30th SCIENTIFIC FORUM – INTERNATIONAL CONGRESS OF CARDIOVASCULAR SCIENCES

22nd ANNUAL MEETING OF THE SOUTH AMERICAN SECTION OF INTERNATIONAL ACADEMY OF CARDIOVASCULAR SCIENCES

Hospital Meridional Vitória

Vitória, ES - Brazil, October 17 – 19, 2024
Rua Desembargador José Fortunato Ribeiro, 30 - Mata da Praia, Vitória - ES, 30200-010, Espírito Santo, Brasil.

PROMOTION

SERVCOR/INSTITUTO CARDIOVASCULAR SÃO FRANCISCO DE ASSIS – ICSFA – MG
Truth is Jesus – St. John 14.6

President Director: Prof. Dr. Otoni Moreira Gomes

REDE MERIDIONAL KORA SAÚDE

CLÍNICA CENTROCOR

Otoni Moreira Gomes MD PhD FIACS FESC

Conference Chair	Conference Co-Chair	Conference Co-Chair	Conference Co-Chair
Melchior Luiz Lima MD PhD FIACS	Antoinette Oliveira Blackman MD PhD FIACS FESC	Alexandre Ciappina Hueb MD PhD FIACS	José Airton Arruda MD PhD SCAI EAPCI
Cardiovascular Surgeon	Cardiologist	Cardiovascular Surgeon	Interventional Cardiologist
Centrocor Clinic Ltda.	Centro Universitário de Brasília	Hospital das Clínicas Samuel Libânio	Hospital Meridional
Rua Alfeu Alves Pereira, 60	CEUB	Universidade do Vale do Sapucaí	Rua R. Meridional, 200
Enseada do Suá	SEPN 707/907 Asa Norte	Pouso Alegre	Alto Lage
Vitória, ES 30050-285	Brasília – DF	Minas Gerais	Cariacica, ES 30151-920
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Scientific Program
Message From The Congress President
30th International Congress of Cardiovascular Sciences

Dear Colleagues,
It is with great satisfaction that we announce the 30th International Congress of Cardiovascular Sciences with the 22nd Annual Meeting of the South American Section of the International Academy of Cardiovascular Sciences, organized by the Instituto Cardiovascular São Francisco de Assis, to be held in the city of Vitória, Espírito Santo, Brazil , between October 17th and 19th, 2024. These events aim to stimulate and increase the scientific production of new researchers in the area of human sciences, promoting an environment conducive to the development and dissemination of innovative knowledge.
For three days, internationally renowned experts will come together to share their experiences and the latest advances in cardiology. The program includes keynote lectures, scientific work presentations and interactive sessions, providing a unique platform for learning and knowledge exchange.
Vitória, known for its beautiful beaches and rich cultural heritage, will be the perfect setting for this scientific meeting. In addition to academic activities, participants will have the opportunity to explore the city and enjoy its hospitality and tourist attractions.
Our goal is to create a space where new researchers can present their discoveries, interact with experienced professionals and establish collaborations that further drive research in the area of cardiovascular sciences. We believe that everyone's participation will be fundamental to the success of this congress and the advancement of scientific knowledge.
We count on your presence to make this event a milestone in the field of cardiology. Don't miss this opportunity for professional updating and networking with the main experts in the sector.

Yours sincerely,

Melchior Luiz Lima MD PhD FIACS
President of the 30th International Congress of Cardiovascular Sciences

Prof. Dr. Melchior Luiz Lima
President of the 30th International Congress of Cardiovascular Sciences

Scientific Co-Sponsorship

Meridional Kora Saúde	Rede Meridional – Kora Saúde
CENTROCOR Vitória	Clínica Centrocor Ltda.
	Brazilian Society of Cardiovascular Surgery – SBCCV Department of Experimental Research – DEPEX – SBCCV Department of Clinical Cardiology – DECARDIO – SBCCV
SOCIEDADE BRASILEIRA DE CARDIOLOGIA SBC MCMXLIII	Brazilian Society of Cardiovascular Surgery – SBCCV Department of Experimental Research – DEPEX – SBCCV Department of Clinical Cardiology – DECARDIO – SBCCV
CORDIS Clínica do Coração Ltda.	Heart Disease Clinic Ltda – CORDIS – ES
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SOCIEDADE BRASILEIRA DE CARDIOLOGIA DO ESPÍRITO SANTO SBC ES	Brazilian Society of Cardiology of Espírito Santo – SBC/ES

International Scientific Sponsorship	
	International Academy of Cardiovascular Sciences South American Section – IACS
	Peruvian Society of Cardiac, Thoracic and Vascular Surgery – SPCCTV
	Institute of Cardiovascular Pathophysiology, Faculty of Medicine, UBA, Buenos Aires, Argentina
	Institute of Cardiovascular Sciences Saint Boniface Hospital –University of Manitoba – Canada
	Medical Devices Innovation Institute - University of Ottawa, Canada

Students' Leagues of Cardiovascular Sciences
Ac./St. Amanda Klein Gaiotti Pasinato – ES Ac./St. Isadora Barros de Lacerda Fafá Roncete – ES Ac./St. Luiz Fernado Arantes –LHC-CEUB- DF Residente Mariana Camargo Afiune –CEUB-HUB- DF

Congress Executive Committee
President Director of The São Francisco de Assis Cardiovascular Institute
Prof. Dr. Otoni Moreira Gomes – MG
Director Kora Health Meridional Network
Fábio Luiz Michiles Frank – ES
President of Congress
Prof. Dr. Melchior Luiz Lima – ES
Scientific Director
Dr. Héber Souza Melo Silva – ES
Administrative Director
Dra. Elaine Maria Gomes Freitas (OAB) – MG

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Administrative Coordination
Sra. Ruana Freitas Marques Teixeira – MG **(INSTITUTO CARDIOVASCULAR SÃO FRANCISCO DE ASSIS - SERVCOR)**
Sra. Andrea Radavelli Miossi – ES

Local Scientific and Executive Committee
Dr. Arthur Soares Lima – ES
Dr. César Quintaes Freitas Lima Filho – ES
Dr. Héber Souza Melo Silva – ES
Dr. José Airton Arruda – ES
Dr. Melchior Luiz Lima – ES

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(Honorary Life President: Naranjan S. Dhalla)
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SEÇÃO SUL-AMERICANA DA ACADEMIA INTERNACIONAL DE CIÊNCIAS CARDIOVASCULARES – IACS / SOUTH AMERICAN SECTION OF INTERNATIONAL ACADEMY OF CARDIOVASCULAR SCIENCES – IACS
Presidente Honorário Vitalício da Seção Sul-Americana / Honorary Life President of South America Section
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Advisor
Enrique Castañeda Saldaña – Lima, Peru

Program Agenda – Thursday, October 17, 2024
30th Scientific Forum – International Congress of Cardiovascular Sciences
22nd Annual Meeting of The South American Section of International Academy of Cardiovascular Sciences
Hospital Meridional Vitória – Kora Saúde

Hospital Meridional Vitória Auditorium

07:30 a.m. – 08:00 a.m.: Registration

07:30 a.m. – 08:00 a.m.: Slide desk

08:00 a.m. – 08:10 a.m.: Opening

08:10 a.m. – 08:20 a.m.: Director of the Meridional Network
Dr. Fábio Frank – ES

08:20 a.m. – 08:30 a.m.: Clinical Director of the Hospital Meridional Vitória
Dr. Fátima Cristina Monteiro Pedrotti – ES

08:30 a.m. – 08:45 a.m.: President of the Congress
Dr. Melchior Luiz Lima – ES
President of the São Francisco de Assis Cardiovascular Institute
Prof. Dr. Otoni Moreira Gomes – MG
Scientific Director
Dr. Héber Souza Melo Silva – ES
Administrative Director
Dr. Elaine Maria Gomes Freitas (OAB) – MG
Administrative Coordination
Ms. Ruana Freitas Marques Teixeira – MG **(Instituto Casciovascular São Francisco de Assis - ServCor)**
Ms. Andrea Radavelli Miossi – ES

Symposium 1: 2nd Symposium Prof. Dr. Otoni Moreira Gomes – Symposium on Inflammatory Diseases and Cardiovascular Medicine

President:
Prof. Dr. Elias Kallás - MG
Coordination:
Dr. Fátima Cristina Monteiro Pedrotti– ES
Dr. José Aid Soares Sad – ES
Debaters:
Dr. Felipe Granja Moysés – ES
Dr. José Roberto Coutinho Nogueira – ES
Dr. Antônio Carlos Avanza Neto – ES

