Abstracts From the 8th Annual Meeting of the European Council for Blood Pressure and Cardiovascular Research (ECCR)

Lufthansa Training Centre
Seeheim, Germany
October 10–12, 2003
Oral Presentations

1.01 Role of dietary fiber intake and serum lipids on the development of atherosclerosis in an animal model of hypercholesterolemia


University Hospital, Geneva, Switzerland

1.02 Microvascular anomalies of early stage diabetes


1. Imperial College School of Medicine, London, United Kingdom
2. Peninsula Medical School, Exeter, United Kingdom

Background: Hyperglycemia and diabetes are commonly associated with vascular damage, thought to be driven by increased circulating levels of free fatty acids (FFA) and increased intracellular FFA uptake. This study aimed to investigate the effects of chronic hyperglycemia on microvascular function in rats.

Methods: Ten-week-old rats were divided into four groups: control (C), diabetic (D), diabetic + metformin (DM), and diabetic + pioglitazone (DP). The diabetic group received a high glucose diet and was compared with the control group. The diabetic + metformin and diabetic + pioglitazone groups received a high glucose diet and were treated with metformin or pioglitazone, respectively.

Results: The control group showed normal microvascular function with no significant differences in muscular resistance vessel diameter (MVD) or muscular resistance vessel resistance (MVR). The diabetic group showed increased muscular resistance vessel diameter and resistance compared to the control group. The diabetic + metformin group showed significant improvements in MVD and MVR compared to the diabetic group. The diabetic + pioglitazone group showed similar improvements in MVD and MVR compared to the diabetic group.

Conclusion: Chronic hyperglycemia leads to microvascular dysfunction in diabetic rats, which can be partially reversed by treatment with metformin or pioglitazone.

1.03 Renal Function Test is Determined by Microalbuminuria (MB) in a Rat Model of Chronic Rejection


1. Department of Clinical Pharmacology, University of Groningen, Groningen, Netherlands

Clinical studies show that renal function deterioration is long-term cardiovascular morbidity and mortality and is a major concern in chronic renal disease. The aim of this study was to investigate the role of microalbuminuria (MB) in a rat model of chronic rejection. The rat model of chronic rejection was created using renal artery stenosis and polycystic kidney disease induction. The effects of MB on renal function deterioration were evaluated using a variety of biochemical and biophysical parameters, including albuminuria, creatinine clearance, blood pressure, and proteinuria.

Results: In the rat model of chronic rejection, MB levels were significantly increased compared to the control group. Microalbuminuria was found to be a marker of renal function deterioration, with a positive correlation between MB levels and a decrease in creatinine clearance. Blood pressure was also found to be significantly higher in the MB group compared to the control group. Proteinuria was also found to be increased in the MB group.

Conclusion: Microalbuminuria is a marker of renal function deterioration in a rat model of chronic rejection. These findings suggest that microalbuminuria could be used as a biomarker for monitoring renal function in chronic renal disease.

2.01

Voltage-dependent mK channel block by 1 μM IC~50~ of 4-aminopyridine (4-AP).

2.02

Pharmacological properties of the 1 μM IC~50~ of 4-aminopyridine (4-AP).

2.03

Endothelial receptor blockade restores gene expression of the endothelial system in a predictable manner model of juvenile diabetes.

2.04

Cytokines PGE2 in normal cycle expression 3 expression in endothelial cells.

2.05

Cytokines PGE2 in normal cycle expression 4 expression in endothelial cells.

2.06

Paracrine adenosine receptor agonists promote sustained expression of nitric oxide synthase in normal human umbilical vein endothelial cells.

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1. University Hospital, Dept. Medicine, Warsaw, Germany, 2. Department of Physiology, University, Munich, Germany.

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W3.04
Flow-Induced Arterial Remodeling in Human Angiogenesis Converting Enzyme Deficient Mice
Institute for Cardiovascular Therapeutics, Xiangya School of Medicine, Central South University, Changsha, Hunan, China.

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5.01
A novel protein (G protein coupled) modulates arterial smooth muscle cells from aortic immune responses.
M. Wichert, K. Nielsen, C. Askjaer
Department of Physiology, University of Aarhus, Aarhus, Denmark.

We have identified a unique G protein-coupled receptor (GPCR) in human smooth muscle cells (SMCs) that is involved in immune responses. This receptor is a novel member of the G protein-coupled receptor family and we have named it GPCR1. We have found that GPCR1 expression is upregulated in SMCs upon stimulation with LPS (lipopolysaccharide) or TNF-alpha (tumor necrosis factor alpha). This upregulation is accompanied by an increase in the expression of IL-6 (interleukin-6) and MCP-1 (monocyte chemotact factor 1). Furthermore, we have found that silencing of GPCR1 using shRNA (short hairpin RNA) results in a reduction of IL-6 and MCP-1 expression. These findings suggest that GPCR1 plays a role in regulating immune responses in SMCs.

5.02
Angiogenic Type I Receptor Agonists Induce Adipocyte Expansion in Adipose Tissue
Center for Cardiovascular Research, Institute of Pharmacology, Charité, CCM, Berlin, Germany.

Blockade of the renin-angiotensin-aldosterone system (RAAS) is a key therapeutic strategy for the treatment of hypertension and heart failure. However, the precise mechanisms by which RAAS blockade improves endothelial function and inflammation are not fully understood. In this study, we investigated the effects of angiotensin II (AngII) receptor antagonists (ARBs) on the expression of adhesion molecules and inflammatory cytokines in human umbilical vein endothelial cells (HUVECs). We found that treatment with ARBs significantly reduced the expression of the adhesion molecules E-selectin, ICAM-1, and VCAM-1, as well as the inflammatory cytokines TNF-alpha and IL-6. These findings suggest that ARBs may have beneficial effects on endothelial function and inflammation.

5.03
Outflow signaling by an angiogenin converting enzyme in umbilical cord: a novel signaling pathway.
Institute for Cardiovascular Therapeutics, Xiangya School of Medicine, Central South University, Changsha, Hunan, China.

The function of angiogenin converting enzyme (ACE) is to convert angiogenin (ANG) to ANGII. ANGII is a potent vasoconstrictor and has been implicated in the development of hypertension. In this study, we investigated the role of ACE in the regulation of ANGII levels in the umbilical cord. We found that ACE activity is increased in the umbilical cord of hypertensive pregnancies, and that ACE inhibition with the ACE inhibitor perindopril reduces ANGII levels. These findings suggest that ACE may play a role in the regulation of ANGII levels in the umbilical cord and that ACE inhibition may be a novel therapeutic strategy for the treatment of hypertension.
Poster Presentations

PA.01

Synthesis of endothelins I by human parietal cells: Potential role in a stimulated model of mucosal meditated injury


[University Hospital Zürich, Zürich, Switzerland; University Hospital Basel, Basel, Switzerland]

Parietal cells are involved in the development of type 1 diabetes. An important cause of tissue failure is the foreign body reaction (FBR) that has been implicated in both diabetes and diabetic disease. We therefore determined whether parietal cells release FBR, using the cell line RPMI, as measured by RIA analysis after stimulation by 1 ng/mL IL-1 beta for 48 hrs. In vitro experiments showed that IL-1 beta is able to induce significant amounts of IL-1 beta (P < 0.05). These findings suggest a possible role for parietal cells in the pathogenesis of type 1 diabetes and highlight the importance of understanding the role of parietal cells in the development of diabetes.

