30º FÓRUM CIENTÍFICO – CONGRESSO INTERNACIONAL DE CIÊNCIAS CARDIOVASCULARES

22ª REUNIÃO ANUAL DA SEÇÃO SUL-AMERICANA DA ACADEMIA INTERNACIONAL DE CIÊNCIAS CARDIOVASCULARES

Hospital Meridional Vitória

Vitória, ES - Brasil, 17 a 19 de outubro de 2024
Rua Desembargador José Fortunato Ribeiro, 30 - Mata da Praia, Vitória - ES, 30200-010, Espírito Santo, Brasil.

PROMOÇÃO

SERVCOR/INSTITUTO CARDIOVASCULAR SÃO FRANCISCO DE ASSIS – ICSFA – MG
A verdade é Jesus – São João 14.6

Diretor Presidente: Prof. Dr. Otoni Moreira Gomes

REDE MERIDIONAL KORA SAÚDE

CLÍNICA CENTROCOR

Otoni Moreira Gomes MD PhD FIACS FESC

Presidente da Conferência
Melchior Luiz Lima MD PhD FIACS

Cardiovascular Surgeon
Centrocor Clinic Ltda.
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Co-presidente da conferência
Antoinette Oliveira Blackman MD PhD FIACS FESC
Cardiologist
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Co-presidente da conferência
Alexandre Ciappina Hueb MD PhD FIACS
Cardiovascular Surgeon
Hospital das Clínicas Samuel Libânio
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Minas Gerais
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Co-presidente da conferência
José Airton Arruda MD PhD SCAI EAPCI
Interventional Cardiologist
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Mensagem do Presidente
30º Congresso Internacional de Ciências Cardiovasculares
22º Encontro Anual da Seção Sul Americana da Academia Internacional de Ciências Cardiovasculares

Prezados Colegas,
É com imensa satisfação que anunciamos o 30º Congresso Internacional de Ciências Cardiovasculares com o 22º Encontro Anual da Seção Sul Americana da Academia Internacional de Ciências Cardiovasculares, organizados pelo Instituto Cardiovascular São Francisco de Assis, a serem realizados na cidade de Vitória, Espírito Santo, Brasil, entre os dias 17 e 19 de outubro de 2024. Estes eventos visam estimular e aumentar a produção científica dos novos pesquisadores na área das ciências humanas, promovendo um ambiente propício para o desenvolvimento e a disseminação de conhecimentos inovadores.
Durante três dias, especialistas de renome internacional se reunirão para compartilhar suas experiências e os mais recentes avanços em cardiologia. A programação inclui palestras magnas, apresentações de trabalhos científicos e sessões interativas, proporcionando uma plataforma única para aprendizado e troca de conhecimento.
Vitória, conhecida por suas belas praias e rica herança cultural, será o cenário perfeito para este encontro científico. Além das atividades acadêmicas, os participantes terão a oportunidade de explorar a cidade e desfrutar de sua hospitalidade e atrações turísticas.
Nosso objetivo é criar um espaço onde novos pesquisadores possam apresentar suas descobertas, interagir com profissionais experientes e estabelecer colaborações que impulsionem ainda mais a pesquisa na área das ciências cardiovasculares. Acreditamos que a participação de todos será fundamental para o sucesso deste congresso e para o avanço do conhecimento científico.
Contamos com sua presença para tornar este evento um marco na área da cardiologia. Não perca esta oportunidade de atualização profissional e de networking com os principais especialistas do setor.

Atenciosamente,

Melchior Luiz Lima MD PhD FIACS
Presidente do 30º Congresso Internacional de Ciências Cardiovasculares

Prof. Dr. Melchior Luiz Lima
Presidente do 30º Congresso Internacional de Ciências Cardiovasculares