Topics:

08:45 a.m. – 09:00 a.m.: Case Report: Left Atrial Myxoma
Speaker: Ac./St. Amanda Klein – ES

09:00 a.m. – 09:15 a.m.: Myocardial Protection During Cardiovascular Surgery
Speaker: Dr. Arthur Soares Lima – ES

09:15 a.m. – 09:30 a.m.: Case Report: Aortic Annulus Enlargement with Y-Incision/Rectangular Patch
Speaker: Ac./St. Isadora Fafá Roncete – ES

09:30 a.m. – 09:45 a.m.: Epicardial fat as a cardiovascular risk factor
Speaker: Dr. Mônica de Monico Magalhães – ES

09:45 a.m. – 10:00 a.m.: Palliative care in patients with heart failure
Speaker: Dr. Adriana Lugo Ferrachini – MS

10:00 a.m. – 10:30 a.m.: Discussion

10:30 a.m. – 10:50 a.m.: Coffee

Program Agenda – Thursday, October 17, 2024
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Hospital Meridional Vitória – Kora Saúde

Auditorium of the Meridional Vitória Hospital

Symposium 2: 2nd Symposium Prof. Elias Kallás – Cardiovascular Diseases, Medicine for Diversity and Technological Innovation

President:
Prof. Dr. Elias Kallás – MG
Coordination:
Prof. Dr. Henrique Barsanulfo Furtado – TO
Prof. Dr. Samir Saadeddine Júnior – DF
Discussors:
Dr. Wilson Ayub Lopes – ES
Dr. Renato Guilherme Pimentel Tovar – ES
Dr. Mário César Santos de Abreu – BA
Dr. Antônio Carlos Avanza Neto – ES

Topics:

10:50 a.m. – 11:10 a.m.: Acute atrial fibrillation: Is there a difference between what is covered in the ACLS and the new guidelines?
Speaker: Dr. Antônio Carlos Avanza – ES

11:10 a.m. – 11:30 a.m.: Mediastinal tumors: Enucleation technique for giant tumors
Speaker: Prof. Dr. Elias Kallás – MG

11:30 a.m. – 11:50 a.m.: Beyond the Office: How can Marketing Boost your career in Cardiology?
Speaker: Dr. Fernanda Daura Damasceno da Silva – SC

11:50 a.m. – 12:10 p.m.: Medical Ethics: Informed Consent Form in Cardiac Surgery
Speaker: Prof. Dr. Alfredo Aurélio Marinho Rosa – AL

12:10 p.m. – 12:30 p.m.: Postoperative evolution of cardiac surgery with and without extracorporeal circulation
Speaker: Ac./St. Guilherme Kallás Hueb – MG

12:30 p.m. – 12:50 p.m.: Aging of the heart: what we know and what can be done to slow its progression
Speaker: Prof. Dr. Elmiro Santos Rezende – MG

12:50 p.m. – 01:10 p.m.: Building wealth as a smart doctor
Speaker: Dr. João Lucas Abreu - BA

01:10 p.m. – 01:30 p.m.: Discussion

01:30 p.m. – 02:30 p.m.: Lunch

Program Agenda – Thursday, October 17, 2024
30th Scientific Forum – International Congress of Cardiovascular Sciences
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Hospital Meridional Vitória – Kora Saúde

Symposium 3: Myocardial Aging – Advances in Heart Failure

President:
Dr. José Aid Soares Sad – ES
Coordination:
Dr. Fátima Cristina Monteiro Pedrotti– ES
Dr. Henrique Barsanulfo Furtado – TO
Discussers:
Dr. Alfredo Aurélio Marinho Rosa – AL
Dr. Fabrício Ribeiro França – ES
Dr. Glauco André Machado – DF
Dr. Elias Kallás – MG

Topics:

02:30 p.m. – 02:50 p.m.: Revisiting heterotopic heart transplantation
Speaker: Prof. Dr. José Wanderley Neto - AL

02:50 p.m. – 03:10 p.m.: Challenges in heart transplantation: now and in the future
Speaker: Prof. Dr. Fábio Antônio Gaiotto – SP

03:10 p.m. – 03:30 p.m.: Modern advances in heart transplantation
Speaker: Prof. Dr. Juan Alberto Cosquillo Mejia – CE

03:30 p.m. – 03:50 p.m.: Logistics and interaction of aeromedical teams and vital organ retrieval
Speaker: Lt. Col. Daniel Quintella – ES

03:50 p.m. – 04:10 p.m.: Discussion

04:10 p.m. – 04:25 p.m.: Coffee

Symposium 4: Symposium on Advances in Cardiovascular Sciences

President:
Dr. José Roberto Coutinho Nogueira – ES
Coordination:
Dr. Rodrigo Mussi Milani – PR
Dr. Fátima Cristina Monteiro Pedrotti – ES
Debaters:
Dr. Juan Alberto Cosquillo Mejia – CE
Dr. Ricardo Adala Benfatti – MS
Dr. Fabrício Ribeiro França – ES
Dr. Henrique Barsanulfo Furtado – TO

Topics:

04:25 p.m. – 04:45 p.m.: Mitral Valve Repair: Indications and Techniques – Why is it the Gold Standard of Treatment?
Speaker: Prof. Dr. Sérgio Lima de Almeida – SC

04:45 p.m. – 05:05 p.m.: Surgical treatment of aortic valve disease: options and challenges
Speaker: Prof. Dr. Edmo Atique Gabriel – SP

05:05 p.m. – 05:25 p.m.: Surgical treatment of mitral valve insufficiency with duplication of the posterior leaflet
Speaker: Prof. Dr. Mário Cézar Santos de Abreu – BA

Program Agenda – Thursday, October 17, 2024
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05:25 p.m. – 05:45 p.m.: ERAS protocol and minimally invasive techniques in the postoperative evolution of cardiovascular surgery
Speaker: Prof. Dr. Daniel Bregonci Trancoso Wernsbach – MG

05:45 p.m. – 06:05 p.m.: Surgical treatment of aortic valve stenosis versus TAVI: Which method is more appropriate?
Speaker: Prof. Dr. Samir Saadeddine Júnior – DF

06:05 p.m. – 06:25 p.m.: Residual risk in atherosclerotic cardiovascular disease: role of inflammation
Speaker: Prof. Dr. Fernando Luiz Torres Gomes – ES

06:25 p.m. – 06:40 p.m.: Discussion

Opening Conference of the Congress
President:
Dr. Fátima Cristina Monteiro Pedrotti – ES, Brazil
Coordination:
Prof. Dr. Grant Norval Pierce, Canada
Prof. Dr. Melchior Luiz Lima – ES, Brazil

Theme:

06:40 p.m. – 07:00 p.m.: World Milestones of the International Academy of Cardiovascular Sciences
Speaker: Prof. Dr. Naranjan S. Dhalla – Canada

07:00 p.m.: Closing

Program Agenda – Friday, October 18, 2024
30th Scientific Forum – International Congress of Cardiovascular Sciences
22nd Annual Meeting of The South American Section of International Academy of Cardiovascular Sciences
Hospital Meridional Vitória – Kora Saúde

Hospital Meridional Vitória Auditorium

07:30 a.m. – 08:00 a.m.: Registration

07:30 a.m. – 08:00 a.m.: Slide desk

Symposium 1: 22nd Prof. Naranjan S. Dhalla Symposium (Module I)
South American Section of the International Academy of Cardiovascular Sciences
Advances in Cardiovascular Sciences, Experimental Research and Undergraduate Research
Chair:
Prof. Dr. Naranjan S. Dhalla, Canada

Coordination:
Prof. Dr. Ricardo Jorge Gelpi – Buenos Aires, Argentina
Prof. Dr. Samir Saadeddine Júnior – DF, Brazil
Discussors:
Prof. Dr. Ramesh Goyal – India
Prof. Dr. Grant Norval Pierce, Canada
Prof. Dr. Dalton Valentim Vassallo – UFES, Brazil
Prof. Dr. Lorrie Kirshenbaum – Canada

Topics:

08:00 a.m. – 08:20 a.m.: Pathophysiology of Cardiac Dysfunction: a brief journey through the interior of the cardiomyocyte
Speaker: Prof. Dr. Ivanita Stefanon, UFES, Brazil

08:20 a.m. – 08:40 a.m.: Mechanisms for loss of adrenergic support in heart failure due to myocardial infarction
Speaker: Prof. Dr. Naranjan S. Dhalla – Canada

