

## Cat. No.: GC61514



## Product Data Sheet

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the clearance-related receptors in its hypocholesterolemic action[2]. Acolbifene (EM-652) shows no agonistic activity on ER $\alpha$  and ER $\beta$  transcriptional function and blocks the estradiol (E2)-mediated activation of both ER $\alpha$  and ER $\beta$ [3]. Acolbifene (EM-652) shows the most potent inhibition of estradiol-stimulated cell proliferation in human breast cancer cells (ZR-75-1, MCF-7, T-47D) and is devoid of any intrinsic estrogenic activity[4].

Acolbifene (ACOL) reduces food intake and strongly decreases cholesterolemia in rats fed a cholesterol-free diet[2]. Acolbifene (ACOL) reduces food intake (16%) and weight gain (45%, mainly fat) similarly in both dietary cohorts[2]. Animal Model: Female Sprague-Dawley rats (n = 42) initially weighing 175-200 g[2].

[1]. Wang T, et al. Recent advances in selective estrogen receptor modulators for breast cancer. *Mini Rev Med Chem*. 2009 Sep;9(10):1191-201. [2]. Christian Lemieux, et al. The selective estrogen receptor modulator acolbifene reduces cholesterolemia independently of its anorectic action in control and cholesterol-fed rats. *J Nutr*. 2005 Sep;135(9):2225-9. [3]. A Tremblay, et al. EM-800, a novel antiestrogen, acts as a pure antagonist of the transcriptional functions of estrogen receptors alpha and beta. *Endocrinology*. 1998 Jan;139(1):111-8. [4]. Sylvain Gauthier, et al. Synthesis and structure-activity relationships of analogs of EM-652 (acolbifene), a pure selective estrogen receptor modulator. Study of nitrogen substitution. *J Enzyme Inhib Med Chem*. 2005 Apr;20(2):165-77.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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