Co-patrocínio Científico

Meridional Kora Saúde	Rede Meridional – Kora Saúde
CENTROCOR Vitória	Clínica Centrocor Ltda.
	Sociedade Brasileira de Cirurgia Cardiovascular – SBCCV Departamento de Pesquisas Experimentais – DEPEX – SBCCV Departamento de Cardiologia Clínica – DECARDIO – SBCCV
SOCIEDADE BRASILEIRA DE CARDIOLOGIA SBC MCMXLIII	Sociedade Brasileira de Cardiologia – SBC Departamento de Fisiologia Cardiovascular e Respiratória – DFCVR – SBC
CORDIS Clínica do Coração Ltda.	Clínica de Doenças do Coração – CORDIS – ES
UNIVERSIDADE FEDERAL DO ESPÍRITO SANTO DOCETE OMNES GENTES	Laboratório de Eletromecânica Cardíaca e Reatividade Vascular – UFES – ES
SOCIEDADE BRASILEIRA DE CARDIOLOGIA DO ESPÍRITO SANTO SBC ES	Sociedade Brasileira de Cardiologia do Espírito Santo – SBC/ES

Patrocínio Científico Internacional	
	International Academy of Cardiovascular Sciences South American Section – IACS
	Sociedad Peruana de Cirugia Cardiaca Toracica y Vascular – SPCCTV
	Instituto de Fisiopatología Cardiovascular – Facultad de Medicina – UBA – Argentina
	Institute of Cardiovascular Sciences Saint Boniface Hospital –University of Manitoba – Canada
	Medical Devices Innovation Institute - University of Ottawa, Canada

Ligas de Estudantes de Ciências Cardiovasculares
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

Comitê Executivo do Congresso
Diretor Presidente do Instituto Cardiovascular São Francisco de Assis
Prof. Dr. Otoni Moreira Gomes – MG
Diretora da Rede Meridional de Saúde Kora
Fábio Luiz Michiles Frank – ES
Presidente do Congresso
Prof. Dr. Melchior Luiz Lima – ES
Diretor Científico
Dr. Héber Souza Melo Silva – ES
Diretor Administrativo
Dra. Elaine Maria Gomes Freitas (OAB) – MG

Coordenação de Marketing Rede Meridional
Sra. Valéria Fracaroli – ES
Coordenação Administrativa
Sra. Ruana Freitas Marques Teixeira – MG **(Instituto Cardiovascular São Francisco de Assis - ServCor)**
Sra. Andrea Radavelli Miossi – ES

Comitê Científico e Executivo Local
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

Agenda do Programa – Quinta-feira, 17 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

07:30 a.m. – 08:00 a.m.: Cadastro

07:30 a.m. – 08:00 a.m.: Slide desk

08:00 a.m. – 08:10 a.m.: Abertura

08:10 a.m. – 08:20 a.m.: Diretor da Rede Meridional
Dr. Fábio Frank – ES

08:20 a.m. – 08:30 a.m.: Diretora Clínica do Hospital Meridional Vitória
Dra. Fátima Cristina Monteiro Pedrotti – ES

08:30 a.m. – 08:45 a.m.: Presidente do Congresso
Dr. Melchior Luiz Lima – ES

Diretor Presidente do Instituto Cardiovascular São Francisco de Assis
Prof. Dr. Otoni Moreira Gomes – MG

Diretor Científico
Dr. Héber Souza Melo Silva – ES

Diretora Administrativa
Dra. Elaine Maria Gomes Freitas (OAB) – MG

Coordenação Administrativa
Sra. Ruana Freitas Marques Teixeira – MG **(Instituto Cardiovascular São Francisco de Assis - ServCor)**
Sra. Andrea Radavelli Miossi – ES

Simpósio 1: 2º Simpósio Prof. Dr. Otoni Moreira Gomes – Simpósio de Doenças Inflamatórias e Medicina Cardiovascular

Presidente:
Prof. Dr. Elias Kallás - MG
Coordenação:
Dra. Fátima Cristina Monteiro Pedrotti– ES
Dr. José Aid Soares Sad – ES
Debatedores:
Dr. Felipe Granja Moysés – ES
Dr. José Roberto Coutinho Nogueira – ES
Dr. Carlos André Magirius Peixoto – ES
Dr. Antônio Carlos Avanza Neto – ES

Temas:

08:45 a.m. – 09:00 a.m.: Relato de Caso: Mixoma do átrio esquerdo
Palestrante: Ac./St. Amanda Klein – ES

09:00 a.m. – 09:15 a.m.: Proteção miocárdica durante cirurgia cardiovascular
Palestrante: Dr. Arthur Soares Lima – ES