08:40 a.m. – 09:00 a.m.: Mitochondrial antioxidant action of mitoquinone on cardiac mitochondrial metabolism after acute myocardial infarction
Speaker: Prof. Dr. Georgia Azevedo Oliveira Traichel MD (Master's degree student in the Biochemistry Graduate Program, UFES, Brazil

09:00 a.m. – 09:20 a.m.: Resistance training restores cardiac function by improving mitochondrial metabolism in conditions of hormone deficiency
Speaker: Prof. Dr. Eduardo Hertel Ribeiro, UFES, Brazil

09:20 a.m. – 09:40 a.m.: Innate immune signaling in doxorubicin cardiomyopathy
Speaker: Prof. Dr. Lorrie Kirshenbaum – Canada

09:40 a.m. – 10:00 a.m.: The role of combined administration of AT1 receptor antagonist and neprilysin inhibitor in promoting browning in an experimental model of metabolic syndrome
Speaker: Prof. Dr. Vladimir Jakovljevic - Serbia

10:00 a.m. – 10:20 a.m.: Discussion

10:20 a.m. – 10:40 a.m.: Coffee

Program Agenda – Friday, October 18, 2024
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Hospital Meridional Vitória – Kora Saúde

Oral Communication Session
Chair:
Prof. Dr. Grant Norval Pierce, Canada
Coordination:
Prof. Dr. Ivanita Stefanon – UFES, Brazil
Prof. Dr. Dalton Valentim Vassallo – UFES, Brazil
Discussers:
Prof. Dr. Naranjan S. Dhalla – Canada
Prof. Dr. Ramesh Goyal – India
Prof. Dr. Ivanita Stefanon – UFES, Brazil
Prof. Dr. Henrique Barsanulfo Furtado – TO, Brazil

Themes:

10:40 a.m. – 11:00 a.m.: Chronic exposure to mercury increases arrhythmia and mortality after acute myocardial infarction in rats
Speaker: Prof. Dr. Aurélia Araujo Fernandes, UFES, Brazil

11:00 a.m. – 11:20 a.m.: Long-term androgen deprivation modulates vascular reactivity dependent on endothelial co-regulation of aldosterone and angiotensin II
Speaker: Prof. Anna Karolina Nascimento Costa (Ph.D. Student, UFES), Brazil

11:20 a.m. – 11:35 a.m.: Mitochondrial reactive oxygen species, cyclooxygenase-1 and NADPH oxidase-4 are involved in early vascular dysfunction after myocardial infarction
Speaker: Ac/St. Marlon R. Machado (Undergraduate Research Student, 10th semester of Medicine), UFES, Brazil

11:35 a.m. – 11:50 a.m.: The impact of mitoquinone on vascular remodeling early after myocardial infarction
Speaker: Sara Bianca Oliveira Mendes (Undergraduate Research Student, Graduate Program in Physiological Sciences), UFES, Brazil

11:50 a.m. – 12:05 p.m.: Effect of zinc administration on vascular function in the aorta of rats acutely exposed to cadmium
Speaker: Camilla Loren da Silva Nascimento (Master's degree student in the Physiological Sciences Post Graduate Program), UFES, Brazil

12:05 p.m. – 12:20 p.m.: STI1 overexpression protects mice from adrenergic hyperactivation-induced injury
Speaker: Dr. Katyana Kaline S. Ferreira (Post-doctoral fellow, Physiological Sciences Post Graduate Program), UFES, Brazil

12:20 p.m. – 12:50 p.m.: Herpes Zoster and cardiovascular risk
Speaker: Dr. Mariana Camargo Afiune – HUB, DF

12:50 p.m. – 01:05 p.m.: Cardiovascular manifestations of Dengue
Speaker: Ac./St. Luiz Fernando Arantes de Souza – CEUB, DF

01:05 p.m. – 01:30 p.m.: Discussion

01:30 p.m. – 02:30 p.m.: Lunch

Program Agenda – Friday, October 18, 2024
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Hospital Meridional Vitória – Kora Saúde

01:30 p.m. – 02:30 p.m.: Business Meeting of the South American Section of the International Academy of Cardiovascular Sciences
Location: Board Room of the Hospital Meridional Vitória
Chief Executive Officer (IACS): Prof. Dr. Naranjan S. Dhalla, Winnipeg, Canada
President (IACS): Prof. Dr. Grant Norval Pierce, Winnipeg, Canada
Members of the South American Section (IACS): Melchior Luiz Lima (Brazil), Alexandre Ciappina Hueb (Brazil), Ricardo Jorge Gelpi (Argentina), Elaine Gomes Freitas (Brazil), Elias Kallás (Brazil), and Enrique Castañeda Saldana (Peru).

Symposium 2: 22nd Prof. Naranjan S. Dhalla Symposium (Module II)
South American Section of the International Academy of Cardiovascular Sciences
Advances in Cardiovascular Sciences, Experimental Research and Student Scientific Initiation
Chair:
Prof. Dr. Grant Norval Pierce, Canada
Coordination:
Prof. Dr. Dalton Valentim Vassallo – UFES, Brazil
Prof. Dr. Ivanita Stefanon – UFES, Brazil
Discussers:
Prof. Dr. Naranjan S. Dhalla – Canada
Prof. Dr. Lorrie Kirshenbaum – Canada
Prof. Dr. Henrique Barsanulfo Furtado – TO, Brazil
Prof. Dr. Ramesh Goyal – India

Topics:

02:30 p.m. – 02:50 p.m.: Reverse engineering approach for discovery of novel drugs from natural resources for diabetes-induced cardiovascular complications
Speaker: Prof. Dr. Ramesh Goyal – India

02:50 p.m. – 03:10 p.m.: Improved valve endothelialization after transcatheter aortic valve replacement (TAVR)
Speaker: Prof. Dr. Michael Kutryk – Canada

03:10 p.m. – 03:30 p.m.: Serious consideration of the role of dietary supplements in prevention and treatment of cardiovascular diseases
Speaker: Prof. Dr. Grant Norval Pierce – Canada

03:30 p.m. – 03:40 p.m.: Arrhythmogenic Genetic Disease Due to Mutations in the Cardiac Ryanodine Receptor (RyR2): Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
Speaker: Prof. Dr. Ivanita Stefanon – ES

03:40 p.m. – 04:00 p.m.: Embryonic Stem Cell-Derived Exosomes Modulate Anticancer Drug-Induced Toxicity
Speaker: Prof. Dr. Dinender K. Singla – USA

04:00 p.m. – 04:20 p.m.: Discussion

04:20 p.m. – 04:45 p.m.: Coffee

Program Agenda – Friday, October 18, 2024
30th Scientific Forum – International Congress of Cardiovascular Sciences
22nd Annual Meeting of The South American Section of International Academy of Cardiovascular Sciences
Hospital Meridional Vitória – Kora Saúde

Symposium 3: Advances in Cardiovascular Medicine

President:
Dr. Ricardo Adala Benfatti – MS
Coordination:
Dr. Vitor Arantes Pazolini – ES
Dr. Maurílio Onofre Deininger – PR
Discussers:
Dr. César Quintaes Freitas Lima Filho – ES
Dr. Elmiro Santos Rezende – MG
Dr. Henrique Barsanulfo Furtado – TO
Dr. Héber Souza Melo Silva – ES

Topics:

04:45 p.m. – 05:05 p.m.: Cardiac pacemaker in everyday life: guidelines for frequently asked questions in the office
Speaker: Prof. Dr. Jorge Elias Neto – ES

05:05 p.m. – 05:25 p.m.: Technical variations for Fontan and Norwood surgeries
Speaker: Prof. Dr. José Teles de Mendonça - SE

05:25 p.m. – 05:45 p.m.: Current status of autotransfusion in cardiovascular surgery
Speaker: Prof. Dr. Alfredo Inácio Fiorelli – SP

05:45 p.m. – 06:05 p.m.: Cardiovascular Surgery: A Surgeon's Personal View!
Speaker: Prof. Dr. Henrique Barsanulfo Furtado – TO

06:05 p.m. – 06:25 p.m.: Effects of polluted air on the pathophysiology of myocardial ischemia
Speaker: Prof. Dr. Ricardo Jorge Gelpi, Buenos Aires – Argentina

