NCX 667, a novel nitric oxide (NO) donor, effectively reduces intraocular pressure (IOP) in three models of ocular hypertension and glaucoma

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A role for nitric oxide (NO) in reducing intraocular pressure (IOP) has been demonstrated both in animal models and in humans. Moreover, a variety of studies support the important role of NO in regulating IOP homeostasis by enhancing aqueous humor drainage via conventional outflow. NCX 667 is a novel NO-donor recently characterized for efficacy and safety in New Zealand white (NZW) rabbits and non-human primates with ocular hypertension.

NCX 667 at different doses or vehicle (phosphate buffer pH 6.0+cremophor EL 5%+ DMSO 0.3%+BAC 0.2mg/ml) was administered as eye drops to ocular transient hypertensive NZW rabbits, to ocular normotensive NZW rabbits and non-human primates with unilateral laser-induced elevated IOP. IOP was measured before treatment (baseline) and at different time points post-dosing up to 5 hours.

In ocular normotensive and hypertensive rabbits, NCX 667 (1%) topical dosing resulted in a maximal IOP lowering of -5.3 ± 0.8 mmHg and of -11.8 ± 0.6 mmHg compared to vehicle, respectively. The IOP lowering effects were dose-dependent from 0.1 to 1%. In addition, topical application of NCX 667 1% was effective (-7.3 ± 2.3 mmHg vs. vehicle) in the ocular hypertensive eyes of non-human primates. NCX 667 was well tolerated following single topical dosing in rabbits and non-human primates.

In conclusion, NO-donation from NCX 667 leads to IOP lowering in different animal models. Therefore, NCX 667 might represent a valid alternative to current IOP-lowering treatments.