09:15 a.m. – 09:30 a.m.: Relato de caso: Ampliação do anel aórtico com incisão em Y/remendo retangular
Palestrante: Ac./St. Isadora Fafá Roncete – ES

09:30 a.m. – 09:45 a.m.: Gordura epicárdica como fator de risco cardiovascular
Palestrante: Dra. Mônica de Monico Magalhães – ES

09:45 a.m. – 10:00 a.m.: Cuidados paliativos no paciente com insuficiência cardíaca
Palestrante: Dra. Adriana Lugo Ferrachini – MS

10:00 a.m. – 10:30 a.m.: Discussão

10:30 a.m. – 10:50 a.m.: Café

Agenda do Programa – Quinta-feira, 17 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

Simpósio 2: 2º Simpósio Prof. Elias Kallás – Doenças Cardiovasculares, Medicina para a Diversidade e Inovação Tecnológica

Presidente:
Prof. Dr. Elias Kallás - MG
Coordenação:
Dr. Fátima Cristina Monteiro Pedrotti– ES
Dr. José Aid Soares Sad – ES
Debatedores:
Dr. Felipe Granja Moysés – ES
Dr. José Roberto Coutinho Nogueira – ES
Dr. Antônio Carlos Avanza Neto – ES
Prof. Dr. José Wanderley Neto – AL

Tópicos:

10:50 a.m. – 11:10 a.m.: Fibrilação atrial aguda: Existe diferença entre o que é abordado no ACLS e as novas diretrizes?
Palestrante: Dr. Antônio Carlos Avanza – ES

11:10 a.m. – 11:30 a.m.: Tumores do mediastino: Técnica de enucleação para tumores gigantes
Palestrante: Prof. Dr. Elias Kallás – MG

11:30 a.m. – 11:50 a.m.: Além do consultório: Como o marketing pode impulsionar sua carreira em cardiologia?
Palestrante: Dra. Fernanda Daura Damasceno da Silva – SC

11:50 a.m. – 12:10 p.m.: Ética médica: Termo de consentimento informado em cirurgia cardíaca
Palestrante: Prof. Dr. Alfredo Aurélio Marinho Rosa – AL

12:10 p.m. – 12:30 p.m.: Evolução pós-operatória de cirurgia cardíaca com e sem circulação extracorpórea
Palestrante: Ac./St. Guilherme Kallás Hueb – MG

12:30 p.m. – 12:50 p.m.: Envelhecimento do coração: o que sabemos e o que pode ser feito para retardar sua progressão
Palestrante: Prof. Dr. Elmiro Santos Rezende – MG

12:50 p.m. – 01:10 p.m.: Construindo riqueza como um médico inteligente
Palestrante: Dr. João Lucas Abreu - BA

01:10 p.m. – 01:30 p.m.: Discussão

01:30 p.m. – 02:30 p.m.: Almoço

Agenda do Programa – Quinta-feira, 17 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
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Hospital Meridional Vitória – Kora Saúde

Simpósio 3: Envelhacimento Miocárdico – Avanços na Insuficiência Cardíaca

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

Temas:

02:30 p.m. – 02:50 p.m.: Revisitando o transplante cardíaco heterotópico
Speaker: Prof. Dr. José Wanderley Neto - AL

02:50 p.m. – 03:10 p.m.: Desafios no transplante cardíaco: agora e no futuro
Speaker: Prof. Dr. Fábio Antônio Gaiotto – SP

03:10 p.m. – 03:30 p.m.: Avanços modernos no transplante de coração
Speaker: Prof. Dr. Juan Alberto Cosquillo Mejia – CE

03:30 p.m. – 03:50 p.m.: Logística e interação das equipes aeromedicas e de captação de órgãos vitais
Speaker: Ten. Cel. Daniel Quintella – ES

03:50 p.m. – 04:10 p.m.: Discussão

04:10 p.m. – 04:25 p.m.: Café

Simpósio 4: Simpósio de Avanços em Ciências Cardiovasculares

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

Temas:

04:25 p.m. – 04:45 p.m.: Plastia Valvar Mitral: Indicações e Técnicas – Porque é o Padrão Ouro de Tratamento?
Speaker: Prof. Dr. Sérgio Lima de Almeida – SC

