in humans (Aggarwal & Shishodia, 2006; Surh, 2003; Thomasset et al., 2007).

More than 50% of patients with cancer report using alternative and complementary medicines after diagnosis and during oncology treatment (Vapiwala, Mick, Hampshire, Meta, & Denittis, 2006). Extracts of foods rich in polyphenolic compounds make up a large portion of the self-prescribed complementary and alternative products used to lower the risk of cancer, its recurrence, or to enhance the effectiveness of physician-prescribed antitumor therapies (Adams & Jewell, 2007; Hemalswarya & Doble, 2006); therefore, oncology nurses must ask patients about their use of botanical foods and herbs and answer questions using research data when possible.

Curcumin is a leading polyphenolic agent in clinical studies of botanical compounds for chemoprevention and

Donna McCarthy Beckett, PhD, RN, FAAN, is a professor in the College of Nursing at Ohio State University in Columbus; Katrina Pycha, BS, RN, is a doctoral student in nursing at the University of Wisconsin–Madison; and Thomas Berg, BS, RN, is a nurse resident at the University of Wisconsin Hospital and Clinics in Madison. Mention of specific products and opinions related to those products do not indicate or imply endorsement by the Oncology Nursing Forum or the Oncology Nursing Society. (Submitted September 2007. Accepted for publication October 8, 2007.)

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Botanical foods contain a large and heterogeneous group of biologically active compounds called phytochemicals. Foods rich in polyphenolic compounds, which include the catechins in green tea, the flavanoids in highly colored fruits and vegetables, and curcuminoids in turmeric, have antioxidant qualities and are thought to be health-promoting (Ferrari, 2004). The compounds also reduce expression or activity of cyclooxygenase-2 (COX-2). COX is the rate-limiting enzyme in metabolism of arachidonic acid; increased expression of the COX-2 isoform of COX is responsible for increased synthesis of eicosanoid mediators of the inflammatory response. Most nonsteroidal anti-inflammatory drugs (NSAIDs) work by inhibiting COX activity. A large body of evidence supports findings that suggest increased expression of COX-2 is associated with tumor growth and that chronic ingestion of NSAIDs reduces the incidence of