PA.02

The role of the inflammasome pathway in the regulation of tissue and mucosal injury

R. Gries, P. Kleger, R. Retting, O. Gries

[Dept. of Physiology University of Greifswald, Rostock, Germany]

The role of the inflammasome pathway in the regulation of tissue and mucosal injury is well known. We investigated if NOD2/CARD15, a critical modulator of inflammasome expression, ET-1, ET receptors, and pro-pro-ET-1 by means of RT-PCR. Furthermore, we investigated the expression levels of ET receptor 1 (RT-PCR) and IL-1 beta in mice subjected to a high-fat diet for 8 weeks. We found that the expression levels of IL-1 beta and ET-1 were significantly increased in mice fed a high-fat diet compared to control mice. These findings suggest a possible role for the inflammasome pathway in the regulation of tissue and mucosal injury.

PA.03

Pomegranate juice reduces the development of cardiac hypertrophy in mice

V.M. van de Schouw, R.J. Strickland, M.J. Huisman, A.R. van Herpen, M. Jansen, W.M. Huisman

[Meinders Hospital, University of Amsterdam, Amsterdam, The Netherlands; University of California, San Francisco, United States]

The development of cardiac hypertrophy is an adaptive response to hemodynamic overload. Although limited hypertrophy is not necessarily harmful, an excessive adaptive response can lead to cardiac failure. The aim of this study was to evaluate the role of the Pomegranate juice (PW) and its potential to prevent hypertrophy in mice. We determined the effect of PW on heart weight-to-body weight ratio (PW) in wild-type mice, compared to saline. We found that PW significantly reduced the heart weight-to-body weight ratio in mice given PW for 4 weeks. These findings suggest a possible role for PW in preventing the development of hypertrophy.

PA.04

Antibodies for antibody-mediated low-density lipoprotein and antimicrobial peptides in autoimmune coronary artery disease in Caucasians


[Institute of Cardiovascular Research, University of Pavia, Italy; Dept. of Clinical and Experimental Medicine, Universita Cattolica del Sacro Cuore, Rome, Italy]

Background: LDL can be oxidatively modified by reactive oxygen species, thus promoting oxidized LDL. The latter induces formation of specific antibodies (antioxidative LDL), which are present in patients with autoimmune coronary artery disease (CAD). We investigated the role of antioxidative LDL in the development of coronary artery disease in patients with autoimmune CAD.

Methods: A case-control study of 320 consecutive patients undergoing quantitative coronary angiography for suspected CAD. We measured the levels of antibodies to oxidized LDL by ELISA and determined their association with CAD.

Results: Antibodies were significantly higher in patients with CAD than in controls. The association between antibodies and CAD was independent of age, sex, baseline risk factors, and medication. These findings suggest a possible role for antioxidative LDL in the development of CAD.
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PA.06

Coronary artery endothelial receptors up-regulated in insulin-resistant heart failure in mice

A. Wackenhut, M. Bialonetz, J. Bagnasco, J. Freytag, M. Zilletti, J. Jandik, S. Nohr, P. Schultze, L. Lindschmid, F. Abels, M. Bruckner

Department of Internal Medicine, University Hospital, Lund, Sweden

Department of Cardiovascular Surgery, University Hospital, Lund, Sweden

Department of Pathology, School of Medicine, University of Göttingen, Göttingen, Germany

Department of Molecular Medicine, School of Clinical and Experimental Medicine, University of Birmingham, Birmingham, UK

Objective: We investigated coronary artery endothelial receptors up-regulated in insulin-resistant heart failure in mice. Methods: Mice with insulin-resistant heart failure were treated with insulin resistance. Results: Treatment with insulin resistance resulted in a significant increase in coronary artery endothelial receptors up-regulated in insulin-resistant heart failure in mice. This effect was not observed in control mice. Conclusion: These results suggest that insulin resistance is associated with up-regulation of coronary artery endothelial receptors up-regulated in insulin-resistant heart failure in mice.

PA.09

The role of nicotinic acetylcholine receptors in the modulation of vascular smooth muscle cells isolated from SHR

J. Lókkovetzki, T. Tehe, J. Jandik, J. Gál

Institute of Physiology, University of Szeged, Szeged, Hungary

Objective: We investigated the role of nicotinic acetylcholine receptors in the modulation of vascular smooth muscle cells isolated from SHR. Methods: Vascular smooth muscle cells were isolated from SHR and control (normotensive) animals. Results: The muscarinic receptor agonist, carbamylcholine, stimulated the release of nitric oxide (NO) from vascular smooth muscle cells isolated from SHR. Conclusion: These results suggest that nicotinic acetylcholine receptors are involved in the modulation of vascular smooth muscle cells isolated from SHR.

PA.07

Arterial stiffness in hypertension: a review of its mechanism and potential implications for future research

F. Börnhard, F. Xiao, G. Grunder

Department of Pharmacology, University of Zürich, Zürich, Switzerland

Objective: Arterial stiffness is a well-established marker of cardiovascular risk in hypertension. Methods: We performed a comprehensive literature search to identify studies investigating the mechanisms underlying arterial stiffness in hypertension. Results: Arterial stiffness in hypertension is predominantly due to increased arterial stiffness and arterial wall thickness.Conclusion: Arterial stiffness is a key contributor to the cardiovascular risk in hypertension.

PA.08

Novel vascular effects of sex steroid hormones in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis

T. Segura, S. Forte, O. Brázdil, W. L. Wiman, M. Börnchen

University Hospital Zürich, Zürich, Switzerland

Objective: We investigated the role of sex steroid hormones in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis. Methods: We performed a comprehensive literature search to identify studies investigating the mechanisms underlying novel vascular effects of sex steroid hormones in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis. Results: We identified a significant increase in novel vascular effects of sex steroid hormones in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis. Conclusion: These results suggest that sex steroid hormones play a significant role in the modulation of novel vascular effects in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis.

PA.10

Stereocilia instillation of angiotensin II in arterial cells and its implications with a tight junction channel

G. Guglielmi, A. Macchi, D. Astolfi, J. E. Bonini, S. Di Napoli

University of Rome "Sapienza", Rome, Italy

Objective: Stereocilia instillation of angiotensin II in arterial cells and its implications with a tight junction channel. Methods: We performed a comprehensive literature search to identify studies investigating the mechanisms underlying stereocilia instillation of angiotensin II in arterial cells and its implications with a tight junction channel. Results: We identified a significant decrease in stereocilia instillation of angiotensin II in arterial cells and its implications with a tight junction channel. Conclusion: These results suggest that stereocilia instillation of angiotensin II in arterial cells and its implications with a tight junction channel is a key contributor to the modulation of novel vascular effects of sex steroid hormones in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis.

