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Increase of Large Conductance Ca^{2+}-Activated K^+ (Maxi-K) Channel Activities by Nitric Oxide (NO) is due to Destabilizing the Long Closure State

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We have investigated whether NO affects the activities of the rat brain Maxi-K channels reconstituted into the lipid bilayer. In order to introduce NO, we utilized an antibiotic, streptozotocin (STZ), which releases NO upon illumination. While adding STZ itself did not affect the channel activity, turning on the light in the presence of STZ induced an increase in the open probability (Po) of the channel. The average percentage of increase was 160.18±136.36 (mean±SD, n=10) (2.5-fold increase). The increase in the Po was also observed with other kinds of NO-donors such as sodium nitroprusside (52.53±4.93%, n=3) and S-nitroso-N-acetylpenicillamine (82.03±49.24%, n=3). The degree of activity increase depends on the initial Po value. Namely, the activity increase was greater when the initial Po was lower and vice versa. The distributions of open and closed time of single channel activities were well-fitted with two and three exponential curves, respectively. After NO introduction, the longest mean closed time became significantly shorter (n=2), suggesting the NO effect is to destabilize the long closure state. These results suggest that the Maxi-K channel might be one of the direct targets of NO, a newly recognized neurotransmitter in brain.