EFFECT OF VITAMIN E ON THE BIOCHEMICAL AND CONTRACTILE RESPONSE OF RABBIT BLADDER SMOOTH MUSCLE TO HYDROGEN PEROXIDE

Aim of Study
There is increasing evidence that ischemia/reperfusion is a major etiological factor in the progression of bladder dysfunction after partial outlet obstruction, and that part of the damage is due to the generation of free radicals and the resultant membrane peroxidation. The objective of this investigation was to determine the direct effects of oxygen radicals (H$_2$O$_2$) on the contractile responses of rabbit bladder smooth muscle to various forms of stimulation and determine if pre-treatment with the antioxidant Vitamin E protects the bladder from oxidative damage.

Methods
A total of 64 male New Zealand White rabbits were separated into group 1 (28 rabbits) and group 2 (36 rabbits). Group 1 was fed a normal diet whereas group 2 was fed a diet high in vitamin E. After 3 weeks, each rabbit was euthanized and 8 isolated strips of bladder detrusor placed in individual 15 ml baths containing oxygenated Tyrode’s solution. The dose-response to H$_2$O$_2$ on the contractile responses to field stimulation (32 Hz, 80V, 1 ms), carbachol (20M), and KCl (120mM) was determined. At the end of the experiment all strips were frozen and stored at –70 °C for analysis of malondialdehyde (MDA) as a measure of peroxidation.

Results
H$_2$O$_2$ produced a dose dependent decrease in contractile responses to all forms of stimulation. Vitamin E had no effect on the dose response of FS or carbachol to H$_2$O$_2$ whereas there was a significant protective effect on the response to KCl. In control rabbits, H$_2$O$_2$ stimulated a dose-dependent increase in MDA levels. In the vitamin E group, H$_2$O$_2$ had no effect on MDA levels (Figure).

Conclusions
Although vitamin E prevented the peroxidation of detrusor strips exposed to H$_2$O$_2$, it had no effect on the level of inhibition of the contractile responses to both field stimulation and carbachol, and only a modest protection of the contractile response to KCl. Thus, MDA analysis can not be used as a biochemical marker for the contractile damage caused by H$_2$O$_2$.

Reference