Methods 36 Wistar rats were divided randomly into three groups: control group (n=12), angioplasty group (n=12) and Bosentan (BA) with angioplasty group (n=12). Bosentan was administrated del to rats of BA with angioplasty group. The balloon catheter injury was performed on left common carotid artery of rat by imitating the process of angioplasty. After 7 days and 14 days, neointimal area (NIA) and stenosis index (SI) were calculated. Prior to angioplasty and 7 days after balloon injury, serum VEGF were assessed. In another experiment, 65 Wistar rats were divided randomly into three groups: control group, angioplasty group and Bosentan with angioplasty group. Bosentan was administrated to rats of Bosentan (BA) with angioplasty group. The balloon catheter injury was performed on left common carotid artery of rat by imitating the process of angioplasty. The process of neointimal and media hyperplasia was observed and α-action and PCNA expressions were determined.
Results NIA and SI of BA with angioplasty were significantly decreased compared with angioplasty group ($p<0.001$). Serum VEGF level significantly increased 7 days after balloon injury both in angioplasty group and BA group. The increase range in BA with angioplasty group was higher than that of the angioplasty group ($p<0.01$). The serum VEGF level 7 days after injury had linearly negative correlation with SI. In the second experiment, arterial neointima hyperplasia reached delsummit at 28 days in the angioplasty group and 14 days in the BA with angioplasty group. Neointimal and media area of BA with angioplasty at different times (14th, 28th, 45thday) were significantly decreased compared with angioplasty group ($p<0.001$). The rate of PCNA positive cell increased statistically in BA with angioplasty group than in angioplasty group at 14th day ($p<0.01$). The rat of α-actin positive cell increased significantly in BA with angioplasty group compared with angioplasty group ($p<0.01$) at 14th day.
Conclusions Bosentan may be effective on artery restenosis by inhibiting neointimal hyperplasia, increasing the serum VEGF level, reducing the proliferation, migration and transconformation of vascular smooth muscle cells.

Note: Combination of paper e0367 and e0371.

e0051 MIR214 IS UPREGULATED DURING VENTRICULAR REMODELLING POST MI

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Objective MicroRNAs (miRNAs) are endogeneous, single-stranded non-coding RNA molecules about 22 nucleotides long, del regulating target gene expression post-transcriptionally by base pairing with specific binding sites located in the 3' - untranslated regions (UTRs) of downstream target mRNAs. miRNAs play important roles in the regulation of a multitude of physiological functions such as cell differentiation, proliferation, apoptosis and immune response. Recent studies suggest that aberrant expression of miRNAs is associated with cardiovascular diseases. Those miRNAs exhibit unique spatial expression patterns that might become biomarker of diagnosis and target of treatment of ventricular remodelling. In present study, the expression level of miR-214 during ventricular remodelling post MI was detected.
Methods Rats underwent left descending coronary ligation or sham surgery. Rats with MI was assigned to two groups (n=5). Realtime PCR was developed to detect the expression of miR-214 in myocardium and plasma.
Results The expression level of miR-214 in both myocardium and plasma were up-regulated in the 14th and 28th day post MI. Compared to sham group, the expression level of miR-214 in myocardium increased by 33% (1.33 ± 0.12 vs 1.00 ± 0.02, $p<0.01$) in the 14th day and by 88% (1.88 ± 0.08 vs 1.00 ± 0.02, $p<0.01$) in the 28th day post MI. Compared to sham group, the expression of miR-214 in plasma increased by 60% (1.60 ± 0.09 vs 1.00 ± 0.06, $p<0.01$) in the 14th day and by 116% (2.16 ± 0.13 vs 1.00 ± 0.06, $p<0.01$) in the 28th day post MI.
Conclusions The expression level of miR-214 in myocardium and in plasma up-regulated in the progress of ventricular remodelling post MI in rat. The dynamic change of miR-214 may potentially become a new biomarker in ventricular remodelling post MI.

e0052 VALSARTAN REVERSED VASCULAR FIBROSIS THROUGH THE BLOCKADE OF THE AT1-MEDIATED TGF-β/SMAD SIGNAL PATHWAY IN THE FAT-FED, STREPTOZOTOCIN-TREATED RATS

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Objective Angiotensin II (AII) and transforming growth factor-β (TGF-β) are closely involved in the pathogenesis of diabetic complications. The aim of this study was to clarify the role of AII in the regulation of the TGF-β system in diabetic vascular dysfunction.
Methods Male Wistar rats were randomly divided into three groups : normal control, diabetic rats and valsartan group. Diabetes was induced by high-calorie diet for 4 weeks and a single intraperitoneal injection of streptozotocin (STZ) thereafter. The expression of TGF-β1/Smads signalling was analysed by real-time reverse transcriptase-PCR and immunohistochemistry in aorta of three groups.
Results Compared with control group, the expression of both TGF-β I (27.4013 ± 10.49256 vs 15.1254 ± 6.64343, $p<0.01$), TGF-β receptor types II (28.0173 ± 10.22042 vs 10.0561 ± 8.22275, $p<0.01$) and activation of the smad2/3 (31.4029 ± 10.44721 vs 12.8769 ± 6.98547, $p<0.001$) signalling pathway were up-regulated in the vasculature in diabetic rats. Compared with diabetic group, active TGF-β (18.5682 ± 10.29359 vs 27.4013 ± 10.49256, $p<0.05$) and Smad2/3 (20.5209 ± 7.82756 vs 31.4029 ± 10.44721, $p<0.01$) protein levels were reduced in the aorta after the treatment of valsartan.
Conclusions Our results suggest that AT1 receptor antagonist has reversed vascular fibrosis through the blockade of the AT1-mediated TGF-β/Smad signal pathway in the diabetic rats with vascular dysfunction. These observations may del support additional, beneficial effects of angiotensin receptor antagonists observed during del diabetic vascular complications.

Note: n=? to be sent back.

e0053 ELECTROPORATION-MEDIATED ANGIOTENSIN II TYPE 2 RECEPTOR GENE TRANSFECTED INTO RAT CAROTID ARTERIES AND THE EFFECTS OF AT2R GENE TRANSFER ON NEOINTIMAL HYPERPLASIA IN RAT CAROTID ARTERIES AFTER BALLOON ANGIOPLASTY

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Aim To study the effects of Electroporation on the angiotensin II type 2 receptor (AT2R) transfected into rat carotid arteries and study the effects of AT2R gene transfer on neointimal hyperplasia in rat carotid arteries after balloon angioplasty.
Methods Electroporation-mediated AT2R gene transfected into rat carotid arteries after the establishment of rat carotid balloon injury restenosis model. The arteries were harvested at 5 days, 14 days and 21 days after gene transfer. The expression of AT2R in arteries and morphology analysis were evaluated by fluorescence microscope, immunohistochemistry, HE staining and in situ hybridisation.
Results Electroporation-mediated AT2R gene delivered into injured rat carotid arteries significantly up—regulated the levels of AT2R

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