04:45 p.m. – 05:05 p.m.: Tratamento cirúrgico da valvopatia aórtica: opções e desafios
Speaker: Prof. Dr. Edmo Atique Gabriel – SP

05:05 p.m. – 05:25 p.m.: Tratamento cirúrgico da insuficiência valvar mitral com duplicação do folheto posterior
Speaker: Prof. Dr. Mário Cézar Santos de Abreu – BA

Agenda do Programa – Quinta-feira, 17 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

05:25 p.m. – 05:45 p.m.: Protocolo ERAS e as técnicas minimamente invasivas na evolução do pós-operatório em cirurgia cardiovascular
Speaker: Prof. Dr. Daniel Bregonci Trancoso Wernsbach – MG

05:45 p.m. – 06:05 p.m.: Tratamento cirúrgico da estenose valvar aórtica versus TAVI: Qual método mais indicado?
Speaker: Prof. Dr. Samir Saadeddine Júnior – DF

06:05 p.m. – 06:25 p.m.: Risco residual na doença cardiovascular aterosclerótica: papel da inflamação
Speaker: Prof. Dr. Fernando Luiz Torres Gomes – ES

06:25 p.m. – 06:40 p.m.: Discussão

Conferência de abertura do Congresso

Presidente:
Dra. Fátima Cristina Monteiro Pedrotti– ES, Brasil
Coordenação:
Prof. Dr. Grant Norval Pierce, Canadá
Prof. Dr. Melchior Luiz Lima – ES, Brasil

Tema:

06:40 p.m. – 07:00 p.m.: Marcos Mundiais da Academia Internacional de Ciências Cardiovasculares
Speaker: Prof. Dr. Naranjan S. Dhalla – Canadá

07:00 p.m.: Encerramento

Agenda do programa – Sexta-feira, 18 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

07:30 a.m. – 08:00 a.m.: Cadastro

07:30 a.m. – 08:00 a.m.: Slide desk

Simpósio 1: 22º Simpósio Prof. Naranjan S. Dhalla (Módulo I)
Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Avanços em Ciências Cardiovasculares, Pesquisa Experimental e Iniciação Científica de Estudantes

Presidente:
Prof. Dr. Naranjan S. Dhalla, Canadá
Coordenação:
Prof. Dr. Ricardo Jorge Gelpi – Buenos Aires, Argentina
Prof. Dr. Samir Saadeddine Júnior – DF, Brasil
Debatedores:
Prof. Dr. Ramesh Goyal – Índia
Prof. Dr. Grant Norval Pierce, Canadá
Prof. Dr. Dalton Valentim Vassallo – UFES, Brasil
Prof. Dr. Lorrie Kirshenbaum - Canadá

Temas:

08:00 a.m. – 08:20 a.m.: Fisiopatologia da Disfunção Cardíaca: uma breve viagem pelo interior do cardiomiócito
Speaker: Profa. Dra. Ivanita Stefanon, UFES, Brasil

08:20 a.m. – 08:40 a.m.: Mecanismos para perda de suporte adrenérgico na insuficiência cardíaca por infarto do miocárdio
Speaker: Prof. Dr. Naranjan S. Dhalla – Canadá

08:40 a.m. – 09:00 a.m.: Ação antioxidante mitocondrial da mitoquinona no metabolismo mitocondrial cardíaco após infarto agudo do miocárdio
Speaker: Profa. Dra. Georgia Azevedo Oliveira Traichel MD (Master's degree student in the Biochemistry Graduate Program, UFES, Brasil

09:00 a.m. – 09:20 a.m.: Treinamento de resistência restaura a função cardíaca, melhorando o metabolismo mitocondrial em condições de deficiência hormonal
Speaker: Prof. Dr. Eduardo Hertel Ribeiro, UFES, Brasil

09:20 a.m. – 09:40 a.m.: Sinalização de imunidade inata na cardiomiopatia por doxorrubicina
Speaker: Prof. Dr. Lorrie Kirshenbaum – Canadá

09:40 a.m. – 10:00 a.m.: O papel da administração combinada de antagonista do receptor AT1 e inibidor da neprilisina na promoção do escurecimento em modelo experimental de síndrome metabólica
Speaker: Prof. Dr. Vladimir Jakovljevic - Sérvia

