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Abstract Reproduction Form B-1

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Institution City Country: Boston University School of Medicine and Boston VA Medical Center, Boston, Massachusetts, USA.

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Title (type in CAPITAL LETTERS): **HYPOXIA-REOXYGENATION AFFECTS FEMALE BLADDER SMOOTH MUSCLE CONTRACTILITY TO A GREATER EXTENT THAN THE MALE**

Aims of study: Recent studies have shown that bladder oxygen tension plays an important role in regulating bladder smooth muscle contractility. The aim of this study was to determine the effects of hypoxia and hypoxia-reoxygenation on bladder smooth muscle contractility in the male compared with the female rabbit. The role of the cyclooxygenase pathway in hypoxia and reoxygenation-induced changes in detrusor smooth muscle contractility was also investigated.

Methods: Strips of bladder tissues from male (n=6) and female (n=6) New Zealand white rabbits were studied in the organ bath containing physiologic solution. Reactivity of bladder strips to electrical field stimulation (EFS, 10 V, 0.8 msec., 0.5 to 40 Hz) and carbachol (10-9 to 10-4 M) was studied while tissues were exposed to normoxic solution (gassed with 21% O_2, 5% CO_2, 74% nitrogen for 30 min.), hypoxic solution (gassed with 2% O_2, 5% CO_2, 93% nitrogen for 30 min) and then reoxygenated solution (gassed with 95% O_2, 5% CO_2 for 30 min). Hypoxia and reoxygenation-induced changes in the contractility of bladder strips were recorded before and after treatment with indomethacin.

Results: Hypoxia inhibited carbachol and EFS-induced contraction of bladder strips from the male and female animals. Tissue reoxygenation following hypoxia led to further impairment of EFS-stimulated contraction while causing supersensitivity to carbachol in both sexes. Both reoxygenation-induced impairment of EFS-stimulated contraction and supersensitivity to carbachol were significantly greater in the female animals compared with the male animals. Treatment with indomethacin, a cyclooxygenase inhibitor, did not affect EFS-induced contraction of tissues exposed to normoxia, hypoxia and reoxygenation. However, indomethacin increased reactivity to carbachol of normoxic bladder strips but not in tissues exposed to hypoxia and reoxygenation. Indomethacin-induced supersensitivity to carbachol in tissues exposed to normoxia was similar to that caused by hypoxia-reoxygenation.

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Conclusions: These studies show that hypoxia-reoxygenation affects detrusor smooth muscle contractility to a much greater extent in female than in male animals. Hypoxia-reoxygenation induced changes in detrusor smooth muscle contractility may be mimicked in some respects by administration of indomethacin. This may be due to blockade of the cyclooxygenase pathway and consequent overproduction of lipooxygenase products such as leukotrienes.