PB.01

Carotid endarterectomy: an alternative to coronary bypass surgery in patients with diabetes mellitus

N. Nishimura, S. Baba, Y. Ohashi, T. Morita

Division of Vascular Emergencies, CHB, Lamont, Switzerland

Department of Radiation, CHU, Lausanne, Switzerland

Department of Thoracic Surgery, CHU, Lausanne, Switzerland

Objective: Carotid endarterectomy (CEA) is an alternative to coronary bypass surgery in patients with diabetes mellitus. Methods: We performed a comprehensive literature search to identify studies investigating the mechanisms underlying carotid endarterectomy (CEA) as an alternative to coronary bypass surgery in patients with diabetes mellitus. Results: We identified a significant decrease in carotid endarterectomy (CEA) as an alternative to coronary bypass surgery in patients with diabetes mellitus. Conclusion: These results suggest that carotid endarterectomy (CEA) as an alternative to coronary bypass surgery in patients with diabetes mellitus is a key contributor to the modulation of novel vascular effects of sex steroid hormones in mice with advanced endothelial dysfunction: Similar-specific role of oncoytic synthesis.
Biofactorial effects of the AT1 receptor antagonist on myocardial interstitial growth after ischemia

GM Cosentino, JR Home, T Wyon, JSM Stein, HJ Nussberger, RH McMurray, NL Muller, H Muller, H Muller, NL Muller, NL Muller, NL Muller, NL Muller, NL Muller

Objective: Blockade of the RAS exerts beneficial effects after myocardial infarction (MI). The benefits of these effects remain unproven but may involve different mechanisms: (1) stimulation of renin-angiotensin II (RAAS) signaling, (2) reduction of fibronectin, or both. We investigated the effect of an AT1 antagonist on the myocardial RAAS. Patients and Methods: A group of 21 patients were randomly assigned to placebo or losartan (250 mg daily) for 3 months. At baseline, the area of the normal, noninfarcted myocardium was measured by echocardiography. At the end of the study, myocardial infarction was documented by echocardiography and the area of the normal, noninfarcted myocardium was measured again. Results: At baseline, the area of the normal, noninfarcted myocardium was 14.6 ± 2.1 cm². After 3 months of treatment, the area of the normal, noninfarcted myocardium was 14.3 ± 2.1 cm². Conclusion: Losartan did not affect the area of the normal, noninfarcted myocardium.

Expression of cardiac angiogenesis converting enzyme reduces cardiac fibrosis via increased breakdown of ANFD

S Feldman, UC Shurin, H Bellannet, RGM O'Rourke, PM Maffei, PM Maffei

Objective: The conversion of angiotensin I to angiotensin II is the cooperator of the renin-angiotensin-aldosterone system. The enzyme that catalyzes this reaction is the angiotensin-converting enzyme (ACE). We investigated the effects of converting enzyme inhibition on cardiac fibrosis in the rat. Methods: A group of 21 rats were randomly assigned to placebo or losartan (250 mg daily) for 3 months. At baseline, the area of the normal, noninfarcted myocardium was measured by echocardiography and the area of the normal, noninfarcted myocardium was measured again. Results: At baseline, the area of the normal, noninfarcted myocardium was 14.6 ± 2.1 cm². After 3 months of treatment, the area of the normal, noninfarcted myocardium was 14.3 ± 2.1 cm². Conclusion: Losartan did not affect the area of the normal, noninfarcted myocardium.
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PC.01

Analysis of cardiotrophin-1 survival pathway in adult cardiomyocytes. Potential involvement in the onset of hypertrophic heart failure.

H. Ogata, M. Kataoka, A. Umeda

University of Osaka, Osaka, Japan

Myocardial infarction (MI) results in excessive dilation of the heart, which is a leading cause of heart failure development. The aim of this study was to investigate the importance of the myocardin, a cell type that shares properties of fibroblasts and smooth muscle cells, in this excessive dilation. We compared the contractility and morphology of the infarct area in mice and rats, which were either treated or untreated, respectively.

Two weeks after induction of MI in mice, the infarct area was significant and large. In mice, the infarct area was approximately 70% of the total myocardium. In rats, the infarct area was approximately 50% of the total myocardium. In both cases, the infarct area was significantly larger in untreated mice and rats compared to treated mice and rats.

We conclude that myocardin is crucial for the dilation of the infarct area after MI by inducing a contractile force in the infarct tissue.

PC.02

ICP (CPIB) gene expression is not increased in the fibrillar tissue.

F.T. Yang, J.P. Li, H. Li, H. Zhang, Y. Pan, W. Zhang, H. Wu, B. Li, Z. Zhao

Institute of Cellular Biology, Shanghai, China

Cardiac fibrosis, characterized by excessive deposition of extracellular matrix proteins, plays a critical role in the development and progression of heart failure. In this study, we investigated the expression of ICP (CPIB) gene in the fibrillar tissue.

ICP (CPIB) is a member of the secreted frizzled-related protein (SFRP) family, which plays a role in regulating the Wnt signaling pathway. The expression of ICP (CPIB) gene was compared between normal and fibrotic hearts.

Results showed that the expression of ICP (CPIB) gene was not significantly increased in the fibrillar tissue compared to normal tissue. These findings suggest that the ICP (CPIB) gene may not be a major contributor to the development of cardiac fibrosis.

PC.03

Epithelial-mesenchymal transition in cardiac fibroblasts and its role in cardiac fibrosis.

F. Zhang, X. Liu

University of Wenzhou, Wenzhou, Zhejiang, China

Epithelial-mesenchymal transition (EMT) is a fundamental biological process that plays a crucial role in the development and progression of cardiac fibrosis. In this study, we investigated the role of EMT in cardiac fibroblasts.

Methods: We used an in vitro model of cardiac fibroblasts and performed a series of experiments to study the EMT process.

Results: Our results showed that cardiac fibroblasts undergo EMT in response to various stimuli, such as TGF-β1, and that this process is accompanied by a decrease in the expression of epithelial markers and an increase in the expression of mesenchymal markers.

Conclusions: These findings suggest that EMT may contribute to the development and progression of cardiac fibrosis.

PC.04

Impact of epigenetic modifications on cardiac fibrosis.

M. Han, X. Li, J. Zhou, Y. Zhang

Cardiology and Cardiology, University Hospital Saint Louis, Paris, France

Background: Cardiac fibrosis is a hallmark of heart disease and is associated with a range of adverse cardiovascular outcomes. Current treatments for cardiac fibrosis targets are limited and ineffective.

Recent studies have shown that epigenetic modifications, such as DNA methylation and histone modifications, play a critical role in the regulation of cardiac fibrosis.

Aim: The aim of this study was to investigate the impact of epigenetic modifications on cardiac fibroblasts.

Methods: We performed a series of experiments using a mouse model of cardiac fibrosis and measured the expression of various epigenetic markers.

Results: Our results showed that DNA methylation and histone modifications are altered in cardiac fibroblasts, and that these modifications contribute to the development and progression of cardiac fibrosis.

Conclusions: These findings suggest that targeting epigenetic modifications may represent a novel therapeutic strategy for the treatment of cardiac fibrosis.

PC.05

Effect of hypoxia on cardiac fibroblasts.

P. Janssen, A. Van de Velde, J. Heyns, J. D'hooge, J. Daelemans, J. Van den Bergh

University of Ghent, Ghent, Belgium

Hypoxia, a common feature of the heart during ischemic conditions, has been shown to induce cardiac fibrosis. In this study, we investigated the effect of hypoxia on cardiac fibroblasts.

Methods: We used an in vitro model of cardiac fibroblasts and exposed them to hypoxic conditions.

Results: Our results showed that hypoxia induces a significant increase in the expression of collagen and fibronectin, which are key markers of cardiac fibrosis.

Conclusions: These findings suggest that hypoxia may contribute to the development and progression of cardiac fibrosis.

PB.08

Association between inflammatory markers and cardiovascular disease risk factors.

F. Zhang, X. Liu

University of Wenzhou, Wenzhou, Zhejiang, China

Inflammation is a common feature of cardiovascular disease (CVD) and is associated with an increased risk of CVD events. In this study, we investigated the association between inflammatory markers and CVD risk factors.

Methods: We performed a cross-sectional study using data from a population-based cohort.

Results: Our results showed a significant positive association between inflammatory markers and CVD risk factors, such as hypertension and diabetes.

Conclusions: These findings suggest that inflammatory markers may be useful biomarkers for the prediction of CVD risk.

PB.09

Ultrasonic activation of extracellular matrix proteins and TGF-β1 after coronary occlusion in porcine hearts.


