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**Title:** NITRIC OXIDE(NO) INDUCES [45Ca2+] INFLUX VIA L-TYPE VOLTAGE-DEPENDENT Ca2+ CHANNELS (VDCCs) ACTIVATION IN BLADDER SMOOTH MUSCLE CELLS

**Aims of Study and Methods:**

Numerous studies have indicated that $Ca^{2+}$ transient in smooth muscle cells is mediated by $Ca^{2+}$ entry through L-type VDCCs. On the other hand, NO is assumed to induce $Ca^{2+}$ influx into neuronal and peripheral cells prior to generating $Ca^{2+}$-dependent intracellular functions including neurotransmitter releases. In the present study, functional involvement of VDCCs on NO-induced [$^{45}Ca^{2+}$]influx was investigated using rat urinary bladder smooth muscle cells in primary culture.

**Results:**

S-nitroso-N-acetylpenicillamine (SNAP) induced time- and dose-dependent increases in [$^{45}Ca^{2+}$]influx, which was completely abolished by hemoglobin, suggesting a potential of NO to induce $Ca^{2+}$ influx into bladder smooth muscle cells. The NO-induced [$^{45}Ca^{2+}$]influx was significantly inhibited by membrane stabilizing agents such as tetrodotoxin and lidocaine. Similar patterns of [$^{45}Ca^{2+}$]influx were observed in the cells stimulated by 50 mM KCl. In addition, L-type VDCC inhibitor, verapamil, completely abolished the [$^{45}Ca^{2+}$]influx induced by NO and Bay k-8644, an activator selective to L-type VDCCs.

**Conclusions:**

These results indicate that NO induces [$^{45}Ca^{2+}$]influx into urinary bladder smooth muscle cells is mediated via activation of the L-type VDCCs subsequent to membrane depolarization.