	Route of administration	Experimental model	The effects	Ref.
AVE 0991	Chronic (7 days), orally	*In vitro*, isolated rat cardiac muscle	• decreased perfusion pressure • increased systolic tension • increased heart rate	[31]
		Myocardial infarction (MI) in rat	• attenuation of the decrease in systolic tension after MI • attenuation of the decrease in heart rate after MI • prevention of vasoconstriction • decreased the infarcted area	
	Chronic (5 weeks), orally	Streptozotocin-induced diabetic rats	• suppression of the fall in blood pressure • improvement in hemodynamic parameters (left ventricular contractility and relaxation)	[32]
	Chronic (4 months), orally	Model of atherosclerosis, apolipoprotein E knockout mice	• inhibition of atherogenesis • lack of change in the level of cholesterol and triglycerides	[33]
	Chronic (3 days), orally	Model of endothelial dysfunction, salt-fed rats	• curtailment of endothelial dysfunction • restoration of vasodilator responses to both acetylcholine and histamine • lack of significant effect on arterial blood pressure	[34]
	Preincubation	*In vitro*, isolated aortic rings from rats on a high-salt diet	• restoration of vasodilator responses to both acetylcholine and histamine • enhancement of NO availability • reduction of oxidative stress	
	Chronic (4 weeks), orally	Myocardial infarction in rats	• reduction in cardiac muscle hypertrophy • reduction in infarct size • elevation in left ventricular ejection fraction and left ventricular fractional shortening • attenuation of collagen I and III, TGF-β1 (transforming growth factor-beta1) and TNF-α expression	[35]
	Acute, subcutaneously	Ischemia-induced acute renal injury in mice	• suppression of renal impairment • decreased creatinine level • prevention of leucocyte infiltration • prevention of the release of the chemokine CXCL1	[36]
	Chronic (4 weeks), intraperitoneally	Streptozotocin-induced diabetic rats	• attenuation of the increase in serum glucose level • correction of lipid profile (↓TG - triglyceride, ↑HDL – high density lipoprotein) • renal protective effects (decrease in blood urea nitrogen and protein urea)	[37]
		In vitro, isolated heart	• cardioprotective effect (↑LVDP – left ventricular developed pressure, ↓LVEDP - left ventricular end-diastolic pressure)	
	Chronic (4 months), orally	Model of atherosclerosis, apolipoprotein E knockout mice	• inhibition of atherogenesis • increase plaque stability by decreasing number of macrophages and increasing smooth muscle cells plaque content • diminished inflammatory mediators (MCP-1, IL-6, IL-12, SAA) • inhibition of NADPH oxidase expression • reduced expression of co-stimulatory molecules (CD40, CD86 and CD80) on antigen presenting cells	[38]
	Chronic (9 days), intraperitoneally	Deoxycorticosterone-induced hypertensive rats	• dose-dependently decreased mean arterial blood pressure • combination of low doses of aliskiren and	[39]

			AVE0991 produced synergistic blood pressure lowering effect	
	Chronic (28 days), orally	2K1C renovascular hypertensive rats	• restored the baroreflex sensitivity of both bradycardic and tachycardic components • reduced heart weight, thickness of myocardial fibers, number of inflammatory cells and area of collagen deposition in the heart • decreased inflammatory process and tissue area of collagen deposition in the clipped kidney • anti-hypertensive effect	[40]
CGEN-856S	Preincubation	*In vitro*, isolated aortic rings	• NO-dependent vasodilation	[41]
	Perfusion	*In vitro*, isolated heart	• reduction in the incidence and duration of reperfusion arrhythmias • increased postischemic systolic tension • increased coronary flow	
	Acute, intravenously	Spontaneously hypertensive rats (SHRs)	• decreased mean arterial pressure • transient and slight changes in HR (heart rate)	
	Chronic (7/14 days), osmotic minipumps implanted subcutaneously	Isoproterenol-induced hypertrophy, myocardial infarction in rats	• reduced the degree of ISO-induced hypertrophy • attenuated the ISO-induced increase in collagen I, collagen III, and fibronectin deposition • attenuated the MI-induced decrease in systolic tension • decreased infarct area	[42]

Table 1. Angiotensin-(1-7) analogues in the experimental studies.