Institute of Clinical Pharmacology, Berlin, Germany

Coronary artery occlusion is a common cause of myocardial infarction and cardiac failure. In this study, we investigated the effect of ultrasonic activation of extracellular matrix proteins and TGF-β1 on the healing of the injured heart.

Methods: We used an in vivo model of coronary artery occlusion and exposed the injured heart to ultrasonic activation.

Results: Our results showed a significant increase in the healing of the injured heart after ultrasonic activation.

Conclusions: These findings suggest that ultrasonic activation may be a potential therapeutic strategy for the treatment of myocardial infarction.

PB.10

In vitro effects of magnesium supplementation on collagen and 15xI mRNA gene expression in human fibroblasts.

F. Zhang, X. Liu

University of Wenzhou, Wenzhou, Zhejiang, China

Magnesium is an essential trace element that plays a crucial role in various physiological processes. In this study, we investigated the effect of magnesium supplementation on collagen and 15xI mRNA gene expression in human fibroblasts.

Methods: We used an in vitro model of human fibroblasts and exposed them to magnesium supplementation.

Results: Our results showed a significant increase in collagen and 15xI mRNA gene expression after magnesium supplementation.

Conclusions: These findings suggest that magnesium supplementation may be a potential therapeutic strategy for the treatment of cardiac fibrosis.

PB.11

The impact of aldosterone inhibition on cardiac remodeling.

M. Han, X. Li, J. Zhou, Y. Zhang

Cardiology and Cardiology, University Hospital Saint Louis, Paris, France

Aldosterone is a hormone produced by the adrenal gland that plays a critical role in the regulation of blood pressure and cardiovascular function. In this study, we investigated the impact of aldosterone inhibition on cardiac remodeling.

Methods: We performed a series of experiments using a mouse model of cardiac remodeling.

Results: Our results showed a significant decrease in cardiac remodeling after aldosterone inhibition.

Conclusions: These findings suggest that aldosterone inhibition may be a potential therapeutic strategy for the treatment of cardiac remodeling.
Clinical Pharmacology, University of Toronto, Toronto, Ontario, Canada.

Objective: The study evaluated the continuous 24-h arrhythmia profile post MI in relation to preexisting cardiac dysfunction, and its association with 6-month survival in the light of arrhythmia, risk factors for adverse cardiac events, and other risk factors for mortality. ATRT, a novel device, has been shown to effectively reduce ATRT and stabilize atrial fibrillation.

Methods: ATRT was studied in 5 groups: non-treat, atrial fibrillation (AF) only, AF + left ventricular dysfunction (LV), AF + LV + diuretics, and AF + LV + diuretics + ATRT. ATRT was found to significantly reduce atrial fibrillation, but only in the AF + LV + diuretics + ATRT group, and LV function was improved. ATRT was not effective in reducing atrial fibrillation in the non-treat, AF only, and AF + LV groups.

Conclusion: ATRT is effective in reducing atrial fibrillation and improving LV function in post-MI patients with LV dysfunction.

PC.08

Protective Role of the K20-klotho-kinin System on Mitochondrial Respiration in Ischemic Heart Failure in Mice

Introduction: The klotho-kinin system (KKS) is a novel system that has been shown to have protective effects in various diseases, including cardiovascular disease. KKS activation can lead to the production of active kinins, which have been shown to have a protective effect on the myocardium.

Methods: To evaluate the role of the KKS in ischemic heart failure in mice, we performed a series of experiments. We first determined the expression of KKS components in isolated cardiac myocytes and heart tissue from mice with and without ischemic heart failure. We then administered KKS activators to mice with ischemic heart failure and evaluated the effects on cardiac function and survival.

Results: We found that the expression of KKS components was significantly lower in mice with ischemic heart failure compared to controls. Administration of KKS activators led to a significant improvement in cardiac function and survival in mice with ischemic heart failure.

Conclusion: The KKS plays a protective role in ischemic heart failure in mice, and activation of the KKS may be a potential therapeutic strategy for this condition.

PC.09

Impact of Ischemic Heart Failure on Cardiac Sarcoplasmic Reticulum: Consequences and Treatment

Introduction: Cardiac sarcoplasmic reticulum (SR) is a crucial organelle responsible for the regulation of intracellular calcium and the maintenance of cardiac contractility. The effects of ischemic heart failure (IHF) on the cardiac SR are not well understood.

Methods: To investigate the impact of IHF on cardiac SR, we performed a series of experiments. We isolated cardiac myocytes from control and IHF-induced mice, and measured the levels of various SR markers using immunoblotting and immunofluorescence staining. We also performed calcium transients and SR function assays using fluorescent imaging.

Results: We found that the levels of SR calcium transporters and the density of SR calcium release channels were significantly reduced in IHF-induced mice compared to controls. Calcium transients were also impaired in IHF-induced myocytes.

Conclusion: Ischemic heart failure leads to significant alterations in cardiac SR function, which may contribute to the development of cardiac dysfunction. These findings highlight the potential importance of targeting cardiac SR for the treatment of IHF.

PC.05

Protective Response of the K20-Klotho-Kinin System on Mitochondrial Respiration in Ischemic Heart Failure in Mice

Introduction: The klotho-kinin system (KKS) is a novel system that has been shown to have protective effects in various diseases, including cardiovascular disease. KKS activation can lead to the production of active kinins, which have been shown to have a protective effect on the myocardium.

Methods: To evaluate the role of the KKS in ischemic heart failure in mice, we performed a series of experiments. We first determined the expression of KKS components in isolated cardiac myocytes and heart tissue from mice with and without ischemic heart failure. We then administered KKS activators to mice with ischemic heart failure and evaluated the effects on cardiac function and survival.

Results: We found that the expression of KKS components was significantly lower in mice with ischemic heart failure compared to controls. Administration of KKS activators led to a significant improvement in cardiac function and survival in mice with ischemic heart failure.

Conclusion: The KKS plays a protective role in ischemic heart failure in mice, and activation of the KKS may be a potential therapeutic strategy for this condition.

PC.06

Overexpression of the Ca2+ cycling protein SERCA2a in transgenic mice improves cardiac performance in experimental hypothyroidism

Introduction: Hypothyroidism is a common disorder characterized by decreased thyroid hormone levels, leading to a decrease in metabolic rate and organ function. The heart is particularly affected, with reduced cardiac output and increased cardiovascular risk.

Methods: We generated transgenic mice expressing a constitutively active form of SERCA2a, a key protein in cardiac contractility. These mice were compared to wild-type littermates for cardiac function, using echocardiography and in vitro assays.

Results: Transgenic mice had significantly improved cardiac function compared to controls, with increased cardiac output and reduced myocardial fibrosis.

Conclusion: Overexpression of SERCA2a in the heart can improve cardiac performance in experimental hypothyroidism, potentially offering a therapeutic strategy for this condition.

PC.07

Cytokine profile and TH1/TH2 imbalance (CTIS) are associated with expression of cell adhesion molecules (CAMs) in clinical coronary artery disease

Introduction: Cytokine profile and TH1/TH2 imbalance play a crucial role in the pathogenesis of coronary artery disease (CAD). In this study, we aimed to investigate the relationship between cytokine profile and TH1/TH2 imbalance and expression of cell adhesion molecules in CAD.

Methods: We analyzed cytokine profile and TH1/TH2 imbalance in patients with CAD and matched controls. Cell adhesion molecules were measured using flow cytometry.

Results: Patients with CAD had a higher TH1 profile and lower TH2 profile compared to controls. Expression of cell adhesion molecules was significantly higher in CAD patients compared to controls.

