Heart

5.17 Effects of Chronic Angiotensin II Administration on MicroRNA Expression in Rat Cardiovascular System
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Introduction. Angiotensin II (Ang II) promotes hypertrophy/hyperplasia, inflammation, and fibrosis in the cardiovascular system through a complex regulation of gene expression/protein translation. MicroRNA are key regulators of gene expression and miR-1,miR-21, miR-133a, and miR-208 are involved in cardiovascular pathology.

Aim. To investigate whether chronic Ang II administration modulates miR-1, miR-21, miR-133a, and miR-208 expression in rat heart and aorta.

Methods. Sprague-Dawley rats were treated for four weeks with a continuous administration of Ang II (200 ng/kg/min, n=8) or saline (n=10) by osmotic minipumps. Systolic blood pressure (SBP) was measured by the tail cuff method. At the end of the experimental period, microRNA expression in the heart and aorta was measured by realtime PCR, and myocardial hypertrophy was evaluated by morphometric analysis.

Results. See table.

	Saline	Ang II	P
SBP, mmHg	136.8 ± 3.9	214.3 ± 5.8	<0.0001
Heart			
miR-1/RnuU6, a.u.	3.18 ± 0.26	2.22 ± 0.29	<0.05
miR-133a/RnuU6, a.u.	0.83 ± 0.04	0.66 ± 0.05	<0.05
miR-21/RnuU6, a.u.	1.70 ± 0.13	1.95 ± 0.28	ns
miR-208/RnuU6, a.u.	0.08 ± 0.01	0.15 ± 0.02	<0.05
Heart/BW, mg/g	2.66 ± 0.05	3.34 ± 0.14	<0.01
Nuclear/total cardiomyocyte area	0.039 ± 0.004	0.087 ± 0.007	<0.0001
Aorta			
miR-21/RnuU6, a.u.	0.28 ± 0.04	1.76 ± 0.36	<0.01

Conclusions. Our data demonstrate that in experimental Ang II-dependent hypertension chronic Ang II administration reduces miR-1 and miR-133a expression and induces miR-208 expression in presence of myocardial hypertrophy. Ang II administration stimulates miR-21 expression in rat aorta. In aorta miR-1, miR-133a and miR-208 expression are absent. These data indicate that microRNA expression is tissue-specific, suggesting a different role of microRNA in the cardiovascular system.