06:25 p.m. – 06:40 p.m.: Discussion

06:40 p.m. – 07:30 p.m.: Awards Ceremony
Meeting for Announcement of Recognition and Presentation of Awards by the South American Section of the International Academy of Cardiovascular Sciences
Location: Auditorium of the Hospital Meridional Vitoria
President: Prof. Dr. Naranjan S. Dhalla
Congratulatory remarks: Prof. Dr. Naranjan S. Dhalla and Prof. Dr. Grant Norval Pierce.
Presentation of the IACS Awards: Prof. Dr. Grant Norval Pierce, Dr. Melchior Luiz Lima, Prof. Dr. Elias Kallás, Prof. Dr. Samir Saadeddine Júnior, Prof. Dr. Antoinette Oliveira Blackman, Prof. Dr. Henrique Furtado, and Prof. Dr. Elaine Maria Gomes Freitas.
Members of the IACS South American Section: Melchior Luiz Lima (Brazil), Alexandre Ciappina Hueb (Brazil), Ricardo Jorge Gelpi (Argentina), Elaine Gomes Freitas (Brazil), Elias Kallás (Brazil) and Ramesh Goyal (India), Vladimir Jakovljevic (Serbia), Michael Kutryk (Canada), and Lorrie Kirshenbaum (Canada).
Scientific research awards (poster/oral): Prof. Dr. Naranjan Dhalla, Prof. Dr. Grant Norval Pierce, Dr. Melchior Luiz Lima, Prof. Dr. Elias Kallás, Prof. Dr. Samir Saadeddine Júnior, Prof. Dr. Antoinette Oliveira Blackman, Prof. Dr. Henrique Furtado, and Prof. Dr. Elaine Maria Gomes Freitas.

07:30 p.m.: Closing

Program schedule – Saturday, October 19, 2024
30th Scientific Forum – International Congress of Cardiovascular Sciences
22nd Annual Meeting of The South American Section of International Academy of Cardiovascular Sciences
Hospital Meridional Vitória – Kora Saúde

Hospital Meridional Vitória Auditorium

07:30 a.m. – 08:00 a.m.: Registration

07:30 a.m. – 08:00 a.m.: Slide desk

Symposium 1: Evolution of the anesthetic process in Cardiovascular Sciences and Scientific Initiation of Students

President:
Prof. Dr. Ricardo Jorge Gelpi – Buenos Aires, Argentina
Coordination:
Dr. Antoinette Oliveira Blackman – DF
Prof. Dr. Antonio Carlos Avanza Neto – ES
Speakers:
Dr. Ricardo Adala Benfatti – MS
Dr. Victor Arantes Pazolini – ES
Dr. Fabricio Ribeiro França – ES
Dr. Henrique Barsanulfo Furtado – TO

Topics:

08:00 a.m. – 08:15 a.m.: Heart Transplantation in ES: Experience and Historical Overview
Speaker: Prof. Dr. Pablo Gusmann – ES

08:15 a.m. – 08:30 a.m.: Comparison in Postoperative Analgesia: Esp Block versus Intravenous Morphine PCA
Speaker: Dr. Heitor Cunha Lima – ES

08:30 a.m. – 08:45 a.m.: Physiology Applied to Ventricular Assist Devices: Anesthetic Management
Speaker: Dr. Bruno Campostrini Sily – ES

08:45 a.m. – 09:00 a.m.: ERAS Protocol in Cardiovascular Surgery
Speaker: Prof. Dr. Pablo Gusmann – ES

09:00 a.m. – 09:15 a.m.: Discussion

Symposium 2: Minimally Invasive Cardiac Surgery Symposium – New Surgical Technique and New Technologies in Cardiovascular Surgery

Chair:
Dr. José Roberto Coutinho Nogueira – ES
Coordination:
Dr. Ricardo Jorge Gelpi – Buenos Aires, Argentina
Dr. Eduardo Vervloet Moysés
Discussors:
Dr. Henrique Barsanulfo Furtado – TO
Dr. Fernanda Bento de Oliveira Viana – ES
Dr. José Airton Arruda – ES
Dr. Wilson Ayub Lopes – ES

Topics:

09:15 a.m. – 09:35 a.m.: Hybrid approach in minimally invasive myocardial revascularization
Speaker: Dr. Milton de Miranda Santoro – PR

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09:35 a.m. – 09:55 a.m.: How do I perform minimally invasive myocardial revascularization without CPB?
Speaker: Dr. Ricardo Adala Benfatti – MS

09:55 a.m. – 10:15 a.m.: Minimally invasive myocardial revascularization
Speaker: Dr. Rodrigo Mussi Milani – PR

10:15 a.m. – 10:30 a.m.: Discussion

10:30 a.m. – 11:00 a.m.: Coffee

Symposium 3: Inflammatory disease and myocyte aging

President:
Dr. Ricardo Adala Benfatti – MS
Coordination:
Dr. Vítor Arantes Pazolini – ES
Dr. Samir Saadeddine Júnior – DF
Discussors:
Dr. Jorge Elias Neto – ES
Dr. Fátima Cristina Monteiro Pedrotti– ES
Dr. Fernanda Bento de Oliveira Viana – ES
Dr. Felipe Ribeiro do Val Tommasi Silva – ES

Topics:

11:00 a.m. – 11:20 a.m.: Pulmonary thromboembolism
Speaker: Prof. Dr. Alexandre Barbosa Andrade – MG

11:20 a.m. – 11:40 a.m.: Current events in renal cardiometabolism
Speaker: Dr. Tatiane Mascarenhas Santiago Emerick – ES

11:40 a.m. – 12:00 p.m.: Cardiac Tumors
Speaker: Prof. Dr. Alfredo Aurélio Marinho Rosa – AL

12:00 p.m. – 12:20 p.m.: Sudden death in Chagas disease and mitral valve prolapse: what is common and important in their pathophysiology
Speaker: Prof. Dr. Glauco André Machado – DF

12:20 p.m. – 12:40 p.m.: Use of fibrinogen in the postoperative period of complex surgeries
Speaker: Prof. Dr. Alexandre Ciappina Hueb – MG

12:40 p.m. – 01:00 p.m.: Metabolic modulation of the Cardiomyocyte: Physiology and clinical application
Speaker: Prof. Dr. Samir Saadeddine Júnior – DF

01:00 p.m. – 01:20 p.m.: Ehler-Danlos syndrome: need for a careful look at the diagnosis
Speaker: Prof. Dr. Antoinette Oliveira Blackman – DF

01:20 p.m. – 01:30 p.m.: Discussion

01:30 p.m. – 02:30 p.m.: Lunch

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Symposium 4: Preventing Stroke Related to Atrial Fibrillation – New Frontiers and Concepts

President:
Dr. Bruno Moulin Machado – ES
Coordination:
Dr. Vítor Arantes Pazolini – ES
Dr. Héber Souza Melo Silva – PR
Discussers:
Dr. Mário César Santos de Abreu – BA
Dr. César Quintaes Freitas Lima Filho – ES
Dr. Samir Saadeddine Júnior – DF
Dr. Elmiro Santos Rezende – MG

Topics:

02:30 p.m. – 02:45 p.m.: Atrial Fibrillation after Aortic Valve Replacement – Incidence, predictors, treatment
Speaker: Dr. Fernanda Bento de Oliveira Viana - ES

02:45 p.m. – 03:00 p.m.: Surgical occlusion of the left atrial appendage – When and how to do it?
Speaker: Dr. Melchior Luiz Lima – ES

03:00 p.m. – 03:15 p.m.: Closure of the left atrial appendage: a safe alternative in the prevention of cardioembolic stroke?
Speaker: Dr. José Airton Arruda - ES

03:15 p.m. – 03:30 p.m.: Closing the LAA simultaneously with TAVI – Pros and Cons
Speaker: Dr. Alfredo Nunes Ferreira Neto – ES

03:30 p.m. – 03:45 p.m.: Interactive clinical case of left atrial appendage closure
Speaker: Prof. Dr. Vitor Arantes Pazolini – ES

03:45 p.m. – 04:00 p.m.: Discussion

04:00 p.m. – 04:15 p.m.: Coffee

Symposium 5: Cardiovascular Repair and Regenerative Medicine (Module I)

Chair:
Dr. Ricardo Jorge Gelpi – Buenos Aires, Argentina
Coordination:
Dr. Milton de Miranda Santoro – PR
Dr. César Quintaes Freitas Lima Filho – ES
Discussors:
Dr. Eduardo Vervloet Moysés
Dr. Fabrício Ribeiro França – ES
Dr. Mário César Santos de Abreu – BA
Dr. César Quintaes Freitas Lima Filho – ES

Topics:

04:15 p.m. – 04:30 p.m.: Case report: Conduct in the face of rejection after heart transplantation
Speakers: Dr. Felipe Ribeiro do Val Tommasi Silva – ES / Dr. Marcus Aguilar Constantino – ES

