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sGC stimulators in experimental pulmonary hypertension

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Remodeling of blood vessels shares many similarities such as medial wall thickening, neointimal formation and endothelial dysfunction. The nitric oxide (NO) – soluble guanylate cyclase (sGC) pathway plays a central role in maintaining physiological organ function. Alterations of this pathway have been attributed to be centrally involved in the course of several cardiopulmonary diseases and are subject to the development of new therapeutic agents. Among the most recent approaches stimulators of the soluble guanylate cyclase, e.g. BAY 41–2272 or BAY 63–2521 and activators e.g BAY 58–2667 proved anti-remodeling efficacy in two well established models of chronic pulmonary hypertension (hypoxia and monocrotaline-induced pulmonary hypertension). These compounds improve pulmonary hemodynamics not only symptomatically (as previously shown for many other substances) but also reversed vascular remodeling. Targeting sGC is of considerable interest as stimulators and activators of this enzyme represent a new class of drugs complementary to currently established therapies for chronic vascular disorders (e.g. PDE inhibitors, ACE inhibitors, endothelin receptor antagonists, etc.).