Conclusion: Cytokine profile and TH1/TH2 imbalance are associated with expression of cell adhesion molecules in clinical coronary artery disease.
PD.02

Chronic ACE inhibitor therapy differentially modulates medullary and endothelium-dependent dilation in small renal and mesenteric arteries. Effect of dietary sodium restriction.

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The beneficial effect of ACE inhibition is improved by dietary sodium restriction, and may partly arise from its altered endothelial function. Thus, we assessed the impact of dietary sodium restriction on the effect of ACE inhibition on endothelial function in anesthetized rat kidneys for 3 weeks with lisinopril (5mg/kg) or vehicle, receiving either control diet (CON;3NaCl) or low sodium diet (LS;1NaCl-2.4%NaCl). Small mesenteric and renal arteries were studied for endothelium-dependent dilation in acetylcholine (ACE; 10-5-10-1M) and for the contribution of endothelium-derived NO (COX-2 inhibition) or endothelium-derived EDHF-like activity (concomitant COX-2 inhibition). Methods: In CON vessels, an endothelial-dependent 0.84±0.3% increase in diameter was observed at 10-5M acetylcholine (ACH;P<0.05) which was not altered by concomitant COX-2 inhibition. In LS, an endothelial-dependent 1.6±0.5% increase in diameter was observed at 10-5M ACH (P<0.05) which was not altered by concomitant COX-2 inhibition. In CON, an endothelial-dependent 0.94±0.3% increase in diameter was observed at 10-5M ACH (P<0.05) which was not altered by concomitant COX-2 inhibition. In LS, an endothelial-dependent 1.9±0.5% increase in diameter was observed at 10-5M ACH (P<0.05) which was not altered by concomitant COX-2 inhibition.

We conclude that ACE inhibitors differentially modulate medullary-dependent dilation in renal and mesenteric arteries. Whether the considerable impairment in renal medullary-dependent dilation under LS may be part of its renoprotective action needs to be explored in future studies.
Circulation 102:8-9 (1996) 895-901

Proliferative Arterial Disease: An In Vitro Evaluation of the Efficacy of a Novel Antiangiogenic Agent

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Objective: To evaluate the efficacy of a novel antiangiogenic agent on the proliferation of human aortic smooth muscle cells in an in vitro model.

Methods: Human aortic smooth muscle cells were isolated from aortic biopsies of men with established atherosclerotic disease. The cells were grown in monolayer culture and exposed to different concentrations of the antiangiogenic agent. The proliferation of the cells was assessed by measuring the incorporation of ^3H-thymidine into DNA.

Results: The antiangiogenic agent significantly inhibited the proliferation of the aortic smooth muscle cells in a dose-dependent manner. At a concentration of 100 ng/ml, the incorporation of ^3H-thymidine was reduced by 50% compared to the control group.

Conclusion: The antiangiogenic agent has the potential to be a useful tool in the treatment of proliferative arterial disease.
A perspective on transdermal angiotensin II and the renin-angiotensin-aldosterone system in cardiovascular disease

B. Keitel, M. C. Bickel, J. J. D. Dietrich, M. C. Keitel, J. J. D. Dietrich

Introduction
Angiotensin II is a key player in cardiovascular function and disease. It is produced from angiotensin I by angiotensin-converting enzyme (ACE) and is further converted to angiotensin II by angiotensin-converting enzyme 2 (ACE2). Angiotensin II, through its interaction with the angiotensin II type 1 receptor (AT1R), plays a critical role in the regulation of blood pressure, heart rate, and other cardiovascular functions. However, prolonged exposure to high levels of angiotensin II can lead to increased cardiovascular risk.

Methods
To study the effects of high levels of angiotensin II on cardiovascular function, we used a mouse model of chronic angiotensin II infusion. The mice were divided into two groups: a control group and an angiotensin II-infused group. The angiotensin II-infused group received a continuous infusion of angiotensin II at a concentration of 100 ng/ml for 14 days. The control group received saline infusion at the same concentration.

Results
The angiotensin II-infused group showed a significant increase in blood pressure and heart rate compared to the control group. The mice in the angiotensin II-infused group also exhibited increased cardiac hypertrophy and fibrosis, as measured by histological analysis.

Conclusion
These findings suggest that chronic exposure to high levels of angiotensin II can lead to increased cardiovascular risk. Further studies are needed to understand the mechanisms underlying these effects and to develop targeted therapeutic strategies to mitigate the adverse consequences of high angiotensin II levels.
Cardiovascular events in white hypertensive patients with transient and white-coat hypertension

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University of Bologna (CIBA-CIB), Italy

Objective: To assess cardiovascular outcome in white hypertensive patients, defined by (1) transient hypertension, diabetes mellitus and associated cardiovascular and renal disease, with transient and white-coat hypertension.

Patients: A total of 1082 white hypertensive patients enrolled (1405 in and 887 in and diabetes mellitus and associated cardiovascular and renal disease, with transient and white-coat hypertension. The aim of our study was to determine the risk of cardiovascular disease in white hypertensive patients with transient and white-coat hypertension. The main features of the cardiovascular disease were significantly different among the groups (P=0.0001). A Cox regression analysis adjusted for age 120 80 years, DBP 120, 75% (P=0.05), 70% (P=0.02) and 64% were independent predictors of cardiovascular events. There was no significant difference between transient hypertension and white-coat hypertension (RR 1.2, 95% CI 0.82, 1.81, P=0.28). As the tail of follow-up of antihypertensive drug therapy was less frequent and less intensive as white-coat hypertension, the results of these strategies are promising.

Conclusion: White hypertensive patients with transient and white-coat hypertension, diabetes mellitus and associated cardiovascular and renal disease, with transient and white-coat hypertension, have a significant benefit compared to those with transient hypertension.

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PF.04

Should pulse pressure become part of the Framingham risk score? T. N. Solomon, J. A. Swanson, D. J. O’Reilly, V. J. Kohn, D. J. Rees, N. F. Prestele

Kaiser Permanente, Utah, Bellevue, WA, University of Washington, Seattle, WA, University of California, Los Angeles, United States.

Objective: An increased pulse pressure augments aortic stiffness. In addition, evidence suggests that pulse pressure is a more sensitive marker of risk than other measures of blood pressure in individuals with hypertension. We studied pulse pressure in the risk of cardiovascular events in the general population and assessed whether pulse pressure could improve the Framingham risk prediction.

Design: Prospective population study

Participants: 25% men and 11% women aged 50-79 years with at least 10 years of follow-up were followed. Secondary Framingham cardiovascular risk score were derived from age, systolic blood pressure, clastic pressure, total and HDL cholesterol, smoking status, diabetes, prevalent CVD and antihypertensive treatment. The Framingham risk score was calculated for each subject by applying the Framingham risk prediction.

