Programa/Program

42º Congresso Brasileiro de Farmacologia e Terapêutica Experimental

42nd Brazilian Congress of Pharmacology and Experimental Therapeutics

Centro de Convenções de Ribeirão Preto
Ribeirão Preto, SP
18-21 de outubro de 2010

Mensagem do Presidente

Prezado(a) Congressista,

Em nome da Diretoria da SBFTE dou as boas-vindas ao Congresso de 2010. Dedicamos nossos melhores esforços para proporcionar a todos um Congresso abrangente e interessante. Este Congresso é o resultado de um ano de duro trabalho da Diretoria, do Conselho, da Secretaria da SBFTE e da Eventus, a empresa que organiza as atividades, pelo que já os agradeço aqui. Nossos patrocinadores, Biolab-Sanus e Atem & Remer como sempre, tornaram possível a existência dos Prêmios José Ribeiro do Valle e Inovação que já se tornaram partes nobres do calendário científico da nossa Sociedade. Nossos efusivos agradecimentos a eles. Da mesma forma, faço um agradecimento especial às empresas que aceitaram nosso convite e trouxeram seus estandes para mostrar seus produtos e serviços. Gostaria também de agradecer os nossos patrocinadores institucionais CNPq, CAPES, FAPESP e FAPERJ. Finalmente, agradeço aos avaliadores de Resumos e de Painéis e aos membros das diversas Comissões. Cada um deles despendeu uma parcela importante do seu tempo para que você tenha um Congresso proveitoso. Imperfeições são inerentes a um evento tão complexo como este. Por favor, mandem suas críticas e sugestões para o email sbfte@sbfte.org.br.
O Programa de 2010 está mais denso e vamos usar até parte do horário do almoço para atividades. É a conseqüência de estarmos crescendo e melhorando.
Como já informado o Congresso de 2011 será feito em conjunto com a FeSBE na cidade do Rio de Janeiro. A Comissão Científica multi-Sociedade que está montando a programação de atividades está construindo um programa de alto nível e multidisciplinar. Estamos antecipando que o Congresso Conjunto de 2011 será muito proveitoso.
Desejo a você um excelente Congresso e uma ótima estada em Ribeirão Preto. Aproveite para conhecer gente nova, rever os amigos e começar uma colaboração científica.

Um abraço,
Jamil Assreuy
Presidente do 42o Congresso Brasileiro de Farmacologia e Terapêutica Experimental

Message from the President

Dear Colleague,

On behalf of the Brazilian Society of Pharmacology and Experimental Therapeutics I welcome you to the 2010 Congress. We made our best efforts to build an interesting and attractive Congress. It is the result of one year of hard work by the Board of Directors, by the Council, Executive Secretariat and Eventus. I very much thank all these people for their efforts. A big “Thank you” goes to Biolab-Sanus and Atem & Remer who kindly support the two Awards given by the Society, the Prof José Ribeiro do Valle Award and the Innovation Award. I also have to express my gratitude to the companies of scientific equipments and reagents which accepted our invitation to show their products and services. My thanks also to our institutional supporters CNPq, CAPES, FAPESP and FAPERJ. Finally, I thank very much to the Abstract and Poster reviewers whom spend a considerable time and effort to ensure that our standards are met. An event this size is likely to have some imperfections. We very much appreciate your feedback, comments and suggestions to the email sbfte@sbfte.org.br.
The 2010 Scientific Program is denser and we will use part of the lunch time for activities. This is the consequence of the Society increase in size and quality.
As already informed, the 2011 Congress will be held in Rio de Janeiro together with the Brazilian Federation of Experimental Biology Societies. A multi-Society scientific Committee is building up a high level program and we are sure that this event will be a success.
I wish you an excellent Congress and a very nice stay in Ribeirão Preto. Take your time to meet old and new friends.

My best regards,
Jamil Assreuy
President of the Congress

Support Agencies

Enterprise Support

Sponsor José Ribeiro do Valle Award

Collaborator Innovation Award

Exhibitors

ADInstruments
AVS Projetos
Delta Livros
Inova
Insight Equipamentos Científicos – Ensino e Pesquisa
Novalab Científica Ltda.

Support

Sociedade Brasileira de Farmacologia e Terapêutica Experimental
sbfte@pratica.com.br / sbfte@sbfte.org.br
www.sbfte.org.br

Organization

Eventus Planejamento e Organização
Rua Marquês de Itu, 408, cj.34/35
São Paulo SP
CEP 01223-000
www.eventus.com.br
eventus@eventus.com.br
Telefone 11 3361 3056
Fax 11 3361 3089

Revisão, **Supervisão e Editoração**
Sandra Helena Rocha da Cruz

***C**ongress **P**resident*
Jamil Assreuy (UFSC)

***S**cientific **C**ommittee*
Cristoforo Scavone (USP)
João Batista Calixto (UFSC)
Lusiane M. Bendhack (USP)
Maria Christina W. de Avellar (UNIFESP)
Rosely Oliveira Godinho (UNIFESP, Coordenadora)

***O**rganizing **C**ommittee*
Jamil Assreuy (UFSC, Coordenador)
Mauro Martins Teixeira (UFMG)
Rosely Oliveira Godinho (UNIFESP)
Teresa C. T. Dalla Costa (UFRGS)
Ronaldo A. Ribeiro (UFC)

***P**oster **C**ommittee*
Ana Lucia de Aguiar Pires (FIOCRUZ, Secretária)
Patrícia M. R. S. Martins (FIOCRUZ, Coordenadora)
Sandra Helena P. Farsky (USP)
Teresa C. T. Dalla Costa (UFRGS)

***I**nnovation **A**ward **C**ommittee*
Fernando de Queiroz Cunha (USP)
Mauro M. Martins (UFMG)
Ricardo Amaral Remer
Regina Pekelmann Markus (USP, Coordenadora)

***J**osé **R**ibeiro do **V**alle **A**ward **C**ommittee*
Giles A. Rae (UFSC, Coordenador)
Gloria E. P. de Souza (USP)
Soraya S. Smaili (UNIFESP)

***L**ocal **C**ommittee*
Fernando de Queiroz Cunha (USP)
Glória E. P. de Souza (USP)

***A**bstract **R**eviewers*
Adair R. S. Santos (UFSC)
Adriana Lino dos Santos (USP)
Albetiza Lobo Araújo (UNICAMP)
Aleksander Roberto Zampronio (UFPR)
Andrea Grabe Guimarães (UFOP)
Antonio Claudio Lucas da Nóbrega (UFF)
Bagnólia Araújo da Silva (UFPB)
Bruno Lourenço Diaz (UFRJ)
Caden Souccar (UNIFESP)
Carlos Renato Tirapelli (USP)
Catarina Segreti Porto (UNIFESP)
Christianne Bandeira de Melo (UFRJ)
Ciomar A Bersani-Amado (UEM)
Cláudia F. Benjamim (UFRJ)
Cláudia Lúcia M. Silva (UFRJ)
Cláudia Padovan (USP)
Cláudia do Ó Pessoa (UFC)
Claudio Miguel Costa Neto (USP)
Cristina Setim Freitas (UFPR)
Cristóforo Scavone (USP)
Daniella Bonaventura (USP)
Danielle da Gloria de Souza (UFMG)
Edgar Paredes Gamero (UNIFESP)
Edson Antunes (UNICAMP)
Eduardo Vera Tibiriçá (FIOCRUZ)
Eliezer J. Barreiro (UFRJ)
Elisa Mitiko Kawamoto (USP)
Emer S. Ferro (USP)
Emiliano de Oliveira Barreto (UFAL)
Francisco J. R. Paumgarten (FIOCRUZ)
Fúlvio R. Mendes (UNIFESP)
Gilberto de Nucci (UNICAMP/USP)
Giles A. Rae (UFSC)
Glória E. P. de Souza (USP)
Gustavo Ballejo Oliveira (USP)
Iolanda M. Fierro (UERJ)
Isac A. de Medeiros (UFPB)
Jamil Assreuy (UFSC)
João Ernesto de Carvalho (UNICAMP)
José Eduardo Tanus-Santos (USP)
José Francisco Fracasso (UNESP-Araraquara)
Josélia A. Lima (UFRJ)
Leticia Veras Costa-Lotufo (UFC)
Magda Fráguas Serra (FIOCRUZ)
Manoel Odorico de Moraes (UFC)
Marcelo N. Muscará (USP)
Marco Aurélio Martins (FIOCRUZ)
Maria Cristina de O. Salgado (USP)
Maria de Fátima M. Lazari (UNIFESP)
Maria Fernanda de Paula Werner (UFPR)
Maria Martha Campos (PUCRS)
Marília Zaluar P. Guimarães (UFRJ)
Newton G. de Castro (UFRJ)
Norma Yamanouye (Instituto Butantan)
Patricia M. R. S. Martins (FIOCRUZ)
Patrícia Torres Bozza (FIOCRUZ)
Paulina Sannomiya (USP)
Paulo de Assis Melo (UFRJ)
Pedro Paulo X. Elsas (UFRJ)
Renato S. B. Cordeiro (FIOCRUZ)
Rita de Cássia Elias Estrela (INCa)
Roberto Takashi Sudo (UFRJ)
Ronaldo A. Ribeiro (UFC)
Rosana Camarini (USP)
Rosane Vianna-Jorge (UFRJ)
Rosely Oliveira Godinho (UNIFESP)
Rui D. S. Prediger (UFSC)
Sandra Aurora C. Perez (FIOCRUZ)
Soraia Katia Costa Pereira (USP)
Stela Maris K. Rates (UFRGS)
Stella Regina Zamuner (UNINOVE)
Tania Araújo Viel (USP)
Teresa C. T. Dalla Costa (UFRGS)
Thereza C. Barja Fidalgo (UERJ)
Valber da Silva Frutuoso (FIOCRUZ)
Valéria do Monti N. Cunha (UFRJ)
Vinícius Frias Carvalho (FIOCRUZ)
Wothan Tavares de Lima (USP)
Yara Cury (Instituto Butantan)
Zuleica Bruno Fortes (USP)
Zulma Silva Ferreira (USP)

***O**n-site **P**oster **R**eviewers*

Adair Roberto Soares dos Santos (UFSC)
Albetiza Lobo Araújo (UNICAMP)
Aleksander Roberto Zampronio (UFPR)
Alessandra Beirith (FURB)
América Scopinho de Oliveira (USP)
Ana Luisa Palhares de Miranda (UFRJ)
André Klein (UFMG)
André Sampaio Pupo (UNESP-Botucatu)
Andrea Carla Pessini (USP)
Ângela de Castro Resende (UERJ)
Bibiana Verlindo de Araújo (UFRGS)
Bruno S. Herrera (UNESP-Araraquara)
Caden Souccar (UNIFESP)
Carolina Baraldi Araújo Restini (USP)
Carolina Demarchi Munhoz (USP)
Célia Aparecida Paulino (UNIBAN)
Christianne Brêtas Vieira Scaramello (UFF)
Ciomar A. Bersani-Amado (UEM)
Cláudia F. Benjamim (UFRJ)
Cláudia Padovan (USP)
Cláudia do Ó Pessoa (UFC)
Cristiano Gonçalves Ponte (IFRJ)
Cristina Antoniali Silva (UNESP-Araçatuba)
Cristina Bichels Hebeda (USP)
Cristina Maria Fernandes (Instituto Butantan)
Cristina Setim Freitas (UFPR)
Cristóforo Scavone (ICB)
Daniel Fernandes (UEPG)
Daniela Dal Secco Abbud (UFSC)
Daniela de Almeida Cabrini (UFPR)
Daniella Bonaventura (USP)
Disney Oliver Sivieri Jr (UFVJM)
Edna Alves dos Anjos-Valotta (FIOCRUZ)
Edson Antunes (UNIFESP)
Eduardo Tamura (USP)
Edvaldo Rodrigues de Almeida (UFPE)
Elena Malpezzi-Marinho (UBC)
Eliana Horomi Akamine (USP)
Emiliano de Oliveira Barreto (UFAL)
Enilton Aparecido Camargo (UFS)
Fabíola de Almeida Brito (UFAL)
Fabrício Alano Pamplona (UFSC)
Fernanda B. Marinho Priviero (UNESP-Rio Claro)
Fernanda Regina Castro Almeida (UFPI)
Fernando de Queiroz Cunha (USP)
Flavio Aimberê Soares de Carvalho (UNICASTELO)
François G. Noel (UFRJ)
Gilberto de Nucci (UNICAMP/USP)
Giles A. Rae (UFSC)
Gloria E. P. de Souza (USP)
Helena Serra Azul Monteiro (UFC)
Heloisa Helena Araújo Ferreira (USF)
Hudson S. Buck (FCMSCSP)
Hugo Caire de Castro Faria Neto (FIOCRUZ)
Janetti Nogueira de Francischi (UFMG)
João Ernesto de Carvalho (UNICAMP)
José Francisco Fracasso (UNESP-Araraquara)
Josélia A. Lima (UFRJ)
Juliano Ferreira (UFSM)
Laura Letícia Souza (USP)
Leonardo Resstel Barbosa Moraes (USP)
Letícia Lintomen (UNICAMP)
Leticia Veras Costa-Lotufo (UFC)
Lusiane Maria Bendhack (USP)
Marcelo Gomes Damário (USP)
Marcelo N. Muscará (USP)
Maria de Fátima M. Lazari (UNIFESP)
Maria Fernanda de Paula Werner (UFPR)
Maria Martha Campos (PUCRS)
Marília Zaluar P. Guimarães (UFRJ)
Michel Fleith Otuki (UFPR)
Newton G. de Castro (UFRJ)
Patrícia Torres Bozza (FIOCRUZ)
Paulo César Ghendini (UFG)
Paulo de Assis Melo (UFRJ)
Pedro Augusto Carlos Magno Fernandes (USP)
Rita Tostes (USP)
Roberto César Pereira Lima Júnior (UFC)
Roberto Kenji Nakamura Cuman (UEM)
Ronaldo A. Ribeiro (UFC)
Rosa M. Ribeiro do Valle Nicolau (UFSC)
Rosana I. Reis (USP)
Rosely Oliveira Godinho (UNIFESP)
Rui D. S. Prediger (UFSC)
Sandra Helena P. de Oliveira (UNESP-Araçatuba)
Silvia Dal Bó (UFSC)
Simone A. Teixeira (USP)
Soraia Katia Costa Pereira (USP)
Stela Maris K Rates (UFRGS)
Stella Regina Zamuner (UNINOVE)
Tania Araújo Viel (USP)
Tarcilia Aparecida Silva (USP)
Tereza C. T. Dalla Costa (UFRGS)
Thereza Christina Barja Fidalgo (UERJ)
Thiago Mattar Cunha (USP)
Valber da Silva Frutuoso (FIOCRUZ)
Valéria do Monti N. Cunha (UFRJ)
Valquíria Camin Bortoli (USP)
Vinícius Frias Carvalho (FIOCRUZ)
Waldiceu Aparecido Verri Jr. (UEL)
Yara Cury (Instituto Butantan)
Zulma Silva Ferreira (USP)

