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Testosterone Prevents Cutaneous Ischemia and Necrosis in Males Through Complementary Estrogenic and Androgenic Actions

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Résumé en anglais	OBJECTIVE: Chronic nonhealing wounds are a substantial medical concern and are associated with morbidity and mortality; thus, new treatment strategies are required. The first step toward personalized/precision medicine in this field is probably in taking sex differences into account. Impaired wound healing is augmented by ischemia, and we previously demonstrated that 17β-estradiol exerts a major preventive effect against ischemia-induced skin flap necrosis in female mice. However, the equivalent effects of testosterone in male mice have not yet been reported. We then investigated the role of steroid hormones in male mice using a skin flap ischemia model. APPROACH AND RESULTS: Castrated male mice developed skin necrosis after ischemia, whereas intact or castrated males treated with testosterone were equally protected. Testosterone can (1) activate the estrogen receptor after its aromatization into 17β-estradiol or (2) be reduced into dihydrotestosterone, a nonaromatizable androgen that activates the androgen receptor. We found that dihydrotestosterone protected castrated wild-type mice by promoting skin revascularization, probably through a direct action on resistance arteries, as evidenced using a complementary model of flow-mediated outward remodeling. 17β-estradiol treatment of castrated male mice also strongly protected them from ischemic necrosis through the activation of estrogen receptor-α by increasing skin revascularization and skin survival. Remarkably, 17β-estradiol improved skin survival with a greater efficiency than dihydrotestosterone. CONCLUSIONS: Testosterone provides males with a strong protection against cutaneous necrosis and acts through both its estrogenic and androgenic derivatives, which have complementary effects on skin survival and revascularization.
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