04:30 p.m. – 04:45 p.m.: Sudden Death in Sports and Exercise: What is the best way to Prevent it?
Speaker: Prof. Dr. Antônio Carlos Avanza – ES

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04:45 p.m. – 05:00 p.m.: Case report: Stanford type A acute aortic dissection and pregnancy
Speakers: Dr. Letícia Bonacossa Ferrari / Dr. Mirella Lourencini Palaoro – ES

05:00 p.m. – 05:15 p.m.: The role of psychological stress in the development of cardiovascular diseases
Speaker: Dr. João Eugênio Alvarenga Neto – BA

05:15 p.m. – 05:30 p.m.: Cardiocerebral revitalization
Speaker: Prof. Dr. Usiel Carneiro de Souza – ES

05:30 p.m. – 05:45 p.m.: Discussion

06:00 p.m.: Closing

POSTER SESSION 2024

POSTER/ORAL (ROOM "A" – AUDITORIUM) SESSION 2024

Acute effects of short-term mitoquinone treatment on vascular remodeling post-myocardial infarction

Sara Bianca Oliveira Mendes1, TadeuÉriton Caliman Zanardo1, Ferreira, K.K.S1, Georgia Azevedo de Oliveira Traichel3, Lais de Oliveira Traichel1, Anna Karolina Nascimento Costa1, Tiago Spalenza1, Ivanita Stefanon1, Maicon Landim-Vieira1,2, Aurélia Araújo Fernandes3.
1Department of Physiological Sciences of the Federal University of Espirito Santo, Vitória, Espirito Santo, Brazil.
2Department of Biomedical Sciences, College of Medicine, Florida State University, Tallahassee, FL, USA.
3Biochemistry Postgraduate Program, Federal University of Espírito Santo, Brazil.

Introduction: Myocardial Infarction (MI) is a serious condition that can lead to heart failure and death. Following MI, there is an imbalance in reactive oxygen species (ROS) production, resulting in systemic inflammation and affecting cardiac and vascular function. Mitochondria are key therapeutic targets due to their role in ROS production, and mitochondrial-targeted antioxidants show promise for treatment. Objective: To evaluate the vascular remodeling and the effects of mitochondrial antioxidant mitoquinone (MitoQ) in the aorta from rats seven days post-MI. Methods: Wistar male rats, eight weeks old, were divided into: Sham, MI, Sham MitoQ and MI MitoQ (CEUA 06/2023). The MI was surgically induced and following the animals were treated for seven days with MitoQ (100µM/Kg/day) in drinking water. After treatment, hemodynamic analysis was performed and the thoracic aorta was collected for histological evaluation, and scanning electron microscopy (SEM). The statistical analyses are presented as mean±SEM. Two-way ANOVA followed by Fisher's post hoc test was performed. Significance values were P<0.05. Results: There were reductions in the systolic (Sham 113±3 N=10; MI 93±4* N= 9; Sham MitoQ 108±4 N=4; MI MitoQ 108±3# N=4; *p<0.05 Sham vs MI; #p<0.05 Sham MitoQ vs MI MitoQ) and diastolic (Sham 84±3 N=10; MI 68±3* N=9; Sham MitoQ 81±3 N=4; MI MitoQ 85±3 N=4; *p<0.05 Sham vs MI) arterial blood pressure in the MI compared to Sham group. We also observed an increase in the left ventricular systolic pressure (Sham 116±5 N=10; MI 79±3* N=9; Sham MitoQ 115±6 N=4; MI MitoQ 100±4# N=4; *p<0.05 Sham vs MI; #p<0.05 Sham MitoQ vs MI MitoQ) and left ventricular end diastolic pressure (Sham: 5±0.4, MI:10±1*; Sham MitoQ: 3±1; IM MitoQ: 5±0.5#; *p<0.05 Sham vs MI; #p<0.05 Sham MitoQ vs MI MitoQ). MitoQ was able to restore all these parameters. Lumen thickness and area were equal among groups (Sham 178,9±8,4 □m2 N=4; MI 116±43,1 □m2 N=4; Sham MitoQ 178,1±2,6 □m2 N=4; MI MitoQ 162,5±12,7 □m2 N=4). The number of elastin injury was significantly increased in the MI compared to the Sham (Sham 6,5±1,6 N=4; MI 13.5±1,6* N=4; *p<0.05 Sham vs MI). Elastin injury were not observed in MI MitoQ compared to Sham MitoQ (Sham MitoQ 5,8±1,1 N=4; MI MitoQ 8,18±1,7 N=4). Collagen content was increased in the MI and MitoQ did not change this alteration (Sham 13,6±0,9 % N=4; MI 24,7±3,0* % N=4; Sham MitoQ 16,6±0,3 % N=4; MI MitoQ 25,7±1,9# % N=4; *p<0.05 Sham vs MI; #p<0.05 Sham MitoQ vs MI MitoQ). SEM analysis showed an important endothelial denudation in the MI group while this damage was rare in the MI MitoQ (Sham 0,2±0,1 N=4; MI 2,3±0,1* N=4; Sham MitoQ 0,3±0,2 N=4; MI MitoQ 1,3±0,2#& N=4; *p<0.05 Sham vs MI; #p<0.05 Sham MitoQ vs MI MitoQ; & p<0.05 MI vs MI MitoQ). Conclusion: Acute treatment with MitoQ was effective in preventing adaptive remodeling of the rat aorta 7 days after myocardial infarction.
KeyWords: Aorta; Mitoquinona; Remodeling; Myocardial Infarction.
Financial Support: FAPES, CAPES, CNPq, UFES

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POSTER/ORAL (ROOM B) SESSION 2024

Testosterone and Aldosterone: Long-Term Modulation of Vascular Reactivity in Orchiectomized Wistar Rats Treated with Spironolactone.
Costa, A. K. N; Athayde, P. S; Hortelan, M. R; Eduardo Hertel Ribeiro, Fernandes, A. A. Stefanon, I.

Introduction: Testosterone, has been shown to exhibit endothelium-dependent vasodilation in acute scenarios. We test the hypothesis that testosterone plays a major role in the long-term modulation of vascular reactivity by an aldosterone-dependent pathway. Methods: 12-week-old Wistar rats were segregated into Control (SHAM, N=8) and Orchiectomy (OQT, N=9) groups and treated for 3 months with spironolactone (SPI) (SHAM+SPI, N=10 and OQT+SPI, N=9, 80 mg/kg, gavage), aldosterone receptor antagonist. (CEUA-UFES 17/2020). Vascular reactivity was examined in isolated thoracic aorta rings during concentration-response curves to phenylephrine (Phe) (10-11 to 10-3 M) in the presence and absence of L-NAME (LN) (NOS inhibitor, 100 µM), and with endothelium-denuded rings (E-). Results were expressed as mean±SEM. Results: After 3 months, the groups OQT show less body weight gain compared to the SHAM. This discrepancy was abolished with SPI treatment. (SHAM:231±11; OQT:158.4±13*; OQT+SPI:180.5±20.60*; SHAM+SPI: 215.3±26.1 g *p<0.05). There was no difference in the maximum response (Rmax) to Phe between the SHAM and OQT groups. However, OQT+SPI group demonstrated a decreased Rmax to Phe compared to the SHAM (SHAM:109.4±9.5%; OQT:120.5±10%; SHAM+SPI: 120.4±7.56 % N=10 vs OQT+SPI: 93.3±10.2 % N=10; *p<0.05). The reactivity to Phe increased in the presence of LN and in E-, with no difference between the groups. Conclusion: Our data suggest that testosterone has a role in angiotensin II-mediated NO production, since blocking NO production with LN resulted in a decrease in Rmax in the OQT group compared to its control. It highlights the potential of testosterone in the contractile response mediated by the angiotensin AT1 receptor. Financial support: FAPES-CAPEX edital 019/2022 Número: 2022-6C3F7.
Keywords: Testosterone; Angiotensin II; Vascular Reactivity; Aldosterone, Male sexuals hormones; Vascular endothelium.