10:00 a.m. – 10:20 a.m.: Discussão

10:20 a.m. – 10:40 a.m.: Café

Agenda do programa – Sexta-feira, 18 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
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Hospital Meridional Vitória – Kora Saúde

Sessão de Comunicação oral

Presidente:
Prof. Dr. Grant Norval Pierce, Canadá
Coordenação:
Profa Dra Ivanita Stefanon – UFES, Brasil
Prof. Dr. Dalton Valentim Vassallo – UFES, Brasil
Debatedores:
Prof. Dr. Naranjan S. Dhalla – Canadá
Prof. Dr. Ramesh Goyal – Índia
Profa Dra Ivanita Stefanon – UFES, Brasil
Prof. Dr. Henrique Barsanulfo Furtado – TO, Brasil

Temas / Themes:

10:40 a.m. – 11:00 a.m.: Exposição crônica ao mercúrio aumenta arritmia e mortalidade pós-infarto agudo do miocárdio em ratos
Speaker: Profa. Dra. Aurélia Araujo Fernandes, UFES, Brasil

11:00 a.m. – 11:20 a.m.: A privação androgênica de longo prazo modula a reatividade vascular dependente da co-regulação endotelial da aldosterona e da angiotensina II
Speaker: Profa. Anna Karolina Nascimento Costa (Ph.D. Student, UFES), Brasil

11:20 a.m. – 11:35 a.m.: Espécies reativas de oxigênio mitocondrial, ciclooxigenase-1 e NADPH oxidase-4 estão envolvidas na disfunção vascular precoce após infarto do miocárdio
Speaker: Ac/St. Marlon R. Machado (Undergraduate Research Student, 10th semester of Medicine), UFES, Brasil

11:35 a.m. – 11:50 a.m.: O impacto da mitoquinona na remodelação vascular precocemente após o infarto do miocárdio
Speaker: Sara Bianca Oliveira Mendes (Undergraduate Research Student, Graduate Program in Physiological Sciences), UFES, Brasil

11:50 a.m. – 12:05 p.m.: Efeito da administração de zinco na função vascular na aorta de ratos expostos agudamente ao cádmio
Speaker: Camilla Loren da Silva Nascimento (Master's degree student in the Physiological Sciences Post Graduate Program), UFES, Brasil

12:05 p.m. – 12:20 p.m.: A superexpressão de STI1 protege camundongos de lesões induzidas por hiperativação adrenérgica
Speaker: Dra Katyana Kaline S. Ferreira (Post-doctoral fellow, Physiological Sciences Post Graduate Program), UFES, Brasil

12:20 p.m. – 12:50 p.m.: Herpes Zoster e risco cardiovascular
Speaker: Dra. Mariana Camargo Afiune – HUB, DF

12:50 p.m. – 01:05 p.m.: Manifestações cardiovasculares da Dengue
Speaker: Ac./St. Luiz Fernando Arantes de Souza – CEUB, DF

01:05 p.m. – 01:30 p.m.: Discussão

01:30 p.m. – 02:30 p.m.: Almoço

01:30 p.m. – 02:30 p.m.: Encontro de Negócios Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Local: Sala de Diretoria do Hospital Meridional Vitória
Diretor-executivo (IACS): Prof. Dr. Naranjan S. Dhalla, Winnipeg, Canadá
Presidente (IACS): Prof. Dr. Grant Norval Pierce, Winnipeg, Canadá
Membros da Seção Sul-Americana (IACS): Melchior Luiz Lima (Brasil), Alexandre Ciappina Hueb (Brasil), Ricardo Jorge Gelpi (Argentina), Elaine Gomes Freitas (Brasil), Elias Kallás (Brasil) e Enrique Castañeda Saldana (Perú).

