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Five-Days of Salicylate Treatment Does Not Alter Endothelial-Dependent Flow-Mediated Dilation in Women with Endometriosis

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Endometriosis, an estrogen-dependent disorder, is characterized by the presence of endometrium-like tissue outside the uterine cavity. Systemic inflammation including increased production of proinflammatory cytokines regulated by the nuclear factor- κ B (NF- κ B) pathway occurs in endometriosis. Administration of nonacetylated salicylate inhibits NF- κ B activation and improves endothelial function in clinical cohorts, but whether it improves endothelial function in women with endometriosis is unclear. **PURPOSE:** To quantify conduit artery endothelial function, via brachial artery flow-mediated dilation (FMD), in women with endometriosis following inhibition of systemic inflammation with five-days of an oral salicylate intervention. We hypothesized that systemic inhibition of NF- κ B activation would improve conduit artery endothelial function in women with endometriosis. **METHODS:** In five women with laparoscopically-confirmed endometriosis [Mean (SD): Age: 31 (5) yrs; BMI: 24.24 (4.48) $\text{kg}\cdot\text{m}^{-2}$], brachial artery FMD was measured (GE Logiq E) in a randomized double-blind placebo-controlled design. Brachial artery diameter was measured at baseline and post-reactive hyperemia. %FMD is expressed as the percent change in brachial artery diameter in response to reactive hyperemia compared to baseline. Edge detection software (Cardiovascular Suite 4) was utilized and placebo and salicylate treatments were compared using a paired t-test. **RESULTS:** Five-day salicylate treatment did not improve %FMD in women with endometriosis [Placebo: 6.06 (3.80)% vs Salicylate: 3.93 (3.72)%; $p=0.08$]. **CONCLUSION:** Conduit artery endothelial function in women with endometriosis was not improved after the five-day salicylate intervention. **SIGNIFICANCE/NOVELTY:** Inhibition of NF- κ B activation did not impact endothelial function in women with endometriosis. Other mechanisms may be mediating endothelial dysfunction in women with endometriosis.

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