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The NO-cGMP-PKG signal pathway is involved in the tentacle regeneration of the snail, Helix pomatia

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Nitric oxide (NO) signaling was followed in a snail model for neural regeneration. In the tentacle ganglion of the snail, Helix pomatia the NOS activity was found 120.3±7.2 nmol nitrite/μg protein/h measured by the sensitized Griess method. 0.1mM L-NAME blocked this activity by 85%. After 9 weeks of the tentacle regeneration NOS activity was reappeared, and began to increase up to 894.5±14.5 nmol nitrite/μg protein/h found at the 13th week, and normalized by the 15th week with a 152.5±7.2 nmol nitrite/μg protein/h activity. NOS protein level was changed similar to activity as revealed by immunoblot experiments. cGMP EIA showed that the intact tentacle contains 12.3±4.5 pmol/mg protein cGMP which could be increased by 472±15% and 629±45% when the tentacle homogenate was incubated with 1mM SNAP and 10 μM YC-1, respectively, in the presence of 1mM IBMX. Stimulated cGMP production was inhibited by 10 μM ODQ. cGMP level of the regenerating tentacle was peaking (38.4±8.4 pmol/mg protein) at the 13th week. ELISA assay of the phosphorylated PKG substrate Akt revealed that PKG mean activity was the highest between the 1-5 weeks and the 11-15 weeks of regeneration (212±25%, and 152±20% of control, respectively). Using the PKG inhibitor peptide, 1 μM RKRARKE indicated that other kinases also possesses significant activity at the early phase of regeneration. Our observations supported that the NO-cGMP-PKG signal pathway is involved in neural regeneration.

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