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Title: EFFECTS OF SUPEROXIDE RADICAL AND THE PROTECTIVE EFFECTS OF GINSENG SAPONIN AGAINST SUPEROXIDE RADICAL ON THE WHOLE RAT BLADDER CONTRACTILITY

Aims of Study:

It was proposed that the damaging effects of oxygen could be attributed to the formation of oxygen radicals. This oxygen toxicity results from excessive formation of superoxide radical (O_2^-). Superoxide radical may affect the function of bladder smooth muscle. Ginseng saponin seems to be a nitric oxide donor and have a tissue protective effect. And so we investigated the effects of superoxide radical on rat whole bladder contractility with duroquinone (superoxide radical generator, DQ) and diethyldithiocabamate (superoxide dismutase inhibitor, DETCA), and the effects of ginseng saponin to superoxide radical injury.

Materials and Methods:

Isometric pressure changes of isolated rat whole bladder were recorded in an organ bath using a pressure transducer. The acute effects of DQ (0.01, 0.1, 1mM) and DQ preincubated with DETCA (3mM) were assessed on resting pressure, electrical field stimulation (EFS), and bethanechol (0.05mM), ATP (2mM), and KCl (127mM) induced contraction. The acute effects of DQ and DQ preincubated with DETCA on the responses of the bethanechol in the presence of 0.1mM sodium nitroprusside (SNP) and Korean ginseng saponin (0.75mg, KGS, known to be nitric oxide donor) were investigated for prevent the radical injury. To elucidate the action mechanism of KGS, N-omega-nitro-L-arginin (1mM. L-NAME) was added to bath before KGS was applied to the bathing medium.

Results:

The resting pressure of the whole bladder was not changed by DQ and DQ preincubated with DETCA. EFS-induced contractility was not attenuated by pretreatment with DQ, but attenuated by pretreatment with 1mM DQ preincubated with DETCA ($p<0.05$, n=13). Bethanechol-induced contractility was attenuated by pretreatment with 1mM DQ, but in the presence of DETCA, attenuated by pretreatment with 0.01mM DQ ($p<0.05$, n=20). ATP-induced contractility was attenuated by pretreatment with 0.01mM DQ ($p<0.05$, n=10), but in the presence of DETCA, attenuated by pretreatment with 0.01 mM DQ ($p<0.01$, n=10). KCl-induced contractility was attenuated by pretreatment with 0.1mM DQ ($p<0.05$), but in the presence of DETCA, attenuated by pretreatment with 0.01mM DQ ($p<0.01$, n=10). In the presence of SNP, bethanechol-induced contractility was not attenuated by pretreatment with DQ and DQ preincubated with DETCA. In the presence of KGS, bethanechol-induced contractility was not attenuated by pretreatment with DQ, but in the presence of DETCA, attenuated by pretreatment with 0.1mM DQ ($p<0.05$, n=8). In the presence of L-

NAME and KGS, bethanechol-induced contractility was attenuated by pretreatment with 0.1mM DQ, but in the presence of DETCA, attenuated by pretreatment with 0.01mM DQ ($p<0.05$, $n=8$).

Conclusion:

It is suggested that superoxide radical affect the function of bladder by impairment of contraction and KGS may have preventive effects against superoxide radical.