BMC Pharmacology

Poster presentation

Open Access

Knock-in mice expressing cGKIα or cGKIβ selectively in smooth muscle

Silke Weber*, Susanne Feil, Sabine Brummer, Anna-Maria Knorn, Franz Hofmann and Robert Feil

Address: Institut für Pharmakologie und Toxikologie, TU München, Germany

Email: Silke Weber* - Weber@ipt.med.tu-muenchen.de

* Corresponding author

from 2nd International Conference of cGMP Generators, Effectors and Therapeutic Implications
Potsdam, Germany, 10–12 June, 2005

Published: 16 June 2005

BMC Pharmacology 2005, **5**(Suppl 1):P59 doi:10.1186/1471-2210-5-S1-P59

Many effects of NO and natriuretic peptides in smooth muscle cells (SMCs) are mediated by activation of cGMP synthesis and the cGMP-dependent protein kinase type I (cGKI). However, the molecular mechanisms of cGKI action, particularly the specific roles of the two cGKI isoforms, cGKIα and cGKIβ, are not clear. To analyze the functions of cGKI isoforms in SMCs, transgenic mice which express the cGKIα or cGKIβ isoform selectively in SMCs were generated (SM-cGKIα or SM-cGKIβ mice. The cGKIα or cGKIβ encoding sequences were integrated into the SMC-specific SM22α gene by homologous recombination in ES cells. Chimeric mice were generated by injection of correctly targeted ES cells into mouse blastocysts. After germline transmission of the modified alleles, the SM-cGKIα and SM-cGKIβ "knock-in" mouse lines could be established. These mouse lines are currently bred on a cGKI-deficient background to eliminate endogenous cGKI expression. This deletion/rescue strategy should help to clarify the specific functions of cGKIα and cGKIβ in SMCs.