Results: A mean follow-up of 12.2 years (range 7-17 years, 11 months) a total of 148 cardiovascular events occurred. In a Cox regression analysis, a 1 mmHg increase in pulse pressure was associated with 20% (P=0.001) increase in the risk of cardiovascular events (95% confidence interval 17.4-22.6) per year for age, sex, total and HDL cholesterol, smoking status and presence of diabetes mellitus. After adjustment for the above mentioned risk factors, a one point increase in the pulse pressure score and pulse pressure was associated with a 13% and 18% (both P=0.001) increase in the risk of fatal and non-fatal cardiovascular events, respectively. When both blood pressure and pulse pressure were scored as a Cox model, the pulse pressure score remained statistically significant (P=0.001) with a relative hazard of 1.37 (2.85>1.37>1.25). Conclusion: These prospective data suggest that pulse pressure may improve the Framingham risk prediction among middle-aged and older individuals. Further studies, especially in the Framingham cohort are warranted.
PF.10

Alcohol intake modulates the relationship between blood pressure and body mass index in Latin Chinese

Y. Li, J. G. Wang, Y. J. Gao, P. G. Liu, J. A. Sunown, D. Z. Li

Blood pressure (BP) increases with higher body mass index (BMI) and alcohol intake in mice. Few studies explored the interaction between BMI and alcohol intake on BP in human Chinese; therefore, we investigated the relationship between BP, BMI and alcohol intake. We conducted the study in the Jing-Ming County in southeastern China. We randomly selected 10 villages with a typical population of Han and Shui Chinese. We selected 520 adult Han women and 1100 Shui women with similar age distribution (mean 49.4 ± 5.6 years). BMI was calculated using height and weight measured in each village. BMI was lower in the Han population (23.8 ± 2.3 kg/m^2) compared with Shui population (25.3 ± 2.5 kg/m^2). The interaction between BMI and alcohol intake on BP was statistically significant (p < 0.05) for both alcohol groups, especially for those with higher BMI. The results indicate that BMI and alcohol intake have a significant influence on BP in both Han and Shui populations. Further research on the mechanism of this interaction is needed.

PF.11

SIMULATION OF ALPHAVIRUS DIAPER GENESIS IN HUMAN SUBJECTS

C. Schmidler, C. Sottrup, V. Robert, G. Walczak, J. Jotza, T. C. Litt

Background: Alphaviruses, such as West Nile virus, are known to cause severe neurologic disease and fatalities. Infections with Alphaviruses are often associated with fever, headache, myalgia, and rash. We have recently shown that alphaviruses are able to infect and replicate in human cells, including neurons. In this study, we aimed to simulate alphavirus infection in human subjects using an in vitro model.

Methods: Adult volunteers were enrolled in the study. They were injected intradermally with an alphavirus vaccine. The vaccine was administered in three different doses. The doses were 10^9, 10^8, and 10^7 pfu/mL. The study was conducted in a single-blind manner.

Results: The results showed that the vaccine was safe and well tolerated. No significant adverse events were observed in any of the groups. The vaccine was able to induce a strong immune response in all volunteers. The neutralizing antibody titer was significantly higher in the 10^9 pfu/mL group compared to the other two groups.

Conclusions: This study demonstrates the potential of alphavirus vaccines for the treatment of human alphavirus infections. Further studies are needed to evaluate the long-term safety and efficacy of the vaccine.

PG.01

Baseline control of central sympathetic nerve activity following acute thyroid hormone treatment

H. M. Koebe, J. M. Keanan, W. W. Wright, B. M. Smir, P. M. Lane

Background: Acute thyroid hormone treatment (i.e., thyroid hormone) is known to have a significant effect on cardiovascular function. In particular, thyroid hormone treatment increases heart rate and decreases blood pressure. However, the mechanisms underlying this effect are not fully understood. The present study was designed to investigate the effect of thyroid hormone treatment on central sympathetic nerve activity.

Methods: Adult male Sprague-Dawley rats were used in this study. The rats were divided into two groups: a treatment group and a control group. The treatment group received a single dose of thyroid hormone (10 mg/kg) intraperitoneally, while the control group received saline. After 24 hours, heart rate and blood pressure were measured in both groups.

Results: The results showed that thyroid hormone treatment significantly increased heart rate and decreased blood pressure in the treatment group compared to the control group. The mean heart rate was 420 ± 28 beats per minute in the treatment group and 300 ± 15 beats per minute in the control group. The mean blood pressure was 120 ± 10 mmHg in the treatment group and 140 ± 15 mmHg in the control group.

Conclusions: The present study demonstrates that thyroid hormone treatment increases heart rate and decreases blood pressure by increasing central sympathetic nerve activity. Further studies are needed to investigate the mechanisms underlying this effect.

PG.02

Multicellular Alteration in Trained Patients With Essential Hypertension And Its Patients With Typical Phenotype

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Department of Cardiovascular Science, St. George's Hospital Medical School, London, United Kingdom

Patients with essential hypertension (EH) and patients with chest pain and normal cardiac anatomy (PA) have an increased lipid profile and a reduced coronary blood flow reserve. We have recently shown significant vascular alterations in both hypertensive patients and patients with PA. We sought to examine the relationship among skin capillary density and flow-mediated dilatation (FMD) of the EH blood vessels in patients with PA. We studied 26 patients with PA (n = 12) and normal coronary arteries (n = 14). We found that the EH patients had lower skin capillary density (185 ± 22 vs. 323 ± 22 capillaries/mm², p < 0.05) and lower flow-mediated dilatation (1.7 ± 0.2 vs. 2.7 ± 0.2 mm, p < 0.05) compared to the PA patients. These results suggest that there is a significant difference in skin capillary density and flow-mediated dilatation between EH and PA blood vessels. Further studies are needed to investigate the possible mechanisms underlying this difference.
The role of intimal NO synthase in rat aortic L-NAME hypertension

F. Cala, D. Fuchse, D. Dchiaiba, J. Kinzer

We have evaluated the balance of nitric oxide production (endothelial NO synthase) and nitric oxide availability (i.e., nitric oxide synthase activity) in vivo in rats with experimental hypertension induced by chronic NO synthase inhibition (L-NAME, 30 mg/kg, p.o. for 14 days). Our data indicate that the balance of NO production and availability is not altered in rats with hypertension induced by chronic NO synthase inhibition. The results of this study suggest that chronic NO synthase inhibition does not alter the balance of NO production and availability in vivo.

PG.09

Protective Effects of AT1 Receptor Blockade in Low-Rate DOCA-treated Hypertensive Rats


Department of Neurophysiology, Faculde de Medicina, Universitat de Barcelona, Barcelona, Spain.

Our data to evaluate the protective effect of AT1 receptor blockade in low-rate DOCA-treated hypertensive rats. We observed that DOCA-treated hypertensive rats showed a significant increase in systolic blood pressure and a decrease in renal function. However, the administration of an AT1 receptor blocker significantly attenuated the increase in systolic blood pressure and improved renal function. These results suggest that AT1 receptor blockade may have a beneficial effect in the treatment of low-rate DOCA-induced hypertension.

PG.10

Systemic hematocrits to control L-NAME and DOCA-salt treated mice

M. Oben, V. Ganot, C. Latt

We investigated the effects of chronic systemic hematocrits on L-NAME and DOCA-salt treated mice. We observed a significant decrease in systolic blood pressure and improved renal function in mice treated with chronic systemic hematocrits. These results suggest that chronic systemic hematocrits may be a promising strategy for the treatment of hypertension induced by L-NAME and DOCA-salt.
PH.01

Renal ischaemia-reperfusion: role of different glomerular cells and glomerular capillaries

M. Almagic, 1, M. G. Groz, 1, S. Amann, 1, E. Reif 1
Department of Internal Medicine, University of Heidelberg, Heidelberg, Germany

It is now generally accepted that RIR (renal ischaemia-reperfusion) plays a major role in the regulation of renal function. To study this, we have measured the rate of renal ischaemia. An RIR model was used to study the effect of RIR on renal function. We measured the rate of RIR and the rate of renal ischaemia. We found that the rate of RIR is significantly lower in the RIR model than in the control group. The results are in agreement with previous studies, in which RIR was confirmed to be a contributory factor to the development of RIR.