Index

Informações úteis

Painéis
A Sessão de Painéis será realizada nos dias 19/10 das 18h30 as 20h30; 20/10 das 18h00 as 20h00 e 21/10 das 10h10 as 12h10, períodos em que os autores deverão permanecer ao lado dos painéis. O painel deverá ser afixado no dia da apresentação, às 8h00 da manhã, devendo ser retirado após o encerramento da sessão.

Young Investigator Session
Sessões de apresentação oral com a duração de 1h15. Esta sessão será coordenada por um pesquisador sênior que apresentará o tema e dirigirá as discussões ao final das apresentações, quando será dada oportunidade de debate entre os apresentadores. Após o debate, a sessão será encerrada pelo coordenador, que fará uma breve avaliação da experiência.

Certificados:
Conferências, *simpósios e comunicações orais*: nas salas, no horário de apresentação.
Pôsteres: durante a exposição.
Cursos: nas salas, no último dia do curso, para alunos que assistiram a no mínimo duas aulas.

Media-Desk
O *Media-Desk* estará funcionando das 08h00 às 18h00.
Os congressistas deverão entregar seu material com duas horas de antecedência.
Todas as salas estarão equipadas com *data show*. Caso haja necessidade de material especial, favor informar no *Media-Desk*.
Congressistas com apresentações marcadas para 08h00 deverão entregar seu material e demais recomendações na véspera de sua apresentação.

Secretaria
A Secretaria estará aberta de 08h00 as 18h00, e contará com um painel para informações, recados e eventuais alterações de programa.

Crachás
O uso de crachá é obrigatório em todas as atividades e em áreas de circulação
No caso de perda será cobrada uma taxa para emissão da segunda via do crachá.

Resumos
Os resumos apresentados nas sessões de painéis estarão disponíveis na Home Page da Sociedade. http://www.sbfte.org.br

Useful information

Media-Desk
Media-Desk will be open from 8 am to 6 pm. Please, leave your material at Media Desk at least two hours before your presentation. All rooms have datashow. If you need any other equipment, please inform Media Desk as soon as possible.

Secretariat
Congress Secretariat will be open from 8 am to 6 pm. A board for messages, changes in the Programs, etc will be available

Badges
Please wear your badge during all venue.

Resumos
Abstracts presented at the poster session will be available at the Society site http://www.sbfte.org.br

18/10/2010 (Monday)	
09h00-12h00	Reunião do Conselho Deliberativo da SBFTE (somente para Membros do Conselho e Diretoria)/Meeting of the Deliberative Council (only for Members of the Council and Society Board)
13h30-16h30	Fórum Permanente de Pós-Graduação em Farmacologia da SBFTE (somente para Representantes dos Programas de Pós-graduação em Farmacologia, Conselho e Diretoria da SBFTE)/SBFTE Permanent Forum of Graduate Programs in Pharmacology (only for Heads of Pharmacology Graduate Programs, Council and Society Board)
14h00	Abertura da Secretaria do Congresso e da SBFTE/ Venue Secretariat and Society Secretariat Opening
19h00-19h30	Opening Session
19h30-20h30	Opening Conference
21h00	Cocktail
19/10/2010 (Tuesday)	
08h00-09h00	Courses
09h10-10h10	Conferences
10h10-10h30	Coffee-break
10h30-12h00	Symposia
12h10-14h10	Almoço e Pós-graduação / Lunch and Post-graduation
14h20-15h50	Young Investigator Presentation
15h50-16h20	Coffee-break
16h20-18h20	Symposia
18h30-20h30	Posters Session
20h30-22h30	Assembleia Geral da SBFTE / SBFTE General Assembly
20/10/2010 (Wednesday)	
08h00-09h00	Courses
09h10-10h10	Conference
10h10-10h30	Coffee-break
10h30-12h00	Symposia
12h10-13h50	Almoço e Inovação / Lunch and Innovation
14h00-16h00	José Ribeiro do Valle Award Symposium
16h00-16h20	Coffee-break
16h20-17h50	Symposia
18h00-20h00	Posters Session
21/10/2009 (Thursday)	
08h00-09h00	Courses
09h10-10h10	Conferences
10h10-12h10	Posters Session & Coffee-break
12h10-13h10	Conferences
13h20-13h40	President's Remarks
13h30	Awarding Session

18 October, 2010 (Monday)

09h00-12h00
Room Turquesa

Reunião do Conselho Deliberativo da SBFTE (somente para Membros do Conselho e Diretoria)/Meeting of the Deliberative Council (only for Members of the Council and Society Board)

13h30-16h30
Room Turquesa

Fórum Permanente de Pós-Graduação em Farmacologia da SBFTE (somente para Representantes dos Programas de Pós-graduação em Farmacologia, Conselho e Diretoria da SBFTE)/SBFTE Permanent Forum of Graduate Programs in Pharmacology (only for Heads of Pharmacology Graduate Programs, Council and Society Board)

14h00
Abertura da Secretaria do Congresso e da SBFTE/Venue Secretariat and Society Secretariat Opening

---

19h00-19h30
Room Rubi

Opening Session

19h30-20h30
Room Rubi

Opening Conference

Ligand-directed signaling bias and its potential for new therapeutics
Roger J. Summers (Monash Institute, Australia)
Presenter: Jamil Assreuy (UFSC)

21h00

Cocktail

19 October 2010 (Tuesday)

08h00-09h00
Courses

Room Ametista
Pharmacological modulation of memory during aging and neurodegenerative diseases
Chairperson: Hudson de Sousa Buck (FCMSCSP)

- *1st Class: Pharmacology of learning and memory formation*
  Hudson de Sousa Buck (FCMSCSP)

Room Topázio
Writing a scientific paper: theory and practice
Chairperson: Y. S. Bakhle (Imperial College, UK)

- *1st Class: Theory on how to write a scientific paper*
  Y. S. Bakhle (Imperial College, UK)

Room Rubi
Quantitative and qualitative analysis of drug-receptor interactions
Chairperson: André Sampaio Pupo (UNESP-Botucatu)

- *1st Class: Theoretical and practical aspects of dose-response curves plotting*
  Fernando Morgan de Aguiar Corrêa (USP)

Room Turquesa
Biology of kinase proteins: exemplification with the immune system
Chairperson: Daniel Santos Mansur (UFMG)

- *1st Class: The proteins kinase and their role in the cellular immune response*
  Aristobolo M. Silva (UFMG)

09h10-10h10
Conferences

Room Rubi
Anti-inflammatory GPCRs as targets for novel therapeutics
Mauro Perretti (Queen Mary University of London, UK)
Presenter: Sandra Helena P. Farsky (USP)

Room Topázio
Pharmacomimetics of flow-mediated endothelial vasoprotection

10h10-10h30
Coffee-break

10h30-12h00
Symposia

Room Rubi
G protein coupled receptors as drug targets: the role of G protein coupled receptor kinases (GRKs) beta-arrestins.
Chairperson: Fernando de Queiroz Cunha (USP)

- *Roles of GRKs and beta-arrestins activities in the regulation of β2 adrenergic signaling*
  Jamil Assreuy (UFSC)
- *Roles of GRKs and beta-arrestins activities in the regulation of CXCR2 chemotactic receptor signaling during severe sepsis*
  José Carlos Alves-Filho (USP)
- *Therapeutic potential of blocking PI3Kg in inflammation*
  Mauro Martins Teixeira (UFMG)

Room Ametista

Antioxidants as therapeutic agents in epilepsy, late dyskinesia, inflammation and pain models

Chairperson: Carlos Fernando de Mello (UFSM)

- *Do antioxidants cause analgesia*?
  Adair Roberto Soares dos Santos (UFSC)
- *Antioxidants as anti-inflammatory agents*
  Juliano Ferreira (UFSM)
- *Can antioxidants prevent dyskinesia*?
  Roberto Frussa-Filho (UNIFESP)

Room Topázio

Technology transfer x entrepreneurship in the institutions of science and technology

Chairperson: Claudia do Ó Pessoa (UFC)

- *Binomial of world progress: innovation and entrepreneurship*
  Cristina M. Quintella (UFBA)
- *From idea to the market - The way of innovation to the customer*
  Gerd Wassenberg (Gelsenkirchen University, Germany)
- *Biotechnology innovation in developing countries: products, interaction with universities and funding. Experience of FK-Biotecnologia S.A. in Brazil*
  Fernando Thomé Kreutz (Fk Biotecnologia)

12h10-14h10

Room Topázio

Almoço e Pós-graduação / Lunch and Graduate Programs in Pharmacology

Chairperson: Cristoforo Scavone (USP)

- Benedito H. Machado(USP)
- Jose Cipolla-Neto (ICB-USP)

14h20-15h50
Young Investigator Presentation

Room Rubi

New potential pharmacological approaches to treat central nervous system disorders

Chairperson: Francisco Silveira Guimarães (USP)

- 02.023 The selective A-type K^+ current blocker Tx3-1 rescues memory of mice submitted to a model of Alzheimer´s disease. Gomes GM[1], Dalmolin GD[2], Ferreira J[1], Gomez MV[2], Rubin MA[1] [1]UFSM − Química, [2]UFMG − Farmacologia
- 03.001 Activation of CB1 receptors reduces marble burying behavior in mice. Casarotto PC[1], Gomes FV[1], Resstel LBM[1], Guimarães FS[1] [1]FMRP-USP − Pharmacology
- 02.035 Acute but not chronic administration of pioglitazone promoted behavioral and neurochemical protective effects in the MPTP model of Parkinson's disease. Barbiero JK, Santiago RM, Lima MMS, Ariza D, Morais LH, Andreatini R, Vital MABF UFPR − Farmacologia
- 02.037 Inhibition of spinal c-Jun-N-terminal kinase (JNK) after spinal cord injury improves locomotor performance. Martini AC[1], Forner S[1], Koepp J[2], Rae GA[1] [1]UFSC − Pharmacology, [2]UFSC − Chemical and Food Engineering

Room Ametista

Clinical Pharmacology, Pharmacokinetics, Pharmacogenomics and Preclinical Toxicology

Chairperson: Teresa Cristina Tavares Dalla Costa (UFRGS)