Agenda do programa – Sexta-feira, 18 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

Simpósio 2: 22º Simpósio Prof. Naranjan S. Dhalla (Módulo II)
Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Avanços em Ciências Cardiovasculares, Pesquisa Experimental e Iniciação Científica de Estudantes

Presidente:
Prof. Dr. Grant Norval Pierce, Canadá
Coordenação:
Prof. Dr. Dalton Valentim Vassallo – UFES, Brasil
Profa Dra Ivanita Stefanon – UFES, Brasil
Debatedores:
Prof. Dr. Naranjan S. Dhalla – Canadá
Prof. Dr. Lorrie Kirshenbaum – Canadá
Prof. Dr. Henrique Barsanulfo Furtado – TO, Brasil
Prof. Dr. Ramesh Goyal – Índia

Temas:

02:30 p.m. – 02:50 p.m.: Abordagem de engenharia reversa para descoberta de novos medicamentos a partir de recursos naturais para complicações cardiovasculares induzidas por diabetes
Speaker: Prof. Dr. Ramesh Goyal – Índia

02:50 p.m. – 03:10 p.m.: Endotelização valvar aprimorada após substituição transcateter da válvula aórtica (TAVR)
Speaker: Prof. Dr. Michael Kutryk – Canadá

03:10 p.m. – 03:30 p.m.: Consideração séria sobre o papel dos suplementos dietéticos na prevenção e tratamento de doenças cardiovasculares
Speaker: Prof. Dr. Grant Norval Pierce – Canadá

03:30 p.m. – 03:40 p.m.: Doença Genética Arritmogênica Devida a Mutações no Receptor de Ryanodina Cardíaco (RyR2): Taquicardia Ventricular Polimórfica Catecolaminérgica (TVPC)
Speaker: Profa. Dra. Ivanita Stefanon – ES

03:40 p.m. – 04:00 p.m.: Exossomos derivados de células-tronco embrionárias modulam a toxicidade induzida por medicamentos anticâncer
Speaker: Prof. Dr. Dinender K. Singla – USA

04:00 p.m. – 04:20 p.m.: Discussão

04:20 p.m. – 04:45 p.m.: Café

Simpósio 3: Avanços em Medicina Cardiovascular

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

Temas:

04:45 p.m. – 05:05 p.m.: Marcapasso cardíaco no dia a dia: orientações para dúvidas frequentes no consultório
Speaker: Prof. Dr. Jorge Elias Neto – ES

Agenda do programa – Sexta-feira, 18 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

05:05 p.m. – 05:25 p.m.: Variações técnicas para as cirurgias de Fontan e Norwood
Speaker: Prof. Dr. José Teles de Mendonça - SE

05:25 p.m. – 05:45 p.m.: Estado atual da autotransfusão em cirurgia cardiovascular
Speaker: Prof. Dr. Alfredo Inácio Fiorelli – SP

05:45 p.m. – 06:05 p.m.: Cirurgia Cardiovascular: Visão pessoal de um Cirurgião!
Speaker: Prof. Dr. Henrique Barsanulfo Furtado – TO

06:05 p.m. – 06:25 p.m.: Efeitos do ar poluído na fisiopatologia da isquemia miocárdica
Speaker: Prof. Dr. Ricardo Jorge Gelpi, Buenos Aires – Argentina

06:25 p.m. – 06:40 p.m.: Discussão

06:40 p.m. – 07:30 p.m.: Cerimônia de entrega de prêmios
Encontro para Anunciação de Reconhecimento e Entrega de Prêmios pela Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares

Local: Auditório do Hospital Meridional Vitoria
Presidente: Prof. Dr. Naranjan S. Dhalla
Observação de felicitações: Prof. Dr. Naranjan S. Dhalla e Prof. Dr. Grant Norval Pierce.
Apresentação dos Prêmios IACS: Prof. Dr. Grant Norval Pierce, Dr. Melchior Luiz Lima, Prof. Dr. Elias Kallás, Prof. Dr. Samir Saadeddine Júnior, Profa. Dra. Antoinette Oliveira Blackman, Prof. Dr. Henrique Furtado, e Profa. Dra. Elaine Maria Gomes Freitas.
Membros da Seção Sul-Americana - IACS: Melchior Luiz Lima (Brasil), Alexandre Ciappina Hueb (Brasil), Ricardo Jorge Gelpi (Argentina), Elaine Gomes Freitas (Brasil), Elias Kallás (Brasil) and Ramesh Goyal (Índia), Vladimir Jakovljevic (Serbia), Michael Kutryk (Canadá), e Lorrie Kirshenbaum (Canadá).
Prêmios de pesquisa científica (pôster/oral):
Prof. Dr. Naranjan Dhalla, Prof. Dr. Grant Norval Pierce, Dr. Melchior Luiz Lima, Prof. Dr. Elias Kallás, Prof. Dr. Samir Saadeddine Júnior, Profa. Dra. Antoinette Oliveira Blackman, Prof. Dr. Henrique Furtado, and Profa. Dra. Elaine Maria Gomes Freitas.