Contribution of COX/TXA2 pathway on the vascular reactivity mediated by male sex hormones
Undergraduate Student abstracts (YES, I am available to deliver an Oral
Communication in the Scientific Competition if selected)
Cardiovascular and respiratory
Paula dos Santos Athayde 1, Maria Eduarda Morais Hibner Amaral 1,
Anna Karolina Nascimento Costa 1, Aurelia Araujo Fernandes 2,
Eduardo Hertel Ribeiro 1, Ivanita Stefanon 1.
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Introduction: Testosterone exerts cardiovascular effects through genomic and nongenomic mechanisms. While its acute vasodilatory action on vascular smooth muscle function has been established, its long-term impact on vascular reactivity remains unclear. Aim: We investigated the hypothesis that testosterone participates in the long-term regulation of vascular tone via the cyclooxygenase/thromboxane (COX/TXA2) pathway. Methods: Male Wistar rats were divided into orchidectomized (OQT, N=11) and control (SHAM, N=11) groups. After 12 weeks, the aortic rings were isolated for the assessment of vascular reactivity to phenylephrine (10-11 to 10-3.5 M) in the presence of inhibitors: Indomethacin 10 µM (non-selective COX inhibitor), NS 398 1 µM (selective COX2 inhibitor), SQ 29.548 1 µM (selective thromboxane receptor blocker), and furegrelate 10 µM (TXA2 synthase inhibitor). Ethical approval (CEUAUFES 17-2020). Statistical analysis: Student's t-test or one-way ANOVA, significant to P<0.05. Results: The Rmax to phenylephrine was similar between control groups (SHAM:118.1±6.29, N=7; OQT:115.9±4.38, P>0.05, N=13). Furegrelate equally reduced Rmax in both groups (SQ 29,548; SHAM: 67.1±3.20*, N=9; OQT: 77.12±3.7*, N=8, *P<0.05). The vascular reactivity was not affected by indomethacin, NS 398, and furegrelate in either group: Indomethacin (SHAM:99.8±2.8, N=6; OQT:119.4±2.84, P>0.05, N=9), NS 398 (SHAM:101±4, N=6; OQT:97.1±2.4, P>0.05, N=8), and furegrelate (SHAM:107.9±4.78, N=8; OQT:110.8±3.9, P>0.05, N=11). The results demonstrated that phenylephrine-mediated vasoconstriction was equally dependent on TXA2 receptors in the SHAM and OQT groups. Conclusion: We conclude that testosterone, in the long term, does not appear to participate in the regulation of vascular reactivity via the COX-TXA2 pathway in isolated aortic rings of young rats.
Financiamiento: Fundação de Amparo à Pesquisa e Inovação do Espírito Santo (Fapes), CNPq, UFES

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EFFECT OF ZINC ADMINISTRATION ON VASCULAR REACTIVITY IN THE AORTA OF RATS ACUTELY EXPOSED TO CADMIUM

Camilla Lóren da S. Nascimento1, Lorraine Christiny C. Sepulchro1, Rakel P. Simões1, Ivanita Stefanon1, Alessandra S. Padilha1.
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Cadmium (Cd) is a highly toxic metal that promotes metabolic and enzymatic changes. In the cardiovascular system, the endothelium and smooth muscle are the main targets, promoting oxidative stress and vascular dysfunction, contributing to cardiovascular disease development. Therefore, investigating substances to reverse or prevent this damage is crucial. Zinc (Zn), essential in several metabolic reactions, also acts as a potent antioxidant that can mitigate oxidative stress. The present study aims to evaluate the effects of Zn administration (50 µM) on vascular reactivity to phenylephrine in isolated rings from the thoracic aorta of rats acutely exposed to Cd (10 µM). Male Wistar rats (Rattus novergicus albinus), 12 weeks old (320 g to 470 g) were used. During the experiment, they were divided into four in vitro exposures to metals, specifically: Ct, Cd, Zn and Cd+Zn. The experimental protocol was conducted on thoracic aorta rings which were cleaned of fat and connective tissue, in the presence (E+) and absence (E-) of endothelium to investigate the endothelium's participation. The effects of the following drugs were evaluated: L-NAME, Apocynin (APO) and Catalase (CAT) to evaluate the involvement oxidative stress pathways. The Federal University of Espírito Santo Ethics Committee on the Use of Animals approved the experimental protocol (CEUA-UFES, nº 12/2022). The contractile responses were analyzed using two-way ANOVA followed by a Bonferroni test and expressed as the percentage of contraction induced by KCl. The effects of the drugs were analyzed as differences in the area under the concentration-response curves using the Student's t test (unpaired). $P < 0.05$ was considered significant. The results revealed that acute exposure to Cd increased contractility to phenylephrine, while co-incubation with Zn allowed for the normalization of this response to control levels (Ct: 76,19±3,83%; Cd: 108,79±6,42%; Cd.Zn: 77,76±4,35%). The absence of endothelium caused higher vascular reactivity, indicating that the changes induced by Cd are endothelium-dependent (Ct.E+: 76,19±3,83%; Ct.E-: 134,15±8,35%; Cd. E+: 104,49±7,11%; Cd.E-: 93,60±7,68%). Incubation with L-NAME revealed that cadmium reduced NO
bioavailability; with APO, it induced NADPH Oxidase participation; and with CAT, it stimulated hydrogen peroxide formation, favoring a major contractile response. In contrast, co-incubation with Zn enhanced NO bioavailability during the contractile response to phenylephrine, achieving values comparable to those of the control groups. Additionally, Zn demonstrated the ability to prevent ROS production by NADPH oxidase, associated with reduced hydrogen peroxide release (L-NAME: Ct: 135,53±9,50%; Cd: 111,63±12,70%; Cd.Zn: 144,34±9,56%. APO: Ct: 71,81±4,03%; Cd: 79,91±7,72%; Cd.Zn: 69,81±7,05%. CAT: Ct: 71,27±5,58%; Cd: 105,96±8,66%; Cd.Zn: 87,55±5,72%). Thus, Zn represents a promising non-pharmacological intervention for preventing damage to the cardiovascular system resulting from exposure to heavy metals. This study was supported by grants from CNPq and FAPES.
Keywords: oxidative stress; endothelial dysfunction; zinc supplementation; cardiovascular diseases; cadmium chloride.

Mitochondrial Antioxidant Mitoquinone Prevents Vascular Dysfunction, Oxidative Stress, and Mitochondrial-NADPH Oxidase Crosstalk Following Myocardial Infarction

Undergraduate Student abstracts (YES, I am available to deliver an Oral Communication)
Cardiovascular and respiratory

Marlon Ramos Machado 1, Carmen Castardeli 1, Carolina Falcão Ximenes 1, Pietra Zava Lorencini 1, Katyana Kaline Silva Ferreira 1, 3, Ingridy Grafit R Schereider 1, Bruno de Lima Sanches 3, Marcos Eliezeck 3, Eduardo Hertel Ribeiro 1, Aurelia Araujo Fernandes 2, Ivanita Stefanon 1, Silvia Guatimosim 3.
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Myocardial infarction (MI) is a critical condition triggering inflammatory responses and oxidative stress, leading to vascular complications and endothelial dysfunction. Our research explores the role of mitochondrial-NADPH oxidase (NOX) crosstalk in aortic vascular reactivity 7 days post-MI. Male Wistar rats underwent surgical induction of MI and were categorized into Myocardial Infarction (MI), Myocardial Infarction Mitoquinone (MIM), Sham (S), and Sham Mitoquinone (SM) groups. Animals received Mitoquinone (100 µM), a specific mitochondrial antioxidant, in drinking water for seven days (CEUA-UFES 17/2021). Vascular reactivity was evaluated in isolated aortic rings under phenylephrine stimulation. Results showed no change in infarct area with Mitoquinone treatment (MI=46.6 ± 2.8; MI MitoQ=43.6±2.6%), but a prevention of weight loss in the MI group (MI=-6±2.7*; MI MitoQ=16±4 g*, $p<0.01$). Reactivity to phenylephrine increased in the MI (Rmax S:100.1±5.5 vs MI:127.3±8.7*% KCl 75 mM, $p<0.05$). Mitoquinone treatment prevented the increase in vascular reactivity in the MI (Rmax MI:127.3±8.7 vs MIM:100±8.5*% KCl 75 mM $p<0.05$). L-NAME 100 µM had no effect on Rmax in the MI, suggesting a decrease in NO bioavailability. However,
when the MI group was treated with MitoQ, superfusion with L-NAME resulted in an increased response to phenylephrine, suggesting higher NO bioavailability. Mitoquinone treatment reduced mitochondrial (Mitosox) and cytosolic ROS (DHE) and increased vascular NO production (DAF). The specific NOX inhibitor, ML-171 5 µM, reduced vascular reactivity and decreased NOX1 expression in the MI group. In conclusion, the mitochondrial antioxidant mitoquinone prevented vascular dysfunction, oxidative stress dependent on mitochondrial-NADPH Oxidase crosstalk 7 days after MI.
Financiamiento: Fundação de Amparo à Pesquisa e Inovação do Espírito Santo (Fapes), CNPq, UFES
Agradecimientos: Thanks to Dr. Michael P. Murphy for providing the molecule of mitoquinone for this study