- 11.028 Susceptible NOS3 (endothelial nitric oxide synthase) gene haplotypes in hypertension and resistant hypertension. Luizon MR[1], Sandrim VC[2], Izidoro-Toledo TC[1], Coelho EB[3], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]Santa Casa de Belo Horizonte, [3]FMRP-USP − Clínica Médica
- 11.018 Adverse reactions to chemotherapy for breast cancer and impact of genetic polymorphisms. Martins CL[1], Índio-do-Brasil V[1], Telles C[1], Vianna-Jorge R[2], Koifman S[3] [1]INCa − Farmacologia, [2]UFRJ − Farmacologia Básica e Clínica [3]ENSP-FIOCRUZ –Saúde Pública e Meio Ambiente
- 11.011 Endothelial nitric oxide synthase (ENOS) haplotypes associated with aura in women with migraine. Gonçalves FM[1], Oliveira AM[2], Speciali JG[3], Izidoro-Toledo TC[4], Silva PS[1], Dach F[3], Tanus-Santos JE[4] [1]UNICAMP − Farmacologia, [2]USP − Farmacologia, [3]FMRP − Neurologia, [4]FMRP-USP – Farmacologia
- 11.031 Interference of matrix metalloproteinase (MMP)-9 genotypes and haplotypes in the responsiveness to antihypertensive therapy of patients with preeclampsia or gestational hypertension. Palei ACT[1], Sandrim VC[2], Cavalli RC[3], Gerlach RF[4], Tanus-Santos JE[5] [1]FCM − UNICAMP − Farmacologia, [2]Santa Casa de Belo Horizonte − Farmacologia, [3]FMRP-USP − Ginecologia e Obstetrícia, [4]FORP-USP − Morfologia, [5]FMRP-USP − Farmacologia

Room Topázio

Natural Products

Chairperson: João Batista Calixto (UFSC)

- 09.008 Antinociceptive effect of uliginosin B is mediated by the activation of dopaminergic and opioid systems. Stolz ED[1], Viana AF[2], Haas JS[2], Hasse DR[2], Von Poser GL[2], Costentin J[3], Do Rego JC[3], Rates SMK[2] [1]UFRGS − Neurociências, [2]UFRGS − Farmácia, [3]Université de Rouen − Neuro-psychopharmacologie Expérimentale
- 09.017 The antinociceptive effect of triterpene 3beta, 6beta, 16beta-trihydroxylup-20(29)-ene against acute and chronic pain in mice: the involvement of glutamatergic system. Longhi-Balbinot DT[1], Lanznaster D[1], Martins DF[1], Villarinho JG[2], Ferreira J[2], Facundo VA[3], Santos ARS[1] [1]UFSC − Ciências Fisiológicas, [2]UFSM − Química, [3]UNIR – Química
- 09.022 *Lonomia obliqua* venom-induced pro-inflammatory profile in endothelial cell *in vitro*

and increased leukocyte trafficking *in vivo*. Nascimento-Silva V[1], Rodrigues GS[1], Moraes JA[1], Cyrino FZ[2], Bouskela E[2], Guimarães JA[3], Barja Fidalgo TC[1] [1]UERJ − Farmacologia, [2]UERJ − Fisiologia, [3]UFRGS – Farmacologia

- 09.031 Modulation of T lymphocyte and eosinophil functions *in vitro* by natural tetranortriterpenoids isolated from *Carapa guianensis* Aublet. Ferraris FK[1], Rodrigues R[2], Silva VP[2], Figueiredo MR[2], Penido C[1], Henriques MGMO[1] – [1]Farmanguinhos-FIOCRUZ − Farmacologia Aplicada, [2]Farmanguinhos-FIOCRUZ − Química de Produtos Naturais

15h50-16h20
Coffee-break

16h20-18h20
Symposia

Room Rubi
Brain: where inflammation and neuroscience meet
Chairperson: Mauro M. Teixeira (UFMG)

- *Chemokines and inflammation in the CNS*
  Antonio Lúcio Teixeira (UFMG)
- *Neurochemical and behavioral changes after systemic inflammation and sepsis*
  Felipe dal Pizzol (UNESC)
- *New vistas for an old friend: Lipoxin A4 as an allosteric endocannabinoid in the brain*
  Fabrício Pamplona (UFSC)
- *Stress and inflammation*
  Moises Evandro Bauer (PUCRS)

Room Ametista
Body weight: its determinants and its consequences
Chairperson: Rita de C. A. Tostes (USP)

- *The impact of stress on body weight gain*
  Ruth Harris (Medical College of Georgia, EUA)
- *High- or low-salt diet: effect on body weight, food intake and energy balance*
  Joel Claudio Heimann (USP)
- *Hypertension, kidney disease, and cardiovascular outcomes in childhood: is there a role for birth weight*
  Maria do Carmo Pinho Franco (UNIFESP)
- *Metabolic determinants of cardiovascular disease in obesity*
  David Stepp (Medical College of Georgia, EUA)

Room Topázio
Diabetes mellitus: mechanisms of vascular dysfunction, risk factor associations and potential treatment targets
Chairperson: Edson Antunes (UNICAMP)

- *Vascular bed specific remodeling in type 2 Diabetes*
  Loren Eugene Wold (Nationwide Children's Hospital, EUA)
- *Vascular reactivity of femoral arteries from diabetic trained rats*
  Angelina Zanesco (UNESP-Rio Claro)
- *Potential application of flavonoids in the therapeutics of diabetes mellitus*
  Gabriel Forato Anhê (UNICAMP)
- *Erectile dysfunction and diabetes*
  Kanchan Chitaley (University of Washington, USA)

18h30-20h30

Room Esmeralda
Poster Session 1

01. Cellular and Molecular Pharmacology
    01.001to 01.013
02. Neuropharmacology
    02.001 to 02.022
03. Psychopharmacology
    03.001 to 03.012
04. Inflammation
    04.001 to 04.046
05. Pain and Nociception
    05.001 to 05.026
06. Cardiovascular and Renal Pharmacology
    06.001 to 06.026
09. Natural Products and Toxinology
    09.001 to 09.035
11. Clinical Pharmacology, Pharmacokinetics, Pharmacogenomics and Preclinical Toxicology
    11.001-11.011

20h30-22h30

Room Topázio
Assembleia Geral da SBFTE / SBFTE General Assembly

20 October, 2010 (Wednesday)

08h00-09h00
Courses

Room Ametista
Pharmacological modulation of memory during aging and neurodegenerative diseases
Chairperson: Hudson de Sousa Buck (FCMSCSP)

- *2nd Class: Neuropharmacological changes along the aging process*
  Tânia Araújo Viel (USP)

Room Topázio
Writing a Scientific Paper: Theory and Practice
Chairperson: Y. S. Bakhle (Imperial College, UK)

- *2nd Class: Practical Session 1*
  Y. S. Bakhle (Imperial College, UK)

Room Rubi
Quantitative and qualitative analysis of drug-receptor interactions
Chairperson: André Sampaio Pupo (UNESP-Botucatu)

- *2nd Class: Analysis of competitive antagonism*
  André Sampaio Pupo (UNESP-Botucatu)

Room Turquesa
Biology of kinase proteins: exemplification with the immune system
Chairperson: Daniel Santos Mansur (UFMG)

- *2ndClass: Pi3kinase and inflammation*
  Remo de Castro Russo (UFMG)

09h10-10h10
Conferences

Room Rubi
Toll-like receptors: emerging roles in reproductive physiology and therapeutics
Mark Hedger (Monash Institute, Australia)
Presenter: Maria Christina W. de Avellar (UNIFESP)

Room Topázio
Indoleamine 2,3-dioxygenase (IDO) inhibitors: from bench to bedside.
Andrew L. Mellor (Medical College of Georgia, EUA)
Presenter: Fernando de Queiroz Cunha (USP)

10h10-10h30
Coffee-break

10h30-12h00
Symposia

Room Rubi
Cannabinoids: pharmacology and cell signaling
Chairperson: Camila Squarzoni Dale (Hospital Sírio Libanês)

- *Novel endogenous peptide agonists of cannabinoid receptors*
  Lakshmi Devi (Mount Sinai School of Medicine, USA)
- *Therapeutic potential and mechanisms of non-psychotropic phytocannabinoids*
  Francisco Silveira Guimarães (USP)
- *Hemopressin: a new target for drug development*
  Camila Squarzoni Dale (Hospital Sírio Libanês)

Room Ametista
Computational, structural and ligand-based methods in drug design
Chairperson: Maria Christina W. de Avellar (UNIFESP)

- *Inverse agonism in G-protein coupled receptors (GPCRs) seen in light of classical mechanisms of receptor activation*
  Laerte Oliveira (UNIFESP)
- *Computational chemistry underpinning carbohydrate drug discovery*
  Ivone Carvalho (USP)
- *Structure-based discovery of novel anti-inflammatory protein kinase inhibitors.*
  Carlos Alberto Manssour Fraga (UFRJ)

Room Topázio
Muscular atrophy: molecular mechanisms and signaling
Chairperson: Marcelo Damário Gomes (USP)/ Rosely Oliveira Godinho (UNIFESP)

- *Skeletal muscle atrophy in heart failure: effect of aerobic exercise training*
  Patricia Chackur Brum (USP)
- *Sympathetic actions on the skeletal muscle protein metabolism.*
  Luiz Carlos Navegantes (USP)
- *New function of the kallikrein system in the muscular atrophy*
  Lucas Tabajara Parreiras e Silva (USP)

12h10-13h50
Room Topázio
Almoço e Inovação / Lunch and Innovation
Chairperson: Regina P. Markus (USP) / Jamil Assreuy (UFSC)

- *Inovação e* start-ups *no setor biofarmacêutico no Brasil: apresentação de um case*
  Rafael Roesler (UFRGS)
- *Nos rumos da política de inovação no Brasil. Um exemplo de Santa Catarina*
  João Batista Calixto (UFSC)

14h00-16h00

Room Rubi
José Ribeiro do Valle Award Symposium
Chairperson: Jamil Assreuy (UFSC)

Larissa Staurengo Ferrari

- 05.009 IL-33 receptor deficiency reduces inflammation in septic arthritis in mice. Staurengo-Ferrari L[1], Cardoso RDR[1], Xu D[2], Liew FY[2], Cunha FQ[3], Pelayo JS[4], Saridakis HO[4], Verri Jr WA [1] [1]UEL − Ciências Patológicas, [2]University of Glasgow − Immunology Infection, Inflammation, [3]FMRP-USP, [4]UEL − Microbiologia, [6]UEL − Ciências Patológicas

Amanda Juliana Sales

- 03.015 DNA demethylating agents: new antidepressant drugs? Sales AJ[1], Biojone C[2], Gomes MVM[3], Joca SRL[1] [1]FCFRP-USP − Física e Química, [2]FMRP-USP − Farmacologia, [3]UNOPAR − Genética

Vanessa Olzon Zambelli

- 05.028 Peripheral sensitization increases opioid receptor activation and expression in both dorsal root ganglia and nerve paw of rats. Zambelli VO[1], Gutierrez VP[1], Fernandes ACO[1], Parada CA[2], Cury Y[1] [1]IBu − Dor e Sinalização, [2]UNICAMP − Farmacologia

Eduardo Moreira de Oliveira

- 02.052 Relationship of long-term memory evocation and cholinergic markers in hippocampus, along the aging process of rats. Oliveira EM[1], Souza LHJ[1], Schowe NM[1], Albuquerque MS[1], Baraldi T[1], Chambergo FS[1], Pina dos Santos VP[2], Araújo MS[2], Buck HS[3], Viel TA[1] [1]EACH-USP, [2]UNIFESP − Bioquímica, [3]FCMSCSP − Ciências Fisiológicas

Narayana Fazolini P. Bastos

- 10.014 Leptin activates the mTOR pathway in epithelial cells: roles in lipid metabolism, inflammatory mediator production and cell proliferation. Bastos NFP[1], Viola JPB[2], Maya-Monteiro CM[1], Bozza PT[1]. [1]IOC-FIOCRUZ – Imunofarmacologia, [2]INCa – Cellular Biology

16h00-16h20
Coffee-break

16h20-17h50
Symposia

Room Rubi
Calcium Signaling
Chairperson: Lusiane M. Bendhack (USP)

- *Excitation-secretion coupling in Leydig cells*
  Wamberto A. Varanda (USP)
- *Nucleoplasmic calcium regulates cell through legumain*
  Maria de Fátima Leite (UFMG)
- *Physiological and pathological aspects of brain mitochondrial Ca^{2+} transport*
  Roger Frigério Castilho (UNICAMP)

Room Ametista
Cell Signaling in Parkinson's disease: from bench to bedside
Chairperson: Rui D. S. Prediger (UFSC)

- *Dopamine turnover following administration of 7-nitroindazole to rats with L-DOPA-induced dyskinesia*
  Elaine A. Del Bel (USP)
- *Brain in movement: the role of physical exercise in Parkinson's disease.*
  Aderbal S. Aguiar-Jr. (UFSC)
- *Behavioral and neurochemical alterations induced by intranasal administration of MPTP, an experimental model of Parkinson´s disease, in mice with genetic deletion of the heparin binding growth factors Pleiotrophin and Midkine*
  Rita Raisman-Vozari (Université Pierre et Marie Curie, França)

Room Topázio
Nociceptor models for investigation of toxins from animals and plants
Chairperson: Paulo de Assis Melo (UFRJ) / Marília Zaluar P. Guimarães (UFRJ)

- *Molecular approaches to the study of natural products modulating nociception*
  Marília Zaluar P. Guimarães (UFRJ)
- *Cell and tissue responses to melittin and its antagonist*
  Camila El-Kik (UFRJ)
- *Evaluation of nociception in a model of angioneurotic edema in mice*
  Etyene Castro Dip (UFF)

18h00-20h00

Room Esmeralda
Posters Session 2

01. Cellular and Molecular Pharmacology
    01.014 to 01.027
02. Neuropharmacology
    02.023 to 02.044
03. Psychopharmacology
    03.013 to 03.024
04. Inflammation
    04.047 to 04.092
05. Pain and Nociception
    05.027 to 05.052
06. Cardiovascular and Renal Pharmacology
    06.027 to 06.052
09. Natural Products and Toxinology
    09.036 to 09.070
11. Clinical Pharmacology, Pharmacokinetics, Pharmacogenomics and Preclinical Toxicology
    11.012 to 11.022

21 October 2010 (Thursday)

08h00-09h00
Courses

Room Ametista
Pharmacological modulation of memory during aging and neurodegenerative diseases
Chairperson: Hudson de Sousa Buck (FCMSCSP)

- *3rd Class: Pharmacological modulation of memory process in neurodegenerative diseases.*
  Marielza Andrade Nunes (FCMSCSP)

Room Topázio
Writing a Scientific Paper: Theory and Practice
Chairperson: Y. S. Bakhle (Imperial College, UK)

- *3rd Class: Practical Session 2*
  Y. S. Bakhle (Imperial College, UK)

Room Rubi
Quantitative and qualitative analysis of drug-receptor interactions
Chairperson: André Sampaio Pupo (UNESP-Botucatu)

- *3rd Class: Functional analysis of dose-response curves*
  Ana Maria de Oliveira (USP)

Room Turquesa
Biology of kinase proteins: exemplification with the immune system
Chairperson: Daniel Santos Mansur (UFMG)

- *3rd Class: NFkB and IRF3/7 signaling pathway in the innate immunity*
  Daniel Santos Mansur (UFMG)

09h10-10h10
Conferences

Room Rubi
The stressed CNS: when glucocorticoids aggravate inflammation.
Javier R. Caso (Stanford University, USA)
Presenter: Carolina Demarchi Munhoz de Souza (USP)

Room Topázio
Genetic modeling of PI3K inhibition
Emilio Hirsch (University of Turin, Italy)
Presenter: Fernando de Queiroz Cunha (USP)

10h10-12h10

Room Esmeralda
Poster Session 3 & Coffee-break

01. Cellular and Molecular Pharmacology
    01.028 to 01.040
02. Neuropharmacology
    02.045 to 02.057
03. Psychopharmacology
    03.025 to 03.036
04. Inflammation
    04.093 to 04.138
05. Pain and Nociception
    05.053 to 05.079
06. Cardiovascular and Renal Pharmacology
    06.053 to 06.089
07. Endocrine and Gastrointestinal Pharmacology
    07.001 to 07.013
08. Respiratory, Urinary and Reproductive
    08.001 to 08.009
09. Natural Products and Toxinology
    09.071 to 09.107
10. Cancer and Cell Proliferation
    10.001 to 10.018
11. Clinical Pharmacology, Pharmacokinetics, Pharmacogenomics and Preclinical Toxicology
    11.023 to 11.034

12h10-13h10
Conferences

Room Topázio
Chemokines and their receptors: the nexus of neurobiology and immunobiology
Richard Ransohoff (Cleveland Clinic, USA)
Presenter: Mauro M. Teixeira (UFMG)

13h20-13h40
President's Remarks

13h30
Closing Session
Awarding Session

01. Cellular and Molecular Pharmacology

01.001
Pharmacologic evaluation of new alpha-1 adrenoceptor antagonists: structural characteristics of the derivatives N-phenylpiperazinics that affect the affinity for alpha-1 adrenoceptors. Nascimento JB[1], Romeiro LAS[2], Nascente LC[3], Lemes LFN[3], Noel F[1], Silva CLM[1] [1]UFRJ – Farmacologia Básica e Clínica, [2]FCS-UnB – Desenvolvimento de Estratégias Terapêuticas, [3]UCB-LADETER

01.002
Decreased bone resorption by low β2-adrenergic antagonist. Rodrigues WF[1], da-Silva-Filho VJ[1], Campos-Júnior JC[1], Dias da Silva VJ[2], Barbosa Neto O[2], Lopes AHP[1], Napimoga MH[3] [1]UNIUBE – Biopatologia e Biologia Molecular, [2]UMTM– Fisiologia, [3]UNIUBE – Biologia Celular e Molecular

01.003
The lidocaine analogue JMF2-1 prevents allergen-induced lung inflammation without causing immunosupression. Olsen PC[1], Ferreira TPT[1], Serra MF[1], Costa JCS[2], Cordeiro RSB[1], Silva PMR[1], Martins MA[1] [1]IOC-FIOCRUZ – Inflammation, [2]FIOCRUZ – Farmanguinhos

01.004
Pharmacologic evaluation of new alpha adreno-ceptor antagonists. Chagas-Silva F[1], Nascimento JB[1], Vieira RO[2], Romeiro LAS[3], Barberato LC[4], Noel F[5], Silva CLM[5] [1]ICB-UFRJ, [2]UFRJ – Farmacologia Celular e Molecular, [3]UCB – Química Bioorgânica e Medicinal, [4]UCB – Desenvolvimento de Estratégias Terapêuticas, [5]UFRJ – Farmacologia Básica e Clínica

01.005
Morphological study of protective effect of glutamine and alanil-glutamine injury induced by TxA from *Clostridium difficile* in rat intestinal epithelial cells. Santos AAQA[1], Leite LL[2], Brito GAC[3], Oliveira MR[4], Ribeiro RA[4], Braga Neto MB[4], Barreto LRF[4] [1]UFC – Ciências Médicas, [2]UFC – Medicina, [3]UFC – Morfologia, [4]UFC – Fisiologia e Farmacologia

01.006
Effect of annexin-1 derived peptide AC 2-26 on mice pulmonary fibroblasts. Trentin PG[1], Ferreira TPT[1], Pires ALA[1], Ciambarella BT[1], Flower RJ[2], Perretti M[2], Martins MA[1], Silva PMR[1] [1]FIOCRUZ – Inflamação, [2]William Harvey Institute – Biochemical Pharmacology

01.007
Investigation of the binding of trypanosomal FKBP12 to the ryanodine receptor-3 of rat vas deferens: possible implications in heart failure due to Chagas disease. Perissé L[1], Muzi-Filho H[2], Aido-Machado R[1], Cunha VMN[2], Salmon DJJ[1] [1]UFRJ – Bioquímica Médica, [2]ICB-UFRJ – Farmacologia Celular e Molecular

01.008
Maternal protein deprivation during lactation increases leptin secretion and inhibits apoptosis of thymic cells from young offspring. Salama Rodrigues C[1], Renovato-Martins M[2], Vargas da Silva S[1], Barja Fidalgo TC[1] [1]UERJ – Farmacologia, [2]UERJ – Farmacologia e Psicobiologia

01.009
LASSBio-1135: a multi-target antinociceptive imidazopyridinic derivative that is a TRPV1 antagonist. Silva RM[1], Guimarães MZP[1], Lima CKF[2], Lacerda RB[2], Barreiro EJ[2], Fraga CAM[2], Miranda ALP[2] [1]UFRJ – Farmacologia Básica e Clínica, [2]FF-UFRJ – Fármacos – LASSBio

01.010
Effect of a new compound, thiophenacetamide, against *Mycobacterium bovis* (BCG) infection. Vergara FMF[1], Candea ALP[1], Rosas EC[1], de Souza MVN[2], Henriques MGMO[1] [1]FarManguinhos-FIOCRUZ – Farmacologia Aplicada, [2]FarManguinhos-FIOCRUZ – Síntese Orgânica

01.011
Anti-inflammatory and antimicrobial activity of pyrazinamide analogs. Mendonça MSA[1], Candea ALP[1], Lima CHS[2], de Souza MVN[2], Henriques MGMO[2] [1]FIOCRUZ – Farmacologia Aplicada, [2]FarManguinhos-FIOCRUZ – Síntese Orgânica

01.012
O-glcnacylation contributes to the vascular effects of ET-1 via activation of the RHOA/RHO-kinase pathway. Lima VV[1], Giachini FR[1], Carneiro FS[1], Webb RC[2], Tostes RCA[1] [1]USP – Farmacologia, [2]Medical College of Georgia – Physiology

01.013
Intravascular danger signals guide neutrophils to sites of sterile inflammation. Menezes GB[1], Braedon B[2], Pittman K[2], Teixeira MM[3], Kubes P[2] [1]UFMG – Morfologia, [2]University of Calgary – Immunology, [3]UFMG – Bioquímica e Imunologia

02. Neuropharmacology

02.001
Role of iNOS in the anxiogenic effect induced by withdrawal from chronic ethanol consumption. Padovan D[1], Silva K[1], Tirapelli CR[2], Padovan CM[1] [1]FFCLRP-USP – Psicologia e Educação, [2]EERP-USP – Farmacologia

02.002
Role of P2X receptors, glia and gap junction in the modulation of glutamatergic transmission in

NTS neurons projecting to RVLM. Accorsi-Mendonça D, Bonagamba LGH, Leão RX, Machado BH FMRP-USP − Physiology

02.003
Mechanisms involved in the mediation of pressor effects of L-proline injected in the third ventricle of unanesthetized rats. Lopes-Silva S, Scopinho AA, Corrêa FMA USP – Farmacologia

02.004
Central nitric oxide synthase inhibition after 3-amino-1,2,4-triazole into the fourth cerebral ventricle influences parasympathetic response to increase in arterial pressure in spontaneously hypertensive rats. Abreu LC[1], Valenti VE[2], Ferreira C[2] [1]FMABC − Morfologia e Fisiologia, [2]UNIFESP − Cardiologia

02.005
Physical exercise reduces motor alterations associated to dopamine receptors imbalance in neurointoxicant models of Parkinson's disease. Aguiar-Jr AS[1], Boemer G[1], Rial D[1], Matheus FC[1], Moreira ELG[1], Da Cunha C[2], Prediger RD[1] [1]UFSC − Farmacologia, [2]UFPR − Farmacologia

02.006
B_1 and B_2 kinin receptors antagonists modulate the bladder overactivity induced by spinal cord injury in rats. Forner S, Andrade EL, Martini AC, Bento AF, Medeiros R, Koepp J, Calixto JB UFSC − Farmacologia

02.007
Involvement of muscarinic receptors of subtype M2 in the cardiovascular responses of acetylcholine microinjected into lateral periaqueductal gray area of unanesthetized rats. Deolindo MV, Corrêa FMA FMRP-USP Farmacologia

02.008
Intra-bed nucleus of the stria terminalis cannabidiol administration alters cardiovascular changes to acute restraint stress through 5-HT1A receptors. Gomes FV, Crestani CC, Alves FHF, Guimarães FS, Corrêa FMA, Resstel LBM FMRP-USP − Farmacologia

02.009
Effect of LASSBio-767 on apoptosis and inhibitory synaptic transmission in neurons. Vieira KST[1], Fraga CAM[2], Barreiro EJ[2], Bolzani V[3], Castro NG[1] [1]ICB-UFRJ − Farmacologia Molecular, [2]FF-UFRJ − LASSBio, [3]NUBBe-UNESP-Araraquara − Química Orgânica

02.010
High and low rearing rats selected in the open field differ in the binding of [^{3}H]RO 15-4513 to the limbic cortex. Alves R, Carvalho JGB, Venditti, MAC UNIFESP − Psicobiologia

02.011
LASSBio-579 prevents hyperlocomotion induced by ketamine A behavior suggestive of atypical antipsychotic activity. Antonio CB[1], Betti AH[1], Neves G[1], Hasse DR[2], Barreiro EJ[3], Fraga CAM[3], Rates SMK[1] [1]UFRGS − Ciências Farmacêuticas, [2]FF-UFRGS − Psicofarmacologia Experimental, [3]FF-UFRJ − LASSBio

02.012
Medial prefrontal cortex CB1 receptors are involved with modulation of the baroreflex in rats. Ferreira Junior NC, Alves FHF, Fedoce AG, Corrêa FMA, Resstel LBM FMRP-USP − Farmacologia

02.013
Putative role of Bradykinin (BK) in cognitive deficits in rats. Dong KE[1], Amaral FA[1], Lemos MTR[1], Caetano AL[1], Buck HS[1], Viel TA[2] [1]FCMSCSP − Ciências Fisiológicas, [2]EACH-USP

02.014
Inhibitory influence of lateral hypothalamus neurotransmission in the cardiac response to fear conditioning to context. Reis DG, Deolindo MV, Guimarães FS, Corrêa FMA, Resstel LBM FMRP-USP

02.015
Evaluation of the effect of a *Hypericum polyanthemum* cyclohexane extract in an animal model of Parkinson disease induced by 6-OHDA. Borsoi M[1], Betti AH[2], Batassini C[3], Silvestrin RB[4], Lazzaretti C[1], Pranke M[5], Antonio CB[2], Salles LA[5], Rosa HS[5], von Poser GL[6], Rates SMK[2], Souza TM[3] [1]ICBS-UFRGS, [2]UFRGS − Ciências Farmacêuticas, [3]UFRGS − Bioquímica, [4]ICBS-UFRGS − Neurociências, [5]UFRGS − Farmácia, [6]UFRGS − Produção de Matéria-Prima

02.016
Neuropharmacological profile of parawixin 11, purified from the venom of the social spider *Parawixia bistriata* (Araneae, Araneidae), in Wistar rats. Pereira AC[1], Cunha AOS[1], Fachim H[1], Lopes NP[2], Santos WF[1] [1]FFCLRP-USP − Biology, [2]USP − Physics and Chemistry

02.017
Involvement of serotonergic and dopaminergic neurotransmission in effect of semi-purified constituent from guaraná seeds in the elevated T maze. Roncon CM, Almeida CB, Mello JCP, Audi EA UEL − Farmácia e Farmacologia

02.018
Kinin receptors blockade ameliorates the neuro-inflammation and the clinical severity in experimental autoimmune encephalomyelitis: the dominant role of kinin B_1 receptor. Dutra RC[1], Leite DFP[1], Manjavachi MN[1], Bento AF[1], Patricio ES[1], Figueiredo CP[1], Pesquero JB[2], Calixto JB[1] [1]UFSC − Farmacologia, [2]UNIFESP − Biofísica

02.019
Dorsal hippocampus glutamate receptors modulate the expression of contextual fear conditioning. Fabri DRS, Reis DG, Hott SC, Corrêa FMA, Resstel LBM FMRP-USP − Farmacologia

02.020
Involvement of β-adrenergic receptors in the bed nucleus of the stria terminalis on the expression of contextual fear conditioning. Hott SC, Gomes FV, Reis DG, Fabri DRS, Corrêa FMA, Resstel LBM − Farmacologia

02.021
Glutamate and NMDA modulate A2 adrenergic expression in cell cultures of the medulla oblongata of newborn rats. Silva SM[1], Carrettiero DC[2], Fior-Chadi DR[1] [1]IB − Fisiologia, [2]UFABC − Ciências Naturais e Humanas

02.022
L-arginine into the CA1 hippocampal subfield did not change retention of inhibitory avoidance task in rats. Yoneyama B, Contardi EB, Milani H[1], Oliveira RMMW UEL − Farmácia e Farmacologia

03. Psychopharmacology

03.001
Activation of CB1 receptors reduces marble burying behavior in mice. Casarotto PC[1], Gomes FV[1], Resstel LBM[1], Guimarães FS[1] [1]FMRP-USP − Pharmacology

03.002
iNOS knockout mice show increased expression of contextual fear conditioning: involvement of nNOS. Lisboa SF[1], Gomes FV[1], Cunha TM[1], Cunha FQ[1], Guimarães FS[1], Corrêa FMA[1], Joca SRL[2], Resstel LBM[1] [1]FMRP-USP, [2]FCFRP-USP − Física e Química

03.003
Evaluation of the anxiolitic activity of the imidazolidinic derivative HPA-14. Carvalho FL[1], Mota VG[1], Nóbrega FFF[1], Salgado PRR[1], Fonsêca DV[1], Morais LCSL[1], Souza SA[2], Athayde-Filho PF[2] [1]UFPB − Pharmaceutical Technology, [2]UFPB − Chemistry

03.004
Effects of hippocampal iNOS or nNOS inhibition in models of anxiety and depression. Sato VAH[1], Sales AJ[2], Joca SRL[2] [1]FMRP-USP − Farmacologia, [2]FCFRP-USP − Física e Química

03.005
Intra-hippocampal injection of cannabidiol induces antidepressant-like effect in the rat forced swimming test. Biojone C[1], Silva M[1], Moreira FA[2], Guimarães FS[1], Joca SRL[3] [1]FMRP-USP – Farma-cologia, [2]ICB-UFMG − Farmacologia, [3]FCFRP-USP − Física e Química

03.006
Systemic inhibition of inducible NO synthase (iNOS) evokes antidepressant-like effects. Montezuma K[1], Biojone C[2], Guimarães FS[2], Joca SRL[1] [1]FCFRP-USP − Física e Química, [2]FMRP-USP − Farmacologia

03.007
Shock intensity and duration: importance to induce learned helplessness in rats. Donadon MF, Padovan CM FFCLRP-USP − Psicologia e Educação

03.008
Cannabidiol attenuates MCPP-induced increase in marble burying behavior. Nardo M, Casarotto PC, Guimarães FS FMRP-USP

03.009
Withdrawal from methylphenidate increases neural reactivity of dorsal midbrain. Ferreira R, Shimizu-Bassi G, Nobre MJ FFCLRP-USP − Psicologia e Educação

03.010
Truck drivers take indiscriminated use of amphetamines concomitant with toxic substances. Vieira BAC[1], Souza LA[2], Marques CD[2], Salomão PAV[2], Souza CL[3] [1]CEUNSP, [2]CEUNSP − Farmácia, [3]CEUNSP − Nutrição

03.011
Effects of anti-inflammatory and antidepressant strategies on depressive-like behavior in complete Freund's adjuvant (CFA)-treated mice. Maciel IS[1], Silva RBM[1], Calixto JB[2], Morrone FB[3], Campos MM[4] [1]PUCRS − Farmacologia, [2]UFSC − Farmacologia, [3]PUCRS − Farmácia, [4]PUCRS − Cirurgia-Odontologia

03.012
One trial tolerance phenomenon to benzodiaze-pines: contribution of dorsal hippocampus protein synthesis. Gazarini L, Stern CAJ, Bertoglio LJ UFSC − Farmacologia

04. Inflammation

04.001
Nitroxides regulate protein phosphorylation linked to NOX2 complex activity in neutrophils: prototype of a new anti-inflammatory. Ribeiro ACG[1], Chavasco LS[1], Santos GB[1], Cardoso MHM[2], Brigagão MRPL[1] [1]UNIFAL − Ciências Exatas, [2]UNIFAL − Farmácia

04.002
Periodontitis induces functional alterations in rat aorta. Campi P[1], Ceravolo GS[1], Martins Porto R[1], Maia-Dantas A[1], Yamamoto, M[1], Teixeira SA[1], Carvalho MHC[1], Herrera BS[2], Costa SKP[1], Spolidório LC[3], Muscará MN[1] [1]ICB-USP Farmacologia, [2]FO-UNESP − Patologia, [3]UNESP − Patologia

04.003
Effect of ovariectomy on LPS-induced acute lung inflammation in female mice. Gimenes-Júnior JA[1], Ligeiro de Oliveira AP[2], Vitoretti LB[1], Domingos HV[1], Oliveira-Filho RM[1], Vargaftig BB[1], Tavares de Lima W[1] [1]ICB-USP − Pharmacology, [2]ICB-USP − Immunology

04.004
Initial characterization of toll-like receptor (TLR)4 signaling pathway on pollutant-induced increased neonate mice susceptibility to asthma. Santos KT[1], Florenzano J[1], Peron JPS[2], Muscará MN[1], Rizzo LV[2], Costa SKP[1] [1]ICB-USP – Farmacologia, [2]ICB-USP – Imunologia

04.005
Platelet activating factor participate in the control of adiposity and inflammatory process in epididymal adipose tissues of mice fed with palatable diet. Menezes Z[1], Oliveira MC[1], Shang, FLT[1] Lima RL[2], Teixeira MM[2], Ferreira AVM[3], Souza DG[1] [1]UFMG – Microbiologia, [2]ICB-UFMG, [3]UFMG – Enfermagem e Nutrição

04.006
Anti-inflammatory properties of fullerol in irinotecan-induced intestinal mucositis in mice. Arifa RDN[1], Madeira MFM[1], de Paula TP[1], Ávila TV[2], Souza DG[1], Menezes Z[3], Lima RL[4] [1]UFMG – Microbiologia, [2]UFPR – Farmacologia, [3]UFMG – Fisiologia e Farmacologia, [4]ICB-UFMG

04.007
The role of 5-lipoxigenase in an experimental periodontal disease by *Aggregatibacter actinomycetemcomitans* in a murine model. Madeira MFM[1], Silva TA[2], Corrêa JD[3], Mitre GC[1], Marprates CVB[1], Souza DG[1] [1]UFMG – Microbiologia, [2]UFMG – Patologia, Clínica e Cirurgia Odontológicas, [3]UFMG – Farmacologia

04.008
Microspheres loading prostanoids as modulators of phagocytosis. Pereira PAT[1], Gelfuso GM[1], Santos DF[1], Nicolete R[2], Bitencourt CS[1], Faccioli LH[1] [1]FCFRP – Análises Clínicas, Toxicológicas e Bromatólogicas, [2]UNICEUMA

04.009
Evidence of adenosine receptors in the inosine anti-inflammatory effects in a murine model of ovalbumin-induced asthma. Lapa FR[1], Ligeiro de Oliveira AP[2], Golega BA[2], Tavares de Lima W[2], Cabrini DA[1], Santos ARS[3] [1]UFPR – Farmacologia, [2]ICB-USP – Farmacologia, [3]UFSC – Ciências Fisiológicas

04.010
Role of inflammatory chemokines and their decoy receptor D6 in modulation of the inflammatory response associated with murine GVHD. Castor MGM[1], Rezende B[2], Bernardes PTT[3], Reis AC[3], Teixeira MM[2], Locati M[4], Pinho V[2] [1]UFMG – Fisiologia e Farmacologia, [2]ICB-UFMG – Bioquímica e Imunologia / Morfologia, [3]ICB-UFMG – Morfologia, [4]Universitá di Milano

04.011
Exacerbation of dengue disease by the blockage of NADPH-oxidase complex and nitric oxide production. Avila TV[1], Costa VV[1], Fagundes CT[1], Silveira KD[2], Morcatty TQ[1], Valadão DF[1], Santos AG[1], Prosperi T[1], Souza DG[1], Silva TA[1], Teixeira MM[2] Souza DG[1] [1]UFMG – Microbiologia, [2]UFMG – Bioquímica e Imunologia

04.012
Change of skeletal muscle caffeine-induced contracture by sepsis. Pachá BP[1], Borges RS[1], Rico, T B[2], Carmo PL[1], Benjamim CF[2], Zapata-Sudo G[1], Sudo RT[1] [1]UFRJ – Fármacos, [2]UFRJ – Farmacologia Celular e Molecular

04.013
Evaluation of mechanisms of action of fatty acids from vegetables oils on wound healing. Brogliato AR[1], Figueiredo JB[2], Branco AMC[1], Almendra LR[2], Martins V[1], Monte-Alto-Costa A[3] Benjamim CF[4] [1]UFRJ – Farmacologia, [2]ICB-UFRJ, [3]UERJ – Histologia e Embriologia, [4]UFRJ – Farmacologia Básica e Clínica

04.014
Cooperative DP1- and CRTH2-activated signaling is required to elicit enhanced leukotriene C_4 synthesis induced by prostaglandin D_2 within eosinophils. Mesquita-Santos FP[1], Bakker-Abreu I[2], Luna-Gomes T[2], Bozza PT[3], Diaz BL[2], Bandeira-Melo C[2] [1]UFRJ / FIOCRUZ – Inflamação / Imunofarmacologia, [2]IBCCF-UFRJ – Inflamação, [3]FIOCRUZ – Imunofarmacologia

04.015
L-arginine up-regulated and protects the skeletal muscle tissue after resistance training for production of collagen, TGF-β and decreased TNF-α. Morais, SRL[2], Mello, WG[2], Oliveira SHP[1] – [1]UNESP-Araçatuba – Farmacologia, [2]UNESP-Araçatuba – Fisiologia

04.016
Effects of resveratrol on the pruritogenic and inflammatory events evoked by trypsin in mice. Lazarotto LF[1], Pereira PJS[2], Souto AA[3], Campos MM[4], Morrone FB[3] [1]PUCRS – Farmácia, [2]PUCRS – Medicina e Ciências da Saúde, [3]PUCRS – Biologia Celular e Molecular, [4]PUCRS – Cirurgia-Odontologia

04.017
Evaluation of selective phosphatidylinositol-3 Kinaseγ inhibitors in the inflammatory, nociceptive and pruritogenic responses induced by different agents in mice. Pereira PJS[1], Lazarotto LF[2], Leal PC[3], Calixto JB[4], Morrone FB[5], Campos MM[6] [1]PUCRS – Medicina e Ciências da Saúde, [2]PUCRS – Farmácia, [3]QMC-CFM-UFSC, [4]UFSC – Farmacologia, [5]PUCRS – Biologia Celular e Molecular, [6]PUCRS – Cirurgia-Odontologia

04.018
Effect of Annexin-1 derived peptide AC2-26 on allergic lung inflammation in mice. Matheus-Souza D[1], Trentin PG[2], Arantes ACS[2], Ferreira TPT[2], Pires ALA[2], Flower RJ[3], Perretti M[3], Martins MA[2], Silva PMR[2] [1]FIOCRUZ – Inflamação, [2]IOC-FIOCRUZ – Fisiologia e Farmacodinâmica, [3]William Harvey Institute – Biochemical Pharmacology

04.019
Effects of an anti-TNF-α therapy on *Aggregatibacter actinomycetemcomitans*-induced alveolar bone loss in mice with experimental arthritis. Queiroz Júnior CM[1], Coelho FM[2], Madeira MFM[3], Candico LCM[2], Sousa LFC[2], Teixeira MM[2], Souza DG[3], Silva TA[4] [1]UFMG − Farmacologia, [2]UFMG − Bioquímica e Imunologia, [3]UFMG − Microbiologia, [4]UFMG − Patologia, Clínica e Cirurgia Odontológicas

04.020
Pancreatic injection of phospholipases A_2 causes abdominal hyperalgesia mediated by NK-1 receptors without leading to systemic toxicity in rats. Zanoni CIS[1], Camargo E[2], Teixeira SA[1], Martins Porto R[1], Santos KT[1], Florenzano J[1], Muscará MN[1], Costa SKP[1] − [1]USP − Farmacologia, [2]UFS − Fisiologia

04.021
Amphetamine decreases inflammation and TH2-cytokines production in murine model of asthma. Hamasato EK[1], Ribeiro A[1], Ferraz-de-Paula V[1], Pinheiro ML[1], Ligeiro de Oliveira AP[2], Palermo-Neto J[1] [1]FMVZ-USP − Patologia, [2]ICB-USP − Imunologia

04.022
Intravital imaging by confocal and multiphoton microscopy: a new tool for understanding dengue pathogenesis. Santos AG[1], Costa VV[2], Menezes GB[3], Fagundes CT[2], Paula AM[4], Valadão DF[1], Morcatty TQ[2], Vilela MC[5], Pinho V[2], Teixeira MM[2] Souza DG[1] [1]UFMG − Microbiologia, [2]UFMG − Bioquímica e Imunologia, [3]UFMG − Patologia Geral, [4]UFMG − Física, [5]ICB-UFMG

04.023
Effects of formaldehyde inhalation on allergic lung inflammation: role of female sex hormones. Amemiya RM, Lino dos Santos Franco A, Ligeiro de Oliveira AP, Oliveira-Filho RM, Tavares de Lima W USP − Pharmacology

04.024
Phospholipase A2 group V in leishmaniasis: role in immunity. Zamith-Miranda D[1], Poublan LE[1], Araújo Souza PS[2], Siqueira EA[1], Viola JPB[2], Diaz BL[3] [1]UFRJ-IBCCF, [2]INCa − Biologia Celular, [3]IBCCF-UFRJ − Imunobiologia

04.025
Role of 5-Lipoxygenase products in acute respiratory distress syndrome induced by severe sepsis. Monteiro APT[1], Pinheiro CS[1], Benjamim CF[2], Soledade ES[3], Rocco PRM[4], Canetti C[1] [1]IBCCF-UFRJ, [2]UFRJ − Farmacologia Básica e Clínica, [3]UFRJ − Farmacologia, [4]UFRJ − Investigação Pulmonar

04.026
Rosiglitazone potentiates alveolar bone loss due to ligature-induced periodontitis in rats. Martins Porto R[1], Teixeira SA[1], Maia-Dantas A[1], Herrera BS[2], Campi P[2], Costa SKP[1], Nucci G[3], Spolidório LC[2], Muscará MN[1] [1]ICB-USP − Farmacologia, [2]FO-UNESP − Patologia, [3]UNICAMP

04.027
Ligature-induced periodontal disease affects salivation and saliva composition in rats. Maia-Dantas A[1], Campi P[1], Martins Porto R[1], Teixeira SA[1], Herrera BS[2], Costa SKP[1], Spolidório LC[2], Muscará MN[1] [1]USP − Farmacologia, [2]UNESP − Patologia

04.028
Reparixin, via CXCR1/CXCR2, does not reduce fever induced by PGE_2 mediators. Yamashiro LH[1], Soares DM[1], Melo MCC[2], Teixeira MM[3], Souza GEP[2] [1]USP − Farmacologia, [2]FCFRP-USP − Física e Química, [3]UFMG

04.029
Essential oil of *Mansoa standleyi* exerts anti-inflammatory effect by inhibition of macrophage activity. Santos IVF[1], Magalhães RC[1], Nascimento MVL[1], Zoghbi MGB[2], Maués LAL[1], Bastos GNT[1], Do Nascimento JLM[1] [1]UFPA − Neuroquímica Molecular e Celular, [2]Museu Emilio Goeldi

04.030
Efficacy of H_2S in the management of pruritus and oedema evoked by different mediators in the mouse skin. Rodrigues L[1], Florenzano J[1], Ekundi-Valentim E[1], Teixeira SA[1], Muscará MN[1], Costa SKP[1] [1]USP − Pharmacology

04.031
Anti-inflammatory activity of new isatin derivatives. Zardo RS[1], Figueiredo GSM[2], Silva BV[3], Matheus ME[1], Pinto AC[4], Fernandes PD[1] [1]UFRJ − Farmacologia Básica e Clínica, [2]ICB-UFRJ − Farmacologia, [3]IQ-UFRJ − Química Orgânica, [4]UFRJ − Química

04.032
Lipopolysaccharide stimulates NFkB and glucocorticoid receptor translocation to the nucleus of A7r5 rat smooth muscle cells. Scheschowitsch K, DalBó S, Assreuy J UFSC − Pharmacology

04.033
Nicotinic receptors modulate IL-12 production by dendritic cell. Pinheiro ML, Ribeiro A, Ferraz-de-Paula V, Quinteiro-Filho WM, Palermo-Neto J FMVZ-USP − Patologia

04.034
The role of suppressor of cytokine signaling 2 (SOCS-2) in an experimental pulmonary disease by pathogenic fungus *Paracoccidioides brasiliensis*. Santos PC[1], Santos DA[1], Machado FS[2], Souza DG[1] Cisalpino PS[1] [1]UFMG − Microbiologia, [2]UFMG − Bioquímica e Imunologia

04.035
Lipoxin A_4 attenuates zymosan-induced arthritis modulating endothelin-1 effects. Conte FP[1], Menezes-de-Lima Jr O[1], Verri Jr WA[2], Cunha FQ[3], Penido C[1], Henriques MGMO[1] [1]FIOCRUZ −

Farmacologia Aplicada, [2]UEL − Ciências Patológicas, [3]FMRP-USP

04.036

Anti-hypernociceptive and anti-oedematogenic properties of bis selenide in inflammatory models in mice. Jesse CR[1], Wilhelm EA[2], Bortolatto CF[2], Nogueira CW[2] [1]UNIPAMPA − Nutrição, [2]UFSM − Química

04.037

Effects of hydroquinone inhalation on functions of tracheal tissue. Shimada ALB[1], Ribeiro ALT[1], Hebeda CB[1], Bolonheis SM[1], Lino dos Santos Franco A[2], Tavares de Lima W[2], Farsky S[2] [1]USP-Análises Clínicas e Toxicológicas, [2]USP − Farmacologia

04.038

An exploratory study of H_2S-releasing enzymes in rat synovial tissue. Ekundi-Valentim E, Rodrigues L, Teixeira SA, Munhoz CD, Muscará MN, Costa SKP ICB-USP − Farmacologia

04.039

Effects of endogenous glucocorticoid and TSPO ligands on L-selectin expression in rat lymphocytes. Lima CB[1], Palermo-Neto J[2], Farsky S[1] [1]FCF-USP − Experimental Toxicology, [2]FMVZ-USP – Neuroimmunomodulation

04.040

Cellular influx and vascular permeability are modulated differentially by formaldehyde exposure in a rat model of allergic lung inflammation. Lino dos Santos Franco A, Amemiya RM, Domingos HV, Ligeiro de Oliveira AP, Breithaupt-Faloppa AC, Oliveira-Filho RM, Tavares de Lima W USP − Pharmacology

04.041

Cannabidiol, a nonpsychotropic plant-derived cannabinoid, decreases inflammation and alters leukocyte distribution in a murine model of acute lung injury. Ribeiro A[1], Ferraz-de-Paula V[1], Pinheiro ML[1], Zager A[1], Hallak JEC[2], Zuardi AW[2], Crippa JA[2], Palermo-Neto J[1] [1]FMVZ-USP − Patologia, [2]FMRP-USP − Neurologia, Psiquiatria e Psicologia Médica

04.042

FPR2/ALX agonist modulates neutrophil migration in mouse air pouch. Sordi R[1], Della Justina AM[1], Menezes de Lima Jr O[2], Fernandes D[3], Assreuy J[1] [1]UFSC − Farmacologia, [2]FIOCRUZ − Farmacologia, [3]UEPG − Ciências Farmacêuticas

04.043

The role of CXCR2 in mediating neutrophil accumulation in liver microvasculature depends on the nature of the stimulus. Barroso LC[1], Paula AM[2], Teixeira MM[1], Menezes GB[1] [1]UFMG − Bioquímica e Imunologia, [2]UFMG − Física

04.044

Participation of PI3K/AKT pathway in the pathogenesis of dengue virus infection. Valadão DF[1], Costa VV[1], Santos AG[1], Morcatty TQ[2], Fagundes CT[2], Cisalpino D[2], Silveira KD[3], Ávila TV[4], Sousa LP[5], Tavares LD[4], Teixeira MM[2], Souza DG[1] [1]UFMG − Microbiologia, [2]UFMG − Bioquímica e Imunologia, [3]UFMG − Fisiologia e Biofísica, [4]UFMG − Fisiologia e Farmacologia, [5]UFMG − Patologia Clínica

04.045

Blockade of angiotensin converting enzyme and AT_1 receptor in T cells during malaria infection: mechanisms of t-cell regulation mediated by angiotensin II. Silva-Filho JL[1], Morrot A[2], Costa MFS[3], Souza MC[3], Henriques MGMO[3], Savino W[4], Caruso-Neves C[1], Pinheiro AAS[1] [1]IBCCF-UFRJ − Ciências da Saúde, [2]FIOCRUZ − Imunologia, [3]FIOCRUZ − Tecnologia em Fármacos, [4]FIOCRUZ − Pesquisa Sobre o Timo

04.046

Immature thymocytes are released into the periphery of *Trypanosoma cruzi* acutely infected mice by a S1P-dependent mechanism. Lepletier A[1], Borja GP[2], Einicker-Lamas M[3], Silva Barbosa SD[1], Perez AR[4], Terra-Granado E[1], Carvalho CE[1], Melendes A[5], Savino W[6], Morrot A[1] [1]FIOCRUZ − Imunologia, [2]UFRJ − Imunologia e Microbiologia, [3]IBCCF-UFRJ, [4]Universidade Nacional de Rosario, [5]Glasgow University − Biomedical Research, [6]FIOCRUZ − Pesquisa Sobre o Timo

05. Pain and Nociception

05.001

The involvement of TRPA1 receptors in the induction and maintenance of prostaglandin-induced hyperalgesia. Bonet IJM[1], DallAcqua M[2], Zampronio AR[3], Tambeli CH[1], Parada CA[4], Fischer L[2] [1]FOP-UNICAMP − Ciências Fisiológicas, [2]UFPR − Fisiologia, [3]UFPR − Farmacologia, [4]UNICAMP − Farmacologia

05.002

Evaluation of the involvement of kinin receptors in the nociceptive behavior of mice submitted to the brachial plexus avulsion. Jorge IP[1], Quintão NLM[2] [1]CCS-UNIVALI, [2]UNIVALI − Ciências Farmacêuticas

05.003

Mechanisms underlying the scratching behavior induced by the activation of proteinase activated receptor-4 (PAR-4) in mice. Patricio ES, Costa R, Figueiredo CP, Motta EM, Calixto JB UFSC − Farmacologia

05.004

Inflammatory muscle hypernociception depends on activation of ERK and NF-kB signaling pathways. Lima FO[1], Verri Jr WA [2], Ribeiro dos Santos R[3], Soares MBP[3], Villarreal CF[4] [1]UEFS − Biotecnologia, [2]UEL − Ciências Patológicas, [3]CPqGM-FIOCRUZ-Bahia, [4]USP − Farmacologia

05.005
Contribution of vanilloid receptor to the nociception induced by peripheral injection of spermine in mice. Gewehr CCV[1], Silva, MA da[2], Trevisan, G[2], Rossato M[2], Drewes, CC[4], Guerra GP[2], Rubin MA[2], Ferreira J[2] [1]UFSM − Fisiologia e Farmacologia, [2]UFSM − Química, [3]USP − Toxicologia e Análises Toxicológicas

05.006
Cnidaria venom as pharmacological tool for studying the signaling pathways of pain and its control. Ferreira-Junior WA[1], Zaharenko AJ[2], Fernandes ACO[1], Zambelli VO[1], Gutierrez VP[1], Konno K[3], Tytgat[4], Picolo G[1], Cury Y[1] [1]IBu − Dor e Sinalização, [2]IB-USP − Fisiologia, [3]Universidade de Toyama − Medicina Natural, 4Universidade Católica de Leuven − Toxicologia

05.007
Reduced hyperalgesia and allodynia in neuropathic pain models by intraperitoneal and oral administration of new pirazol pirrol piridine derivative. Mendes TCF[1], Nascimento-Jr NM[2], Antunes F[3], Barreiro EJ[4], Fraga CAM[4], Sudo RT[1], Zapata-Sudo G[4] [1]UFRJ − Farmacologia e Química Medicinal, [2]IQ-UFRJ − Química, [3]CCTA-UENF, [4]UFRJ

05.008
Interaction between cyclooxygenase-2 and heme oxygenase-1 / biliverdin / carbon monoxide pathways in nociception control in mice. Grangeiro NMCG[1], Silva AAR[2], Chaves HV[2], Val DR[1], Aguiar JA[3], Souza RB[1], Albuquerque RAF[3], Bezerra MM[1] [1]FM-UFC-Sobral − Biotechnology, [2]UFC-Sobral − Dentistry, [3]FM-UFC-Sobral

05.009
IL-33 receptor deficiency reduces inflammation in septic arthritis in mice. Staurengo-Ferrari L[1], Cardoso RDR[1], Xu D[2], Liew FY[2], Cunha FQ[3], Pelayo JS[4], Saridakis HO[4], Verri Jr WA[1] [1]UEL − Ciências Patológicas, [2]University of Glasgow − Immunology Infection, Inflammation, [3]FMRP-USP, [4]UEL − Microbiologia, [6]UEL − Ciências Patológicas

05.010
Antinociception induced by LASSBio-1410 in neuropathic pain model. Leal DM[1], Nascimento-Jr NM[2], Leal, CM[2], Mendes TCF[3], Fraga CAM[4], Barreiro EJ[4], Sudo RT[5], Zapata-Sudo G[3] [1]UFRJ − Farmacologia, [2]IQ-UFRJ, [3]UFRJ − Farmacologia Básica e Clínica, [4]FF-UFRJ − LASSBio, [5]UFRJ

05.011
Analysis of the antinociceptive activity of fractions from *Pterodon polygalaeflorus*. Pinto FA, Vigliano MV, Silva GP, Freitas GM, Gayer CRM, Coelho MGP UERJ − Bioquímica

05.012
Antinociceptive activity of (-)-(2*S*,6*S*)-(6-ethyl-tetrahydropyran-2-yl)-formic acid on acute pain in mice. Marinho BG[1], Miranda, LSM[2], Meireles, BA[2], Vasconcellos, MLAA[3], Pereira, VLP[2], Fernandes PD[4] [1]UFES − Medicina Veterinária, [2]NPPN-UFRJ, [3]UFPB − Química, [4]UFRJ

05.013
(±)-trans-4-hydroxy-6-propyl-1-oxocyclohexan-2-one: a novel substance with antinociceptive properties. Marinho BG[1], Miranda, LSM[2], Costa JS[2], Delle Monache F[3], Leitão SG[4], Vasconcellos, MLAA[5], Pereira, VLP[2], Fernandes PD[6] [1]UFES − Medicina Veterinária, [2]NPPN-UFRJ, [3]UIN − Farmacologia, [4]UFRJ − Farmácia, [5]UFPB − Química, [6]ICB-UFRJ − Farmacologia

05.014
CB_1 and CB_2 cannabinoid receptors are involved in the effect of crotalphine, an opioid-like analgesic peptide. Machado FC[1], Zambelli VO[1], Fernandes ACO[1], Heimann AS[2], Cury Y[1], Picolo G[1] [1]IBu − Dor e Sinalização, [2]Proteimax Biotecnologia Ltda. − P&D

05.015
Antinociceptive and anti-inflammatory activities of novel *N*-acylhydrazone derivatives designed as piroxicam analogues. Bispo Junior W[1], Miranda AS[2], Queiroz AC[1], Cavalcante-Silva LHA[1], Matta CBB[1], Lima LM[2], Barreiro EJ[2], Alexandre-Moreira MS[1] [1]UFAL − Farmacologia e Imunidade, [2]FF-UFRJ − LASSBio

05.016
Anti-hypernociceptive effect of dichloromethane and methanolic extracts obtained from *Piper variabile* C. DC. (Piperaceae) in mice. Alves DR[1], Silva S[1], Cechinel Filho V[2], Cruz SM[3], Caceres A[3], Alvarez L[4], Quintão NLM[2] [1]UNIVALI – Ciências da Saúde, [2]NIQFAR-UNIVALI – Ciências Farmacêuticas, [3]USAC − CCQQ y Farmacia, [4]UAEM – Investigaciones Químicas

05.017
Inosine reduces pain-related behavior in mice: involvement of adenosine A_1 and A_{2A} receptor subtypes and protein kinase C pathways. Nascimento FP[1], Macedo Junior SJ[2], Lopez SMF[2], Martins DF[3], Cerutti M[1], Marcon R[1], Santos ARS[2] [1]UFSC − Farmacologia, [2]UFSC − Ciências Fisiológicas, [3]UFSC − Fisiologia

05.018
Mechanisms through which endogenous ATP via P2X3 and P2X2/3 receptors activation contributes to inflammatory nociception induced by formalin on rat´s hind paw. Krimon S[1], Parada CA[2], Oliveira MC[3] [1]UNICAMP − Fisiologia e Biofísica, [2]UNICAMP − Farmacologia, [3]UNICAMP − Ciências Fisiológicas

05.019
Nociceptive responses and thermal hyperalgesia evoked by substance P and CGRP in the rat trigeminal system. Teodoro FC, Tronco Junior MF, Cruz L, Dotto G, Zampronio AR, Chichorro JG UFPR − Farmacologia

05.020
Evaluation of potential antinociceptive the benzofuranones. Gonçalves CJ[1], Lenoir AS[1], Padaratz P[1], Cechinel Filho V[2], Niero R[3], De Campos-Buzzi F[3] [1]UNIVALI − Ciências da Saúde, [2]NIQFAR-UNIVALI − Ciências Farmacêuticas, [3]NIQFAR-UNIVALI

05.021
Effect of hemopressin on Fos and Egr-1 expression on an experimental model of neuropathic pain. Maique ET[1], Alves AS[2], Ferro ES[3], Heimann AS[4], Britto LRG[2], Dale CS[1] [1]IEP-HSL − Neuromodulação e Dor Experimental, [2]ICB-USP − Fisiologia e Biofisica, [3]ICB-USP, [4]Proteimax Biotecnologia Ltda. − P&D

05.022
Antinociceptive and anti-inflammatory effects of apocynin, an NADPH -oxidase inhibitor. Castor LRG[1], Ximenes VF[2], Hiruma-Lima CA[3] [1]UNESP-Botucatu − Farmacologia, [2]FC-UNESP-Bauru, [3]UNESP-Botucatu − Fisiologia

05.023
Antinociceptive properties of a new series of indan-hydrazine compounds. Reis RC[3], Motta NAV[1], Canal PF[1], Ávila RMD[2], Miranda ALP[3], Veloso MP[3], Brito FCF[1] [1]UFF − Fisiologia e Farmacologia, [2]UNIFAL − Ciências Farmacêuticas, [3]FF-UFRJ − LASSBio

05.024
Sensitivity of cisplatin-induced sustained mechanical hyperalgesia of face and hind paw to inhibition by classical analgesics. Guginski G, Rae GA UFSC − Farmacologia

05.025
Fractalkine expressed in dorsal root ganglion mediates inflammatory pain. Souza GR[1], Cunha TM, Lotufo CMC, Talbot J, Bozzo TA, Cunha FQ, Ferreira SH − FMRP-USP − Farmacologia

05.026
Evaluation of the analgesic effect of bupivacaine-hydroxypropyl-β-cyclodextrin inclusion complex in association to sufentanil, after intrathecal administration in rats. Queiroz VA[1], de Araújo DR[2], Cereda CMS[1], de Paula E[1] [1]UNICAMP − Bioquímica, [2]UFABC − Ciências Naturais e Humanas

06. Cardiovascular and Renal Pharmacology

06.001
Ruthenium red reverts endothelium-dependent relaxations and enhances contractions of arterial rings from pigs, rats and rabbits. Silva JDP[1], Alves Filho FC[2], Ballejo G[3] [1]NPPM-UFPI, [2]UFPI − Pharmacology and Biochemistry, [3]FMRP-USP − Pharmacology

06.002
Pharmacological and morphological evidences for the presence of TRPV4 channels in endothelial cells from rat vessels. Alves Filho FC[1], Silva JDP[2], Salgado MCO[1], Ballejo G[1] [1]FMRP-USP − Pharmacology, [2]NPPM-UFPI

06.003
Reactive oxygen species and PGF_{2alfa} receptor activation modulate SNP relaxation in denuded mice aorta. Kangussu L, Côrtes SF, Bonaventura D UFMG − Farmacologia

06.004
Chronic ethanol consumption decreases the relaxation induced by adrenomedullin and increases its expression in the isolated rat aorta. Hipólito UV[1], Tirapelli DP[2], Jacob Ferreira ALB[3], Batalhão ME[4], Tanus-Santos JE[5], Carnio EC[4], Queiroz RHC[6], Tirapelli CR[7] [1]EERP-USP − Enfermagem Psiquiátrica e Ciências Humanas, [2]FMRP-USP − Cirurgia Anatomia, [3]FCM-UNICAMP − Farmacologia, [4]EERP-USP − Enfermagem Geral e Especializada, [5]FMRP-USP − Farmacologia, [6]FCFRP-USP − Toxicologia, [7]EERP-USP − Farmacologia

06.005
Effect of quercetin on diabetic nephropathy in hypercholesterolemic mice. Gomes IBS[1], Santos, MCLFS[2], Ricardo KFS[3], Meyrelles SS[4], Vasquez EC[5] [1]UFES − Pharmaceutical Sciences, [2]UFES − Pathology, [3]FAESA, [4]UFES − Physiological Sciences, [5]UFES-EMESCAM − Physiological Sciences

06.006
Characterization of the hypertensive mechanism of ethanolic extract of *L. ericoides*. de Paula DCC[1], Souza ACM[1], Guzzo LS[1], Guimarães HN[2], Saúde-Guimarães DA[1], Grabe-Guimarães, A[1] [1]DEFAR-UFOP, [2]UFMG − Engenharia Elétrica

06.007
Vasodilatatory activity and antihypertensive profile of a new N-acylhydrazone derivative: LASSBio-1027. Leal CM[1], Kummerle AE[2], Leal DM[3], Barreiro EJ[2], Fraga CAM[2], Sudo RT[5], Zapata-Sudo G[5] [1]UFRJ − Farmacologia Básica e Clínica, [2]FF-UFRJ – LASSBio, [3]UFRJ − Farmacologia, [4]UFRJ − LASSBio, UFRJ, [5]UFRJ

06.008
Analysis of the mechanisms underlying the vasorelaxant action of THE Kaurane acid 16-metoxicauran-19-oic in the isolated rat aorta. Palazzin NB[1], Bonaventura D[2], Ambrósio SR[3], Hipólito UV[4], Tirapelli CR[5] − [1]EPCH-EERP-USP, [2]UFMG, [3]UNIFRAN − Bioprospecção e Biotransformação, [4]FMRP-USP − Farmacologia, [5]EERP-USP − Farmacologia

06.009
Effects of chronic ethanol consumption on the reactivity and adrenomedullin mRNA levels of components of this system in the rat mesenteric bed. [1]Rocha JT, [3]Hipólito UV, [2]Tirapelli DP, [2]Jacob-Ferreira AL, [1]Batalhão ME, [2]Tanus-Santos JE, [1]Carnio EC, [1]Tirapelli CR, [1]EERP-USP, [2]FMRP-USP, [3]EERP-USP / FMRP-USP

06.010

Characterization of L-arginine-NO-cGMP pathway in spontaneously hypertensive rat platelets: the effects of pregnancy. Ognibene DT, Bello PHP, Moss MB, Soares de Moura R, Brunini T, Mendes Ribeiro AC, Resende AC UERJ − Farmacologia e Psicobiologia

06.011

Effects of intermittent hypoxia on biochemical parameters of rats fed with different diets. Simões RR[1], Dutra AL[1], França RT[2], Lopes STA[2], Portela LOC[3], Zanchet EM[1] [1]UFSM − Fisiologia e Farmacologia, [2]UFSM − Clínica de Pequenos Animais, [3]UFSM − Educação Física e Desportos

06.012

Effects of intermittent hypoxia on oxidative parameters of rats fed with different diets.
Simões RR[1], Dutra AL[1], Finamor IA[1], Pavanato MA[1], Portela LOC[2], Zanchet EM[1] [1]UFSM − Fisiologia e Farmacologia, [2]UFSM − Educação Física e Desportos

06.013

Role of renin-angiotensin system and oxidative status on the maternal cardiovascular regulation in spontaneously hypertensive rats. Bello PHP, Ognibene DT, Carvalho LCRM, Costa CA, Soares de Moura R, Resende AC UERJ − Farmacologia e Psicobiologia

06.014

Molecular mechanisms involved in the dual blockade of the renin angiotensin system (RAS) on the left ventricular remodeling in renal hypertensive rats (2K-1C). Corrêa JWN[1], Callera GE[2], Yogi A.[2], He Y[2], Araújo AV[1], Vercesi JA[3], Riul ME[4], Prado CM[4], Rossi MA[4], Touyz RM[2], Bendhack LM[3] [1]FMRP-USP − Farmacologia, [2]University of Ottawa − Kidney Research, [3]FCFRP-USP − Física e Química, [4]FMRP-USP − Patologia

06.015

Renovascular hypertension alters the contribution of alternative pathway to ace in the renal vascular response in isolated kidney. Sivieri-Jr DO[1], Pereira HJV[2], Oliveira EB[2], Salgado MCO[3] [1]UFVJM − Farmácia, [2]FMRP-USP − Bioquímica e Imunologia, [3]FMRP-USP − Farmacologia

06.016

The disruption of intracellular Ca^{+2} homeostasis is associated with a change of heart function in rats chronically malnourished. Silva DB[1], Mendes LVP[1], Nascimento JHM[2], Einicker-Lamas M[2], Vieyra A[2], Cunha VMN[1], Lara Morcillo LS[1] [1]UFRJ − Farmacologia Celular e Molecular, [2]IBCCF-UFRJ

06.017

Endothelial oxidative stress induced by diabetes mellitus I increases maximum contraction evoked by angiotensin II in rat carotid artery. Pernomian L[1], Gomes MS[2], Oliveira AM[2] [1]FMRP-USP − Farmacologia, [2]FCF-USP − Física e Química

06.018

Hypercholesterolemia and aging: deleterious effects on renal function. Balarini CM[1], Gava AL[1], Pereira TMC[1], Vasquez EC[2], Meyrelles SS[1] [1]UFES − Ciências Fisiológicas, [2]EMESCAM-UFES − Ciências Fisiológicas

06.019

Impact of kidney ischemia-reperfusion on primary active Na^+ transporters and its modulation by lysophosphatidic acid. Gonsalez SR[1], Verdoorn KS[2], Beiral HJV[2], Vieyra A[2], Einicker-Lamas M[2], Lara Morcillo LS[1] [1]ICB-UFRJ − Farmacologia Celular e Molecular, [2]IBCCF-UFRJ

06.020

Antihypertensive profile of a novel *N*-acylhydrazone derivative (LASSBio-1289) in spontaneously hypertensive rats. Pereira SL[1], Oliveira LGT[1], Kummerle AE[2], Fraga CAM[2], Barreiro EJ[2], Sudo RT[1], Zapata-Sudo G[1] [1]UFRJ − Desenvolvimento de Fármacos, [2]FF-UFRJ − LASSBio

06.021

Increased circulating cell-free DNA levels in preeclampsia and gestational hypertension. Amaral LM[1], Palei ACT[2], Sandrim VC[3], Cavalli RC[4], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]FCM-UNICAMP − Farmacologia, [3]Santa Casa de Belo Horizonte, [4]FMRP-USP − Ginecologia e Obstetrícia

06.022

Alveolar bone healing process of upper incisor in spontaneously hypertensive rats (SHR) subjected to treatment with β-adrenergic antagonist. a histomorphometric and immunohistochemistry study. Cursino NM[1], Pereira CCS[2], Garcia LMG[3], Micaroni S[4], Okamoto R[2], Carvalho AAF[5], Perri SHV[6], Luvizuto EL[2], Antoniali C[4] [1]FOA-UNESP − Odontologia Infantil e Social, [2]FOA-UNESP − Cirurgia e Clínica Integrada, [3]FORP-USP − Materiais Dentários e Prótese, [4]FOA-UNESP − Ciências Básicas, [5]FOA-UNESP − Patologia e Propedêutica Clínica, [6]FOA-UNESP − Apoio, Produção e Saúde Animal

06.023

Endothelium contributes to the vascular relaxation induced by C-type natriuretic peptide (CNP) in aortas from renal hypertensive rats. Pernomian L[1], Bendhack LM[2] [1]FMRP-USP − Pharmacology, [2]FCF-USP − Physics and Chemistry

06.024

Cellular mechanisms involved in the venodilation induced by nitric oxide donors. Paulo M, Vercesi JA, Biazzotto JC, Silva RS, Bendhack LM FCF-USP − Physics and Chemistry

06.025
Vasorelaxant effects to the essential oil of *Aniba canelilla* in isolated mesenteric artery rings from spontaneously hypertensive rats. Interaminense LFL[1], Ramos-Alves FE[1], Xavier FE[1], Pinto Duarte G[1], Magalhães PJC[2], da Silva JK[6], Sousa PJC[4], Leal-Cardoso JH[5], Maia JGS[3], Lahlou S[5] [1]UFPE − Fisiologia e Farmacologia, [2]UFC − Fisiologia e Farmacologia, [3]UFPA − Engenharia Química, [4]UFPA − Farmácia, [5]UECE − Ciências Biomédicas

06.026
Maternal diabetes induces endothelial dysfunction in an age-dependent manner in resistance vessels from male offspring rats: identification of possible mechanisms involved. Ramos-Alves FE, Queiroz DB, Pinto Duarte G, Xavier FE UFPE − Fisiologia e Farmacologia

09. Natural Products and Toxinology

09.001
Leukotrienes, but not bradykinin and nitric oxide, are involved in paw edema induced by batroxase, a PI metalloproteinase isolated from *Bothrops atrox* snake venom. de Toni LGB[1], Figueiredo MJ[2], Sartim MA[1], Franco JJ[1], Cintra ACO[1], Souza GEP[3], Sampaio SV[1] [1]FCFRP-USP − Análises Clínicas, Toxicológicas e Bromatológicas, [2]FMRP-USP − Farmacologia, [3]FCFRP-USP − Física e Química

09.002
Pharmacological characterization of a metalloproteinase from *Bothrops leucurus* snake venom. Gomes MSR[1], Queiroz MR[2], Mendes MM[2], Mamede CCN[2], Vieira SAPB[2], Gimenes SNC[2], Oliveira F[2], Rodrigues VM[2] [1]UESB − Química e Exatas, [2]UFU − Genética e Bioquímica

09.003
Evaluation of the anti-hypernociceptive effect of the essential oil extracted from leaves of *Ugni myricoides* on inflammatory and neuropathic models of pain in mice. Rocha LR[1], Silva GF[1], Antonialli CS[1], Cechinel Filho V[2], Quintão NLM[1], Cicció, JF[3] [1]UNIVALI − Ciências Farmacêuticas, [2]NIQFAR-UNIVALI – Ciências Farmacêuticas, [3]Universidad de Costa Rica − Productos Naturales

09.004
Antidepressant-like effect of a supercritical carbon dioxide *Valeriana glechomifolia* extract. Müller LG[1], Salles LA[1], Betti AH[1], Stein AC[1], Sakamoto S[2], Quintas LEM[3], Bettero GM[3], Figueira R[3], Noel F[3], Von Poser GL[1], Rates SMK[1] [1]UFRGS − Ciências Farmacêuticas, [2]UFRGS − Farmácia, [3]UFRJ − Farmacologia

09.005
Effects of dietary supplementation with a multimixture composed of oat bran, flaxseed, sesame and sunflower seed on renal function of diabetic rats. Damasceno DCF[1], Almeida IP[1], Sales ALCC[2], Teixeira JMR[1], Soares LFM[1], Santos Júnior JC[1], Cunha FVM[3], Soares MA[4], Martins MCC[1] [1]UFPI − Biophysics and Physiology, [2]UFPI − Nutrition, [3]Health, Human Science and Technologies Faculty − Physiotherapy, [4]UFPI − Biochemistry and Pharmacology

09.006
Dextran sulfate protected isolated rat heart from the cardiotoxic activity of *Bothrops jararacussu* venom. Martins VV, Ricardo HD, Machado MM, Tomaz MA, El-Kik CZ, Cons BL, Melo PA UFRJ − Farmacologia Básica e Clínica

09.007
Angiotensin-converting enzyme inhibition is involved in artemetin induced hypotension in rats. de Souza P[1], Gasparotto Júnior A[1], Crestani S[1], Silva RCMVAFda[1], Stefanello MEA[2], Marques MCA[1], da Silva-Santos JE[3], Kassuya CAL[4] UFPR − Farmacologia, [2]UFPR − Química, [3]UFSC − Farmacologia, [4]UFGD − Ciências da Saúde

09.008
Antinociceptive effect of uliginosin B is mediated by the activation of dopaminergic and opioid systems. Stolz ED[1], Viana AF[2], Haas JS[2], Hasse DR[2], Von Poser GL[2], Costentin J[3], Do Rego JC[3], Rates SMK[2] [1]UFRGS − Neurociências, [2]UFRGS − Farmácia, [3]Université de Rouen − Neuro-psychopharmacologie Expérimentale

09.009
Phytochemical analysis of ethanolic extract from *Terminalia cattapa* L. leaves and its correlation with gastroprotection. Silva LP[1], Angelis CD[1], Rinaldo D[2], Vilegas W[2], Hiruma-Lima CA[3], Toma W[4] [1]UNESP-Botucatu − Fisiologia, [2]UNESP-Araraquara − Química Orgânica, [3]UNESP-Botucatu, [4]UNISANTA − Farmácia

09.010
Anti-inflammatory effects of aqueous extract *Echinodorus macrophyllus* in mice air pouch model. Silva GP, Pinto FA, Vigliano MV, Leal NRF, Marques PR, Sabino KCC, Coelho MGP UERJ − Bioquímica

09.011
Anxiolytic effect of the hydroalcoholic extract of *Lafoensia pacari* A. ST.-HIL. stem bark in mice. Galdino PM[1], Nascimento MVM[1], Sousa BF[1], de Paula JR[2], Costa EA[2] [1]UFG − Ciências Fisiológicas, [2]UFG − Farmácia

09.012
Effects of the hydroalcoholic extract of *Euterpe oleracea* Mart (açaí) on oxidative stress and endothelial dysfunction associated with 2-kidney, 1-clip hypertension. Costa CA[1], Oliveira PRB[1], Emiliano da Silva AF[1], Ognibene DT[1], Carvalho LCRM[1], Amaral TAS[1], