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cells can recover from

photic injury through mechanisms that are not well understood. The TRPM1 cation channel is one candidate mechanism. We compared the differential regulation of Na⁺ and Ca²⁺ channels on the single PR cell. Our previous studies demonstrated that Na⁺ channels in PR cells are

selectively upregulated following light damage, whereas Ca^{2+} channels are downregulated. To investigate their roles in regeneration, we now tested the hypothesis that the upregulation of Na^{+} channels and the downregulation of Ca^{2+} channels contribute to the

recovery from photic injury. Using electrophysiology, we found that two days after light exposure, PRs had higher Na⁺ channel density (activated by high frequency stimulation) and inactivation (latency to recovery from inactivation), but lower Ca²⁺ channel density (activated by

elevated K^+). In contrast, a week after the light exposure, PR cells had reduced Na^+ channel density (resting currents) and increased Ca^{2+} channel density (activated by high K^+). Both the Na^+ channel density (due to the reduction in Ca^{2+} channel density) and the

inactivation of Na⁺ channels (due to upregulation of the Na⁺ channel density) were correlated with PR cell regeneration. Thus, upregulation of Na⁺ channels together with downregulation of Ca²⁺ channels support regeneration. We propose

that the modulation of Na⁺ and Ca²⁺ channels play a role in the cellular processes that are involved in the recovery from light damage in the retina. For example, the PR cells at a certain stage of regeneration can be activated by stimulating multiple Na⁺ channels

(upregulated Na⁺ channels)
and inhibited by stimulating
single Ca²⁺ channels
(downregulated Ca²⁺
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