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NITRIC OXIDE (NO) DIRECTLY ACTIVATES CALCIUM-ACTIVATED POTASSIUM CHANNELS FROM RAT BRAIN RECONSTITUTED INTO PLANAR LIPID BILAYER

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Nitric oxide (NO) has been reported to have many roles in vivo ranging from the neurotransmitter in brain to the relaxant in smooth muscles. Recently, using inside-out patches, Bolotina et al. (1) showed that relaxing effect of NO is aortic smooth muscle is through direct activation of Ca2+-activated K+ channels (maxi-K), resulting in hyperpolarization.

Since the cellular function of NO in brain is unknown, we have investigated whether NO affects the activity of maxi-K channel from rat brain using the lipid bilayer reconstitution method, which would provide well defined environment for channels free from many modulators and effectors. Maxi-K channels were identified by their calcium-dependency and large conductance (220 pS). In order to apply NO, we utilized an antibiotic, streptozotocin (STZ), which releases NO upon illumination (2). When the light was turned on, the channel activity increased about 2-fold within several tens of sec. The effect was reversed several seconds after the light was turned off. Considering the half-life of NO in solution, the effect of STZ can be explained to be due to the released NO. The single channel analysis revealed that the effect of NO is to shorten the mean closed time, while leaving the mean open time unchanged. These results suggest that the maxi-K channels might be one of the targets of NO in brain.

1. Bolotina et al. (1994) Nature 368: 850-853
2. Kwon et al. (1994) FASEB J. 8: 529-533