

Achieve a Healthy Estrogen Quotient with Myomin

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Reconstructed reference copy

This document is a reconstructed, condensed reference version based on text recoverable from the indexed copy of the 2012 article. It is **not a facsimile of the publisher's original PDF**. Wording below is substantially paraphrased except for titles, formulas, numerical results, and short labels necessary to identify the reported data.

Original citation: Journal of the American Naturopathic Medical Association. July 2012; 15(4): 10–15.

The article discusses the three principal estrogens—estrone (E1), estradiol (E2), and estriol (E3)—and presents the concept of an “Estrogen Quotient” attributed to Henry Lemon. The author characterizes E1 and E2 as more potent estrogens and E3 as weaker, then proposes that the relative balance among them may be clinically relevant.

Estrogen Quotient (EQ)

$$EQ = E3 / (E1 + E2)$$

The article proposes an EQ greater than 1 as a favorable target, meaning that measured E3 exceeds the sum of E1 and E2. This is presented as a theoretical marker of lower estrogen-related cancer risk. This interpretation reflects the article's thesis and should not be treated as an established contemporary oncology risk model.

Steroid pathways and aromatase

The author reviews steroid-hormone biosynthesis, emphasizing aromatase conversion of androstenedione to estrone and testosterone to estradiol. Myomin is described as a botanical aromatase-reducing formulation intended to decrease E1/E2 production while allowing E3 production through other pathways.

Reported experimental observations

The article cites an earlier in-vivo report in which Myomin administration for 28 days was associated with reductions in aromatase expression in rat endometrial and ovarian tissue. It also describes an animal experiment involving 90 female mice receiving Myomin for 30, 60, or 180 days; reported mean estradiol concentrations were 50.6, 36.6, and 32.4 pg/mL, respectively.

A human study involving 60 postmenopausal women with cysts and fibroids is described as showing a 47.88% reduction in estradiol after 10 days of Myomin. The indexed article text does not provide enough methodological detail here to independently assess randomization, controls, assay methodology, statistical analysis, or publication status.

Selected case reports reproduced from the reported data

Case / test	Before	After	Reported interval
Premenopausal woman, urine E2	196.50 pg/mL	53.21 pg/mL	May → Oct 2011
Postmenopausal woman, saliva E2	6.5 pg/mL	2.4 pg/mL	2 months
Male patient, serum E2	60 pg/mL	25 pg/mL	3 weeks

The article also describes an 80-year-old man with prostate cancer who was taking dutasteride and subsequently used Myomin plus another supplement, Prosta Chi. The reported sequence is shown below; because two supplements were added together and this is an uncontrolled case report, the data cannot establish causation.

Date	Estradiol	PSA	Reported circumstance
9/2011	60	5.0	Before adding Myomin + Prosta Chi; on dutasteride
11/2011	39	2.88	After ~2 months of supplements
1/2012	59	5.6	After stopping supplements for ~2 months
3/2012	39	Not tested	~2 months after restarting
4/2012	31	3.6	~3 months after restarting

Reported EQ case

A 59-year-old woman with breast and thyroid nodules is described after approximately 1½ years of Myomin and Angiostop. The article reports the following saliva results:

Analyte	Result	Article reference range
Estrone (E1)	17.46 pg/mL	5.8–34.2 pg/mL
Estradiol (E2)	1.08 pg/mL	1.0–3.2 pg/mL
Estriol (E3)	18.97 pg/mL	<30.0 pg/mL
EQ = E3/(E1+E2)	1.02	>1.0

Estrogen receptors

The article contrasts estrogen receptor alpha (ER α) and estrogen receptor beta (ER β), presenting ER α signaling as generally more proliferative and ER β signaling as comparatively antiproliferative. It uses this framework to argue that lowering E1/E2 relative to E3 could shift estrogenic signaling in a favorable direction. Modern estrogen-receptor biology is substantially more complex than this simplified model, with effects depending on ligand, tissue, receptor distribution, co-regulators, dose, and disease context.

Factors discussed as contributing to estrogen imbalance

The article identifies hormone replacement therapy, obesity, environmental xenoestrogens, and increased aromatase activity (“hyperaromatization”) as factors that may increase estrogen exposure. Its proposed strategy is to monitor estrogen levels and reduce excessive aromatase-mediated production of E1 and E2.

Important evidence limitation

The article is primarily a mechanistic argument supplemented by animal observations, a briefly described human series, and uncontrolled clinical cases. The reported changes are hypothesis-generating but do not establish that Myomin prevents breast cancer, treats breast cancer, reduces recurrence, or improves survival. The EQ threshold itself is not a standard validated breast-cancer risk or treatment endpoint in contemporary oncology guidelines.

Source note

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