07:30 p.m.: Encerramento

Agenda do programa – Sábado, 19 de outubro de 2024
30º Fórum Científico – Congresso Internacional de Ciências Cardiovasculares
22ª Reunião Anual da Seção Sul-Americana da Academia Internacional de Ciências Cardiovasculares
Hospital Meridional Vitória – Kora Saúde

07:30 a.m. – 08:00 a.m.: Cadastro

07:30 a.m. – 08:00 a.m.: Slide desk

Simpósio 1: Evolução do processo anestésico em Ciências Cardiovasculares e Iniciação Científica de Estudantes

Presidente:
Prof. Dr. Ricardo Jorge Gelpi – Buenos Aires, Argentina
Coordenação:
Dra. Antoinette Oliveira Blackman – DF
Prof. Dr. Antônio Carlos Avanza Neto – ES
Debatedores:
Dr. Ricardo Adala Benfatti – MS
Dr. Vítor Arantes Pazolini – ES
Dr. Fabrício Ribeiro França – ES
Dr. Henrique Barsanulfo Furtado – TO

Temas:

08:00 a.m. – 08:15 a.m.: Transplante Cardíaco no ES: Experiência e visão histórica
Speaker: Prof. Dr. Pablo Gusmann – ES

08:15 a.m. – 08:30 a.m.: Comparação em analgesia pós-operatória: Esp Block versus PCA venoso de morfina
Speaker: Dr. Heitor Cunha Lima – ES

08:30 a.m. – 08:45 a.m.: Fisiologia aplicada aos dispositivos de assistência ventricular: manejo anestésico
Speaker: Dr. Bruno Campostrini Sily – ES

08:45 a.m. – 09:00 a.m.: Protocolo ERAS em Cirurgia Cardíovascular
Speaker: Prof. Dr. Pablo Gusmann – ES

09:00 a.m. – 09:15 a.m.: Discussão

Simpósio 2: Simpósio de Cirurgia Cardíaca Minimamente Invasiva – Nova Técnica Cirúrgica e Novas Tecnologias em Cirurgia Cardiovascular

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

Temas:

09:15 a.m. – 09:35 a.m.: Abordagem híbrida na revascularização do miocárdio minimamente invasiva
Speaker: Dr. Milton de Miranda Santoro – PR

09:35 a.m. – 09:55 a.m.: Como faço a revascularização miocárdica minimamente invasiva sem CEC?
Speaker: Dr. Ricardo Adala Benfatti – MS

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09:55 a.m. – 10:15 a.m.: Revascularização do miocárdio minimamente invasiva
Speaker: Dr. Rodrigo Mussi Milani – PR

10:15 a.m. – 10:30 a.m.: Discussão

10:30 a.m. – 11:00 a.m.: Café

Simpósio 3:Doença inflamatória e envelhecimento do miócito

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

Temas:

11:00 a.m. – 11:20 a.m.: Tromboembolismo pulmonar
Speaker: Prof. Dr. Alexandre Barbosa Andrade – MG

11:20 a.m. – 11:40 a.m.: Atualidades em cardiometabolismo renal
Speaker: Dra. Tatiane Mascarenhas Santiago Emerick – ES

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

12:00 p.m. – 12:20 p.m.: Morte súbita na doença de Chagas e no prolapso mitral: o que há de comum e importante na sua fisiopatologia
Speaker: Prof. Dr. Glauco André Machado – DF

12:20 p.m. – 12:40 p.m.: Uso do fibrinogênio no pós-operatório de cirurgia complexas
Speaker: Prof. Dr. Alexandre Ciappina Hueb – MG

12:40 p.m. – 01:00 p.m.: Modulação metabólica do Cardiomiócito: Fisiologia e aplicação clínica
Speaker: Prof. Dr. Samir Saadeddine Júnior – DF

01:00 p.m. – 01:20 p.m.: Síndrome de Ehler-Danlos: necessidade de um olhar criterioso para o diagnóstico
Speaker: Profa. Dra. Antoinette Oliveira Blackman – DF

