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Transgenic mice with a NO-insensitive soluble guanylate cyclase

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In order to distinguish between the physiological and pathogenic role of heme-dependent activation of sGC at one hand, and basal activity or heme-independent activation at the other hand, we generated knock-in mice with a H105F mutation of the beta1 subunit of sGC. These mice might furthermore be a good model for pathological situations in which the heme is functionally inactive due to oxidation, as has been suggested to be the case in a number of cardiovascular pathologies.

We observed that the mutant mice were viable, although they had a reduced life span and displayed growth retardation. They present a number of gastro-intestinal abnormalities as well as hypertension. Their response to NO donor compounds and NOS inhibition is altered but not absent. These results will be compared with those obtained in sGCalfa1-/- mice, showing testosterone dependent gender specific hypertension, altered cardiac function, altered responses to inhaled NO and to vascular injury.