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Bimatoprost mildly stimulates the rate of aqueous humor flow during the day (13%) and at night (14%), its ocular hypotensive action is due primarily to a 26% reduction in the tonographic resistance to outflow. Bimatoprost enhances the pressuresensitive outflow pathway. [1] Bimatoprost displaces [3H]prostaglandin F(2alpha) from FP receptors with K(i) of 6.31 µM. Bimatoprost rapidly mobilizes intracellular Ca(2+) via cloned human FP receptors expressed in human embryonic kidney cells and via native FP receptors in 3T3 mouse fibroblasts with EC(50) of 2.94 μ M and 2.2 μM. [2] Bimatoprost up-regulates Cyr61 mRNA expression in the cat iris. Bimatoprost-induced up-regulation of Cyr61 mRNA expression is not because of the activation of the prostaglandin FP receptor but a different receptor. [3] Bimatoprost consistently evokes responses in different cells within the same tissue preparation, whereas prostaglandin F(2 alpha) and 17-phenyl prostaglandin F(2 alpha) elicites signaling responses in the same cells. Bimatoprost selectively stimulates intracellular calcium signaling in different cat iris sphincter cells. [4]Bimatoprost is the ethyl amide derivative of 17-phenyl trinor PGF2 α , a potent prostaglandin FP receptor agonist. Bimatoprost elicits an immediate, robust spike in [Ca2+] that rapidly decayes back to baseline levels. Bimatoprost possess direct agonist activities at the rat, mouse, and human FP prostanoid receptor. [5]

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