01:20 p.m. – 01:30 p.m.: Discussão

01:30 p.m. – 02:30 p.m.: Almoço

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Simpósio 4: Prevenindo o AVC relacionado à Fibrilação Atrial – novas fronteiras e conceitos

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

Temas:

02:30 p.m. – 02:45 p.m.: Fibrilação Atrial após Troca Valvar Aórtica – Incidência, preditores, tratamento
Speaker: Dra. Fernanda Bento de Oliveira Viana - ES

02:45 p.m. – 03:00 p.m.: Oclusão cirúrgica do apêndice atrial esquerdo – Quando e como fazer?
Speaker: Dr. Melchior Luiz Lima – ES

03:00 p.m. – 03:15 p.m.: Fechamento do apêndice atrial esquerdo: uma alternativa segura na prevenção do AVC cardioembólico?
Speaker: Dr. José Airton Arruda - ES

03:15 p.m. – 03:30 p.m.: Fechar o AAE simultaneamente a TAVI – Prós e Contras
Speaker: Dr. Alfredo Nunes Ferreira Neto – ES

03:30 p.m. – 03:45 p.m.: Caso clínico interativo de fechamento do apêndice atrial esquerdo
Speaker: Prof. Dr. Vitor Arantes Pazolini – ES

03:45 p.m. – 04:00 p.m: Discussão

04:00 p.m. – 04:15 p.m: Café

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Simpósio 5: Reparação Cardiovascular e Medicina Regenerativa (Módulo I)

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

Temas:

04:15 p.m. – 04:30 p.m.: Relato de caso: Conduta frente à rejeição após transplante cardíaco
Speakers: Dr. Felipe Ribeiro do Val Tommasi Silva – ES / Dr. Marcus Aguilar Constantino – ES

04:30 p.m. – 04:45 p.m.: Morte Súbita no Esporte e Exercício: Qual a melhor forma de Prevenção?
Speaker: Prof. Dr. Antônio Carlos Avanza – ES

04:45 p.m. – 05:00 p.m.: Relato de caso: dissecção aguda da aorta "tipo A" de Stanford e gestação
Speakers: Dra. Letícia Bonacossa Ferrari / Dra. Mirella Lourencini Palaoro – ES

05:00 p.m. – 05:15 p.m.: O papel do estresse psicológico no desenvolvimento das doenças cardiovasculares
Speaker: Dr. João Eugênio Alvarenga Neto – BA

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

05:30 p.m. – 05:45 p.m.: Discussão

06:00 p.m.: Encerramento

Agenda do programa – Sábado, 19 de outubro de 2024
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SESSÃO DE POSTERS 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

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.

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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.
(1) Federal University of Espirito Santo, Department of Physiological Sciences, Health Sciences Center, Vitoria, ES, Brazil.
(2) Federal University of Espirito Santo, Department of Morphology, Health Sciences Center, Vitoria, ES, Brazil.
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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

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.
1Postgraduate Program in Physiological Sciences, Federal University of Espírito Santo, Vitória/ES, Brazil.

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.

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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.
(1) Federal University of Espirito Santo, Department of Physiological Sciences, Health Sciences Center, Vitoria, ES, Brazil.
(2) Federal University of Espirito Santo, Department of Morphology, Health Sciences Center, Vitoria, ES, Brazil.
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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 Cardiovascular Physiology

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.
(1) Universidade Federal do Espírito Santo (UFES) – Vitória (ES, Brasil)
(2) Centro Universitário Multivix – Vitória (ES, Brasil)

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-Mg^2 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.

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Hospital Meridional Vitória – Kora Saúde

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

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

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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.

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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
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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 Cardiovascular Physiology

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

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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-Mg^2 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.

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

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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.
Financial Support: FAPES, CAPES, CNPq, UFES,
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

Livia Seif Eddine1,2; Livia Suzano de Paula dos Santos1,2; Paula dos Santos Athayde2; Michelle Rossana Martins Hortelan2; Aurélia Araujo Fernandes2; Ivanita Stefanon2
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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.
Financial Support: FAPES (Fundação de Amparo à Pesquisa do Espírito Santo).

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). Basckó 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 Basckó 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.
Financial Support: CAPES, CNPq, UFES, FAPES

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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.