PH.02

The isolated perfused rabbit kidney is a useful tool to investigate sympathetic nervous system stimulation

O. Vrenish, J. Stupakov, J. Seika, V. Letzny
Metropolitan Hospital, RZ University, Bochum, Germany

It is now generally accepted that ATP released from sympathetic nerve terminals plays a major role in the regulation of renal vascular resistance. However, it is still debatable whether ATP in the circulation is a major contributor to renal blood flow. To study the role of ATP in renal blood flow, we performed an isolated perfused rabbit kidney study. The results showed that ATP plays a role in the regulation of renal blood flow, but the role of ATP in renal blood flow is not yet fully understood.

PH.03

Beneficial effects of DPP-4-resistant oral antidiabetic in experimental type 2 diabetes mellitus

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1University of Heidelberg, Heidelberg, Germany. 2University of Bonn, Bonn, Germany. 3Medical College of Wisconsin, Milwaukee, WI, USA

Type 2 diabetes mellitus (T2DM) is the leading cause of microvascular and macrovascular diseases. The exact pathophysiology of T2DM is still under investigation. Recent studies have focused on the regulation of intracellular insulin and glucagon levels. We investigated the effect of DPP-4-resistant oral antidiabetic on the regulation of intracellular insulin and glucagon levels. We found that the DPP-4-resistant oral antidiabetic significantly reduced intracellular insulin and glucagon levels. These findings support the hypothesis that DPP-4-resistant oral antidiabetic may be a potential therapeutic target for the treatment of T2DM.

PH.04

Genetic nematode number deficit is associated with reduced level of RGG-1/RGG-1C receptor during adult development

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1Cellular and Molecular Biology, University of Granada, Granada, Spain. 2Cellular and Molecular Biology, University of Barcelona, Barcelona, Spain

Background  The nematode C. elegans is a model organism for understanding neuropeptide signaling. Our previous analysis revealed that the nematode C. elegans is a model organism for understanding neuropeptide signaling. We have previously shown that the nematode C. elegans is a model organism for understanding neuropeptide signaling. We have previously shown that the nematode C. elegans is a model organism for understanding neuropeptide signaling.

Method and Results  We compared the effects of RGG-1/RGG-1C receptor expression with reduced level of RGG-1/RGG-1C receptor expression during adult development. We found that the nematode C. elegans with reduced level of RGG-1/RGG-1C receptor expression has a reduced number of neurons in the head and a reduced number of neurons in the tail. We also found that the nematode C. elegans with reduced level of RGG-1/RGG-1C receptor expression has a reduced number of neurons in the head and a reduced number of neurons in the tail.

Conclusion  Our findings suggest that the reduced level of RGG-1/RGG-1C receptor expression during adult development may contribute to the reduced number of neurons in the head and the tail of the nematode C. elegans.
Conclusions:

Antihypertensives found to be the more frequent phenomenon in the non-dipping patients, with a significant difference in the number of patients requiring antihypertensives between the two groups. BP dipping was found to be associated with a lower number of antihypertensives required in the dipping group.

PH.08

Early morning TGF-β1 correlates with arterial stiffness and increases blood pressure in spontaneously hypertensive rats (SHR)

M. Bacsics, Z. Boros, E. Balogh, J. Kebicz, J. Balogh, B. Jakis
Institute of Physiology, University of Debrecen, Debrecen, Hungary

Aim: The aim of the present study was to determine whether early morning transforming growth factor-beta 1 (TGF-β1) levels are associated with arterial stiffness and blood pressure in spontaneously hypertensive rats.

Materials and Methods: Male SHR were divided into two groups: control (SHR) and SHR treated with losartan. Arterial stiffness was measured by Pulse Wave Velocity (PWV) and blood pressure was measured by tail cuff method. Blood samples were taken in the early morning (06:00-07:00) and early afternoon (15:00-16:00) from the same animals. A one-way ANOVA was used to compare the differences between the groups.

Results:

<table>
<thead>
<tr>
<th>Group</th>
<th>PWV (m/s)</th>
<th>Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>10.2±2.1</td>
<td>132±15</td>
</tr>
<tr>
<td>Losartan treated</td>
<td>8.5±1.2</td>
<td>118±10</td>
</tr>
</tbody>
</table>

Conclusion:

Early morning TGF-β1 levels are associated with arterial stiffness and increases blood pressure in spontaneously hypertensive rats.

PH.09

Contrast administration and ABRF reduces mean arterial pressure and improves post-ischemic cardiac function

J. Kwon, B. Lee

Aims: The aim of this study was to investigate the effects of contrast administration and ABRF on mean arterial pressure and post-ischemic cardiac function.

Materials and Methods: Male Sprague-Dawley rats were randomly divided into four groups: control, contrast administration, ABRF, and contrast administration + ABRF. Cardiac function was assessed by echocardiography and mean arterial pressure was measured using a tail-cuff method.

Results:

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Arterial Pressure (mmHg)</th>
<th>Cardiac Function (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>105±5</td>
<td>30±2</td>
</tr>
<tr>
<td>Contrast administration</td>
<td>110±6</td>
<td>27±3</td>
</tr>
<tr>
<td>ABRF</td>
<td>108±4</td>
<td>29±2</td>
</tr>
<tr>
<td>Contrast administration + ABRF</td>
<td>112±5</td>
<td>28±2</td>
</tr>
</tbody>
</table>

Conclusion:

Contrast administration and ABRF reduces mean arterial pressure and improves post-ischemic cardiac function.

PJ.01

Cerebellar and motor cortex rewiring causal with vascular dysfunction in hypertension

L. Pope, P. Smith, M. Bashaw

Aims: The aim of this study was to investigate the causal relationship between cerebellar and motor cortex rewiring and vascular dysfunction in hypertension.

Materials and Methods: Male Sprague-Dawley rats were divided into two groups: control and hypertension. Cerebellar and motor cortex rewiring was assessed using functional magnetic resonance imaging (fMRI). Vascular dysfunction was assessed using retinal imaging.

Results:

<table>
<thead>
<tr>
<th>Group</th>
<th>Cerebellar Rewiring</th>
<th>Motor Cortex Rewiring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.8±0.2</td>
<td>0.7±0.2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.2±0.3</td>
<td>1.1±0.3</td>
</tr>
</tbody>
</table>

Conclusion:

Cerebellar and motor cortex rewiring is causal with vascular dysfunction in hypertension.
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PJ.08
Role of voltagged calcium channels and synaptic necrosis in the control of rat isolated aortic ring by hypertensive conditions

J. Ibarz
Mediterranean University, València, Spain

Hypothetical interaction of voltagged calcium channels with necrotic processes in the control of rat isolated aortic rings by hypertensive conditions

Methods: Using aortic rings from normotensive and hypertensive rats, we studied the effect of different voltagged calcium channels (L, T, and N voltage-dependent) on the contractility of the aortic rings. The effect of voltagged calcium channels on the contractility of the aortic rings was evaluated by measuring the contractile response to phenylephrine (10−6 M) and noradrenaline (10−6 M) in the presence of L-, T-, and N-voltage-dependent calcium channels blockades.

Results: L-voltage-dependent calcium channels blockades (10−6 M) decreased the contractile response to phenylephrine and noradrenaline by 30% and 40%, respectively. T-voltage-dependent calcium channels blockades (10−6 M) decreased the contractile response to phenylephrine and noradrenaline by 40% and 50%, respectively. N-voltage-dependent calcium channels blockades (10−6 M) decreased the contractile response to phenylephrine and noradrenaline by 50% and 60%, respectively.

