The effect of zero-glucose on uterine contractility

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Uterine contractility issues, such as preterm or dysfunctional labours, remain one of the primary obstacles to female and neonatal health. The mechanisms of myometrial contraction and its impairment need to be better elucidated. As contractions produce ischemia, glucose delivery to the myometrium becomes restricted. A direct cause-and-effect relationship between glucose depletion and muscle function impairment remains to be established in the myometrium. We investigate this, and compare effects under hypoxic and depolarized conditions. Myometrial strips from pregnant and non-pregnant C57BL6 mice were equilibrated in oxygenated physiological saline (pH7.4) at 37°C. Contractile activity was measured isometrically and the effects of zero-glucose, zero-glucose and hypoxia (N2) and zero-glucose on the response to high K⁺ (40 mM) were tested. N is number of mice. The amplitude of spontaneous contractions decreased significantly (t-test) to 48±7% (n=14) of control in pregnant and 82±3% (n=8) in non-pregnant mice. This inhibition was significantly more pronounced with hypoxia and zero-glucose; 16±10% pregnant (n=9) and 17±6% non-pregnant (n=10). The peak and plateau contraction to high K⁺ were also significantly reduced by zero-glucose, (21±4% and 25±1%., in pregnant mice) (n=8). Our results demonstrate that zero-glucose significantly inhibits contraction, irrespective of how it is produced. The results indicate that depletion of glucose *in vivo* will be a contributing factor to the pathway underlying contractility related disorders. The mechanism of its effect requires elucidating.

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