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ESTROGEN DEPRIVATION AGGRAVATES CARDIOMETABOLIC DYSFUNCTION AND INTRACELLULAR CALCIUM DYSHOMEOSTASIS IN OBESE-INSULIN RESISTANCE RATS

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Background: Cardiovascular disease and metabolic syndrome incidence is increased after the onset of menopause, suggesting the vital role of estrogen. Intracellular calcium $[Ca^{2+}]_i$ regulation plays an important role for left ventricular (LV) contractile function. Although either estrogen deprivation or obesity has been shown to strongly affect metabolic status and LV function, the combined effects of estrogen deprivation and obese-insulin resistance on cardiometabolic status and cardiac $[Ca^{2+}]_i$ regulation has never been investigated. We hypothesize that estrogen deprivation aggravates LV dysfunction through the increased impairment of $[Ca^{2+}]_i$ homeostasis in obese-insulin resistance rats.

Methods: Female rats either fed with high fat (HF) or normal (ND) diet for 14 weeks. Then, rats were divided to sham (HFS and NDS) operated or ovariectomized (HFO and NDO) groups. Six weeks after surgery, metabolic status, %LV fractional shortening (%LVFS) and $[Ca^{2+}]_i$ transients in isolated cardiomyocytes were determined.

Results: NDO, HFS and HFO rats had obese-insulin resistance. Although both NDO and HFS had markedly reduced %LVFS and decreased $[Ca^{2+}]_i$ transient amplitude and decay rate, HFO had the most severe impairments (Figure), indicating that estrogen deprivation had the strong impact on abnormal LV function through $[Ca^{2+}]_i$ regulation.

Conclusions: In obese-insulin resistance rats, estrogen deprivation severely aggravates LV dysfunction via increasing an impairment of $[Ca^{2+}]_i$ homeostasis.