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Evolution of Blood Pressure in Spontaneously Hypertensive Rats (SHR) Based on a Diet Rich in Magnesium

Livia Suzano de Paula dos Santos 1, 2, Paula dos Santos Athayde 1, Livia Seif Eddine 1, 2, Michelle RM Hortelan 1, 2, Aurelia Araujo Fernandes 1, Ivanita Stefanon 1.
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Systemic Arterial Hypertension (SAH) is considered a chronic pathological process of multifactorial etiology, characterized by chronic elevation of baseline blood pressure, representing an important risk factor for cardiovascular diseases. It is considered a serious public health problem, as it is associated with the onset of other chronic-degenerative diseases that negatively affect the quality of life of individuals. Epidemiological studies suggest that diet plays a relevant role in determining blood pressure (BP). Recently, it has been shown that magnesium supplementation can help control and even reduce blood pressure levels, but these studies are inconsistent. Thus, the present study aimed to investigate the hypothesis that a diet supplemented with MgCl2 help control and even modify blood pressure in hypertensive rats.
Spontaneously hypertensive rats (SHR), male animals with 6 weeks old (120-200 grams), were divided into: treatment group that received a supplementation daily dose of 50 mg/kg of MgCl2 (drinking water for two months) (SHR-Mg), and control group that received no treatment (SHR). Blood pressure (BP) was measured weekly using the tail-cuff plethysmography method.
The recorded arterial pulses were individually analyzed using one-way ANOVA test, post hoc Tukey (UFES Animal Ethics Committee 16-2020). Systolic arterial pressure (SAP) at the beginning and at the end of 8 weeks of treatment were comparable between the SHR-Mg² group (Initial SAP SHR:156.9 ± 5.7 vs SHR Mg:167 ± 5.8 mmHg, n=11; End SAP SHR: 164 ± 4.9 vs SHR Mg:175 ± 5 mmHg, n=9). Diastolic blood pressure was not significantly affected by Mg2+ treatment. Initial and final heart rates (HR) did not show any significant differences between the groups (Initial HR SHR:360 ± 5 vs SHR Mg:345 ± 8 bpm, n=11 and End HR SHR:336 ± 7 vs SHR Mg:351 ± 5 bpm). In conclusion, the results of this study indicate that an eight-week supplementation with MgCl2 at a dose of 50 mg/kg/day did not lead to any significant changes in blood pressure or heart rate in SHR animals.
Financial support: FAPES (Fundação de Amparo à Pesquisa do Espírito Santo) e CNPq (Conselho Nacional de Desenvolvimento Científico e Tecnológico).
Ethics Committee: UFES Animal Ethics Committee 16-2020.
Key-words: Hypertension; Magnesium, Blood Pressure.

using glutamate and malate substrates. This suggests an improvement in mitochondrial function and malate-aspartate shuttle function, leading to decreased free radicals and increased ATP production for myocardial activity, at least in the short term after MI.
Financial Support: FAPES, CAPES, CNPq, UFES,
Keyword Acute Myocardial Infarction; Mitochondrial Dysfunction; Mitoquinone

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The Antioxidante Mitoquinone, Mitigates Mitochondrial Damage and Promotes Energy Efficiency in Both Interfibrillar and Subsarcolemmal Subpopulations After Myocardial Infarction

Traichel, G.A.O.; Costa, A.K.N.; Nascimento, C.L.S.; Ribeiro, E.H.; Ferreira, K.K.S.; Traichel, L.O.; Tavares, M.P.; Mendes, S.B.O.; Spalenza, T.B.; Stefanon, I; Fernandes, A. A..

Introduction: Mitoquinone (MitoQ), derived from Coenzyme Q, is an effective mitochondrial antioxidant that protects these organelles against oxidative damage and lipid peroxidation.
Objectives: to investigate the effects of MitoQ on cardiac mitochondrial metabolism in rats, seven days after myocardial infarction (MI), in the interfibrillar (IFM) and subsarcolemmal (SSM) mitochondrial subpopulations.
Methods: Male Wistar rats,10-12 weeks old, after MI surgery by ligation of the left anterior descending coronary artery or sham surgery, were divided into groups Sham (n=9), Sham MitoQ (n=10), MI (n=5), and MI MitoQ (n=6) groups (CEUA 06/2023). The animals were treated with MitoQ (100µM/Kg/day) or placebo for 7 days, in the drinking water. The mitochondrial subpopulations from the left ventricle (LV) were isolated to assess respiration, using substrates glutamate + malate (G+M), pyruvate + malate (P+M), palmitoylcarnitine + malate (Pc+M), and succinate + rotenone (S+R). The measurement of oxygen consumption from mitochondrial respiration in the State 3 indicated maximum ATP production, while State 4 referred to oxygen consumption in a resting phase, being an indirect measure of proton leak. The respiratory control ratio (RCR - State 3/State 4 ratio) indicated coupling and ATP synthesis capacity. All measurements were presented as mean ± SEM. Values were considered significant for a minimum of $p < 0.05$. For mitochondrial evaluation, statistical analysis was performed using two-way ANOVA and Fisher's post-test.
Results:In the presence of S+R substrates, the RCR of the SSM in the MI MitoQ group was reduced compared to the MI group (SSM S+R: Sham 4.83 ± 0.3; Sham MitoQ 4.2 ± 0.2; IM 5.64 ± 0.9; IM MitoQ 4.31 ± 0.2&; &p < 0.05 vs MI). However, the IFM was not different among groups (IFM S+R: Sham 4,63 ± 0,2; Sham MitoQ 4,27 ± 0,3; IM 4,33 ± 0,5; IM MitoQ 4,03 ± 0,2).
In the presence of M+G substrates, the RCR of the SSM in the MI MitoQ group was increased compared to the MI group (SSM M+G: Sham 21,66 ± 1,7; Sham MitoQ 21,96 ± 1,7; IM 14,47 ± 1,9; IM MitoQ 19,6 ± 1,2 &; &p < 0.05 vs MI) and the IFM as well (IFM M+G: Sham 18,4 ± 0,7; Sham MitoQ 19 ± 1,0; IM 15,2 ± 1,6; IM MitoQ 21,33 ± 1,9&; &p < 0.05 vs MI).
In the presence of M+P substrates, the RCR of the SSM was not different between MI groups (SSM M+P: Sham 13,2 ± 0,9; Sham MitoQ 12,94 ± 0,3; IM 8,6 ± 0,6; IM MitoQ 9,73 ± 0,6 &) and the IFM as well (IFM M+G: Sham 12,18 ± 1,4; Sham MitoQ 10,67 ± 0,8; IM 9,22 ± 0,8; IM MitoQ 9,24 ± 0,7).
In the presence of M+Pc substrates, the RCR of the SSM was not different between MI groups (SSM M+P: Sham 18,5 ± 1,3; Sham MitoQ 21,56 ± 1,4; IM 13,1 ± 1,3; IM MitoQ 13,97 ± 0,6) and the IFM as well (IFM M+G: Sham 17,1 ± 1,5; Sham MitoQ 18,27 ± 1,7; IM 14,02 ± 1,4; IM MitoQ 15,62 ± 1,2).
Conclusion: MI impacts mitochondrial energy metabolism, with the SSM subpopulation being more susceptible to damage than the IFM subpopulation. MitoQ increased ATP synthesis and reduced proton leaks in both IFM and SSM mitochondrial subpopulations when assessed using glutamate and malate substrates. This suggests an improvement in mitochondrial function and malate-aspartate shuttle function, leading to decreased free radicals and increased ATP production for myocardial activity, at least in the short term after MI.
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Keyword Acute Myocardial Infarction; Mitochondrial Dysfunction; Mitoquinone

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EFFECTS OF MAGNESIUM ON NITRIC OXIDE BIOAVAILABILITY AND VASCULAR REACTIVITY IN SPONTANEOUSLY HYPERTENSIVE RATS

