Response to "Understanding CYP2D6 and Its Role in Tamoxifen Metabolism"

In light of the article "Understanding CYP2D6 and Its Role in Tamoxifen Metabolism" (Smith, 2013), we feel it imperative to comment on the recent, unexpected approval by the U.S. Food and Drug Administration (2013) of the selective serotonin reuptake inhibitor paroxetine as a nonhormonal treatment for menopausal hot flashes. Paroxetine is a strong inhibitor of CYP2D6, the same enzyme system that converts tamoxifen to its active form, endoxifen. Thus, women receiving tamoxifen therapy for hormone-positive breast cancer should be informed of the risk of reducing tamoxifen efficacy with concomitant use of paroxetine to treat their tamoxifen-induced hot flashes (Kaplan & Mahon, 2013). National Comprehensive Cancer Network (2013) guidelines currently recommend against coadministration of strong inhibitors of CYP2D6 with tamoxifen. In addition, a systematic, evidence-based review of interventions to manage hot flashes in women with breast cancer and men with prostate cancer found only two agents likely to be effective, the serotonin-norepinephrine reuptake inhibitor venlafaxine, and gabapentin. Paroxetine was among the many interventions ranked evidence not established (Kaplan et al., 2011).

Clearly, there is an unmet need for safe and effective nonhormonal means to manage hot flashes, particularly in women with breast cancer who are experiencing severe hot flashes because of tamoxifen therapy or premature menopause related to chemotherapy. Oncology nurses should anticipate continued research and more studies to identify safe and effective means to prevent and decrease hot flashes. In the meantime, nurses must educate women on the very real risk of decreasing the effectiveness of tamoxifen when it is combined with paroxetine.

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The authors take full responsibility for the content of the article. No financial relationships relevant to the content of this letter have been disclosed by the authors or editorial staff.

Digital Object Identifier: 10.1188/14.ONF.12