## Crisina, flavona que inibe a aromatase. Seria benéfica no câncer de mama e próstata?

Em 1984: Muitas flavonas inibem a aromatase: apigenina, flavona, flavanona e quercetina sendo a crisina a mais potente. Em 2001 sai trabalho indicando que as flavonas não funcionam "in vivo" devido à baixa absorção e biodisponibilidade. Vide estudo abaixo. Jose de Felippe JUnior

Science. 1984 Sep 7;225(4666):1032-4.

Inhibition of human estrogen synthetase (aromatase) by flavones.

Kellis JT Jr, Vickery LE.

Abstract

Several naturally occurring and synthetic flavones were found to inhibit the aromatization of androstenedione and testosterone to estrogens catalyzed by human placental and ovarian microsomes. These flavones include (in order of decreasing potency) 7,8-benzoflavone, chrysin, apigenin, flavone, flavanone, and quercetin; 5,6-benzoflavone was not inhibitory. 7,8-Benzoflavone and chrysin were potent competitive inhibitors and induced spectral changes in the aromatase cytochrome P-450 indicative of substrate displacement. Flavones may thus compete with steroids in their interaction with certain monooxygenases and thereby alter steroid hormone metabolism.

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## No evidence for the in vivo activity of aromatase-inhibiting flavonoids.

<u>Saarinen N, Joshi SC, Ahotupa M, Li X, Ammälä J, Mäkelä S, Santti R.</u> <u>J Steroid Biochem Mol Biol.</u> 2001 Sep;78(3):231-9.

Source

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**Abstract** 

Measurements of the aromatase-inhibiting and antioxidative capacities of flavonoids in vitro showed that slight changes in flavonoid structure may result in marked changes in biological activity. Several flavonoids such as 7-hydroxyflavone and chrysin (5,7-dihydroxyflavone) were shown to inhibit the formation of 3H-17beta-estradiol from 3H-androstenedione (IC(50)<1.0 microM) in human choriocarcinoma JEG-3 cells and in human embryonic kidney cells HEK 293 transfected with human aromatase gene (Arom+HEK 293). Flavone and quercetin (3,3',4',5,7-pentahydroxyflavone) showed no inhibition (IC(50)>100 microM). None of the requirements for optimal antioxidative capacity (2,3-double bond with 4'-hydroxy group, 3-hydroxyl group, 5,7-dihydroxy structure and the orthodihydroxy structure in the B-ring) is relevant for the maximum inhibition of aromatase by flavonoids. After oral administration to immature rats at a dose of 50 mg/kg body weight, which considerably exceeds amounts found in daily human diets, neither aromatase-inhibiting nonestrogenic flavonoids, such as chrysin, nor estrogenic flavonoids, such as naringenin and apigenin, induced uterine growth or reduced estrogenor androgen-induced uterine growth. The inability of flavonoids to inhibit aromatase and,

consequently, uterine growth in short-term tests may be due to their relatively poor absorption and/or bioavailability.

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