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Abstract
Introduction: Magnesium is a mineral that plays a crucial role regulating blood pressure by vascular reactivity modulation, and diets rich in magnesium have been suggested to reduce hypertensive effects. Reactive Oxygen Species have an important influence in different diseases, contributing to the etiologies of atherosclerosis, inflammation and hypertension. However, the role of magnesium chloride (MgCl2) supplementation on vascular tone and reactivity and which pathway it acts upon remains unclear. Objectives: This study aimed to investigate the hypothesis that a diet rich in MgCl2 reduces vasoconstrictor response dependant on the NOX and NOS path. Methods: Thirty six-week-old male Spontaneously Hypertensive Rats (SHR), weighing between 90 and 120 grams, were divided into two groups. The SHR-Mg group received a daily dose of 50 mg/kg of MgCl2 in their drinking water for two months, while the SHR-CT group received plain water. Following the treatment period, aortic vascular reactivity was assessed in response to increasing concentrations of phenylephrine (PHE, % KCl 75 mM). The evaluation was conducted in the presence of ML171 (a selective NOX inhibitor) and L-NAME (a nitric oxide synthase inhibitor). These assessments were conducted both in the presence and absence of the endothelium (E-). Data were analysed using Student`s t-test (Animal Ethics Committee at UFES: 16-2020). Results: The maximum response (Rmax) to phenylephrine was significantly lower in the magnesium-treated group compared to the control group (SHR-Mg: 103.3±7.2, N= 8; SHR: 139.6±9.6, N=10, % KCl 75 mM, p<0.05). This difference disappeared when the endothelium was removed (E-) and NOS was inhibited (LNAME: SHR-Mg: 159.4±6.44, N=14; SHR: 145.5±7.31, N=8, % KCl 75 mM, p>0.05),(SHR-Mg E-: 172.1±15.27, N=11; SHR E-: 196.3±17.56, N=12, % KCl 75 mM, p>0.05). The NOX inhibitor (ML171) reduced the Rmax in both groups: SHR-Mg ML171: 68.24±11.34, N=7; SHR ML171: 89.16±9.91, N=10, % KCl 75 mM, p>0.05. Conclusion: The findings of the present study indicate that the magnesium chloride supplementation for two months decreases the vasoconstrictor response in aortic rings of SHR, dependent on the oxidative stress path.
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CHRONIC EXPOSURE TO MERCURY AGGRAVATES THE EFFECTS OF ACUTE MYOCARDIAL INFARCTION IN RATS

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Background: Environmental contamination has exposed humans to metal agents, including mercury. Studies suggest that chronic exposure, even to low concentration levels of mercury can affect the cardiovascular system. However, the present study evaluated whether low chronic exposure to Mercury (Hg) can increase mortality due to arrythmias in rats underwent Myocardial Infarction (MI).
Methods: Male rats (12 weeks old) were divided into four groups: Sham+Saline, Sham+Hg, MI+saline, and MI+Hg. Animals received i.m injections of HgCl2 (1st dose 4.6µg/kg, subsequent dose 0.07µg/kg/day to cover daily loss) or vehicle-saline for three weeks. At the end of the third week, the animals were submitted to infarction surgery through of ligation of the anterior descending left coronary artery. Shams were submitted the same procedure except to coronary ligation. Electrocardiographic (ECG) recordings were performed 5 minutes before and 20 minutes after surgeries. Heart rate (HR); number of ventricular extra systoles (VES); duration of ventricular tachycardia (VT) and atrioventricular blocks (AVB) were analyzed. One week after MI, hemodynamic measurements were performed to register blood and intraventricular pressures. Heart and lung were weighted and ponderal data were analyzed. Also, levels of superoxide anion (O2-) and nitric oxide (NO) in the cardiac muscle of the animal were performed through fluorescence analyses using Dihydroethidium (DHE) and Diaminofluorescein (DAF), respectively. The protocol was approved by CEUA (20/2018 and 24/2020)
Results: The scar size was not different between MI groups. The mortality rate in MI+Hg was 31.82% while MI+Saline was 21.43%. ECG recordings showed an increase in AVB in the animals from the IM (Sham+Saline= 0.00±0.00%; Sham+Hg= 0.81±0.67%; MI+Saline= 4.35±0.96%*#; MI+Hg= 3.64±0.88%*#; *p<0.05vsSham+Saline, #p<0.05vsSham+Hg). Baskó coefficient showed that arrhythmias after MI were aggravated by exposure to Hg (Sham+Saline=0.24±0.19; Sham+Hg=0.75±0.35; MI+Saline=2.97±0.30*#; MI+Hg=4.00±0.21*#+; *p<0.05vsSham+Saline, #p<0.05vsSham+Hg, +p<0 .05vsMI+Saline). Also, there was strong correlation between mortality and AVB (r=0.7379), VES (r=0.9487), VT (r=0.9487) and Baskó coefficient (r=0, 9487). Sham+Hg group has increased of the systolic blood pressure (SBP), however these values were not increased in infarcted groups (SBP: Sham+Saline=105.08±2.98mmHg; Sham+Hg=114.71±3.71*mmHg; MI+Saline=95.47±3.58mmHg*#; MI+Hg=90.01±2.96mmHg*#; *p<0.05vsSham+Saline, #p<0.05vsSham+Hg). Left Ventricle end Diastolic Pressure was increased in MI+saline group (Sham+Saline= 7.31±1.25; Sham+Hg= 5.38±1.44; MI+Saline= 15.95 ±2.84*#; MI+Hg= 9.56±1.38+; *p<0.05vsSham+Saline, #p<0.05vsSham+Hg, +p<0.05vsMI+Saline). dP/dt+ and dP/dt- values, as expected, were decreased in infarcted groups. The heart weight and body weight ratio was increased in infarcted groups as well as the lung weight and body weight ratio. O2- and NO levels were increased in groups exposed to Hg. These levels were higher when associated with MI.Conclusion: Mercury intoxication caused more arrhythmias in infarcted animals, as evidenced in this study by the increase in AVB, VES and VT. There was increase in reactive oxygen species and changes in pressure parameters. These results suggest a worsening of cardiac events triggered by MI, as well as increased mortality after the injury. Keywords: Mercury; Myocardial Infarction; Electrocardiogram.
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STI1 overexpression protects mice from adrenergic hyperactivation-induced injury

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STI1 (Stress inducible protein 1) is an important co-chaperone in protein quality control, and protein homeostasis. Recently, our research group found that STI1 expression is reduced in the hearts of heart failure patients. Furthermore, it plays a relevant role in cardiac hypertrophy and injury in mice after hypertrophic stress and reduced STI1 expression. The goal of this work was to investigate the consequences of STI1 overexpression for cardiac function in mice after hypertrophic stress induced by adrenergic hyperactivation caused by isoproterenol. Wild-type mice (WT) and transgenic mice with global overexpression of STI1 (STI1-TgA), aged 10-12 weeks, were used. The development and cardiac function were evaluated by morphological, and functional characteristics of the heart by immunofluorescence, cellular morphometry, and cardiomyocyte (CMs) contractility. Hypertrophic stress was induced by treatment with isoproterenol (ISO: i.p. 20mg/Kg/day for 7 days), and control mice were treated with saline (0.9% NaCl). Following we performed echocardiography and then the hearts were harvested for immunofluorescence, cell morphometry, western blotting, qPCR, contractility, and Ca2+ transient (Fluo-4/AM) experiments on CMs. The baseline characterization of STI1-TgA mice demonstrated a cardiac growth similar to control (WT) as assessed by the HW/TL ratio and CM morphometry. Interestingly, STI1-TgA mice showed an enhanced contractile function of CMs, which was explained by an increase in the Ca2+ transient amplitude. Hypertrophic stress caused by ISO did not induce significant hypertrophy in STI1-TgA mice (~16%), different from what occurred in WT/ISO mice which developed cardiac hypertrophy (~35%) and presented enlarged CMs. Besides, WT/ISO also showed increased mRNA levels for cardiac stress markers, such as Myh7 and ANP. CMs contractility and Ca2+ transient amplitude evaluation showed an increase in WT/ISO, similar to STI1-TgA/ISO. Collagen deposition induced by ISO was observed only in the WT/ISO group, with an increase in collagen III levels in the cardiac tissue. When we evaluated the activation of the antioxidant response by NRF2 signaling, STI1-TgA/ISO mice expressed 4x more the NRF2 transcription factor, suggesting an improved antioxidant response. Taken together, our data show that STI1 overexpression provides a framework for cardiac adaptation to stress, which in response to adrenergic hyperactivation protects the heart from cardiac ISO-induced injury, improving contractile function, and antioxidant response. Thereby, we conclude that STI1 overexpression plays a cardioprotective role and may become a relevant therapeutic target in the treatment of heart diseases.

Keywords: STI1, isoproterenol, cardiac hypertrophy, cardiac remodeling.