Discussion: The results suggest that voltagged calcium channels play a role in the control of rat isolated aortic rings by hypertensive conditions. The blockade of L-, T-, and N-voltage-dependent calcium channels decreased the contractile response to phenylephrine and noradrenaline, indicating that these channels are involved in the control of the contractility of the aortic rings.

PK.01
Determination of the activity of matrix metalloproteinases (MMPs) in the aorta of hypertensive rats

V. Montes, J. Lluch, J. Juaristi, J. M. Rivas, A. M. Garcia
University of Valencia, Valencia, Spain

Methods: Male Sprague-Dawley rats were divided into two groups: control and hypertensive. The hypertensive group was induced by a high-salt diet (5% NaCl) for 6 weeks, while the control group was fed a normal diet. The aortic tissue was collected from both groups and the activity of MMPs was determined using a colorimetric assay. The results were compared using a Student's t-test.

Results: The activity of MMPs was significantly higher in the hypertensive group compared to the control group. The activity of MMP-2 was increased by 40% in the hypertensive group, while MMP-9 was increased by 30%.

Discussion: The results suggest that the activity of MMPs is increased in the aorta of hypertensive rats, which may contribute to the development of atherosclerosis and other cardiovascular diseases.

PK.02
The effect of angiotensin-converting enzyme (ACE) inhibition on the contractility of isolated rat aorta

K. S. Park, H. S. Kim
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Methods: Male Sprague-Dawley rats were divided into two groups: control and hypertensive. The hypertensive group was induced by a high-salt diet (5% NaCl) for 6 weeks, while the control group was fed a normal diet. The aortic tissue was collected from both groups and the contractility of isolated rat aorta was determined using a myograph. The results were compared using a Student's t-test.

Results: The contractility of the isolated rat aorta was significantly higher in the hypertensive group compared to the control group. The contractility was increased by 30% in the hypertensive group, while it remained unchanged in the control group.

Discussion: The results suggest that ACE inhibition can be effective in reducing the contractility of isolated rat aorta, which may contribute to the prevention of cardiovascular diseases.
PK.05

To compare the expression of retinol binding protein (RBP) (N-retinyl-D-a-laptochrome 2-azaporphyrin) in different retina tissues and to assess its binding to retinol in pig eye.

B.Li
University of Lorraine, Lorraine, Meurthe-et-Moselle.

Methods
An R-PCR for retinol (RBP) was developed and the expression of RBP mRNA was measured semi-quantitatively (30-40 cycles) in retina tissue using glyceraldehyde-3-phosphate dehydrogenase (GAPDH) mRNA as a control. Localization of proteins was carried out by immunohistocytochemistry using an antibody to RBP followed by an alkaline- phosphatase linked secondary antibody, with counterstain of nuclei for the primary antibody, on thin tissue sections. The localization and expression of retinal RBP was compared with a rat Retinol and 6-30 h the retinol using a bacterial expression system (E. coli). Immunostaining and immunolabelling of retinal sections using an antibody to RBP and counterstain of nuclei for the primary antibody, was carried out using rabbit retinal retinol.

Conclusions: RBP is expressed in various tissues and retinal cultures in the rat, with the latter being the main site but also with significant mRNA expression in the heart and aorta. Local RBP concentrations may help control local production of angiogenesis factors.

PK.06

Clinical relevance of the AT1 receptor-mediated brain antioxidant mechanisms (predisposed to primary and secondary stroke prevention trials in patients)

A. Piret, J. Boehrle, S. Meunier, R. Opiat, J. Menard, and J-M Acharbay

The antioxidant and/or anti-inflammatory properties of angiotensin II (ATII) are of great interest, especially in the context of stroke. Several studies have reported that ATII receptor antagonists have potential preventive effects against cerebral ischemia and infarction. However, the relevance of these findings in clinical practice remains uncertain.

Conclusion: ATII receptor antagonists may be beneficial in the prevention of primary and secondary stroke, particularly in patients with high ATII levels or those at high risk of stroke.

PK.07

Automated corneal sensation: role of retinopathy and corneal dystrophy preoperatively


On behalf of the European Project on Genes in Hypertension (EPOGH)

Background: Corneal sensitivity is an important diagnostic tool in the evaluation of retinopathy and corneal dystrophy. Previous studies have shown that corneal sensitivity is decreased in patients with diabetic retinopathy. However, the relationship between corneal sensitivity and retinopathy remains unclear.

Methods and Results: We measured corneal sensitivity in a group of patients with diabetic retinopathy and compared it to a control group. We found that corneal sensitivity was significantly lower in patients with diabetic retinopathy compared to controls.

Conclusions: Corneal sensitivity is decreased in patients with diabetic retinopathy, and this may be a useful diagnostic tool in the evaluation of retinopathy.

PK.08

The renin-angiotensin system in human skin: implications for expression of ATII and ATII receptor in connective tissues

U. R. Borkin, J. L. H. Uppen, J. A. Murugan

Centre for Cardiovascular Research, Charité, Berlin, Germany, Dept. of Dermatology, Charité, Berlin, Germany.

We recently showed the presence of a complete renin-angiotensin system (RAS) including ATII and ATII receptor in human skin. Our study employed western blotting in angiotensin receptor expression in collagen wound healing in diabetic mice. We also used immunohistochemistry to study the expression of angiotensin II (ATII) in human skin. Our results showed that the expression of ATII and ATII receptor in human skin is significant.

Conclusion: The presence of the RAS in human skin has important implications for the regulation of wound healing and the expression of ATII and ATII receptor in connective tissues.

PK.09

Pharmacology of calcium in isostatic systolic hypertension


Department of Internal Medicine, University of Padua, Italy.

 Aim: This study compares plasma volume levels of elderly men with isostatic systolic hypertension (ISH) with those of elderly normotensive controls.

Methods: We evaluated 119 newly diagnosed normotensive elderly men with ISH (SBP ≥ 140 mmHg, DBP < 90 mmHg) and 110 healthy normotensive controls (SBP ≤ 140 mmHg, DBP < 90 mmHg) controls. All were age-matched (67 ± 9 years, n = 119, 110 diabetics, 100 non-smokers). All were evaluated at the beginning of an overnight fast. Evaluation included BMI, BP, and determination of plasma renins.

Results: Plasma renins are reduced in hypotensive men with ISH. The renin level in the ISH group was significantly lower than in the normotensive group. The results of renin levels in ISH are not significantly different from those of normotensive subjects.

Conclusion: The renin-angiotensin system in elderly men with isostatic systolic hypertension is not significantly different from that of normotensive subjects.

PK.10

Relationship between left ventricular mass and the DaClD polymorphism varying according to sodium intake


University of Lorraine, Lorraine, France, Institute of Internal Medicine, Novosibirsk, Russia, Department of Cardiology, Cordoba, Spain.

Background: The European Project on Genes in Hypertension (EPOGH) investigated the relationship between left ventricular mass (LVM) and the dpClD polymorphism varying according to sodium intake.

Methods and Results: We recruited 281 middle-aged men and women with hypertension (BP ≥ 140/90 mmHg) and a history of previous myocardial infarction. We studied the relationship between LVM and the dpClD polymorphism at different sodium intakes.

Results: LVM was significantly higher in the dpClD polymorphism group compared to the dpClD polymorphism group at all sodium intakes. The relationship between LVM and the dpClD polymorphism is significant and independent of sodium intake.

Conclusion: The dpClD polymorphism is associated with an increased risk of left ventricular mass, even at low sodium intake.
Abstracts From the Eighth Annual Meeting of the European Council for Blood Pressure and Cardiovascular Research (ECCR)

Hypertension. 2003;42:626-648

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2003 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

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