The Effects of Estrogen and Progesterone on Relaxation Rates in Rat Caudal Artery

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During menopause, natural production of the reproductive hormones estrogen (E) and progesterone (P) declines. Additionally, postmenopausal women are at increased risk for cardiovascular complications such as hypertension, which could arise from altered arterial smooth muscle function. Whether reproductive hormones impact cardiovascular health by directly altering vascular muscle contractility is not yet known. To find out if arteriolar contractility changes with the peak and decline in cyclic hormone levels, we tested the effect of natural reproductive hormone levels on arterial muscle relaxation rates using caudal arteries from a retired breeding rat as a model. Experiments were performed during phases of the reproductive cycle corresponding to either high P or high E, which were identified using Pap-stained cells from a swab of the vaginal epithelium. Arterial muscle relaxation rates were examined by recording changes in isometric tension with electrical field stimulation of vascular tissue in a muscle bath under physiological conditions. Relaxation curves analyzed using JSIM modeling software matched a double-exponential decay curve. The changing hormone levels caused no significant difference in the relaxation rates for either the slow or the fast portion of the relaxation curve. These results imply that the protective effect of natural hormone replacement therapy may not directly impact vascular function as a measure to prevent cardiovascular complications.

Research advisor Nancy Pelaez says, “Scientists showed that a natural progesterone cream therapy reduces the risk of heart attack by altering blood vessel function, but synthetic hormones in birth control pills do not have a protective effect. To understand the mechanism, it was important to find out if natural cyclic variation in reproductive hormones alters blood vessel function. By examining cell types from the vaginal epithelium, this research team was able to track changing hormone levels in rats.”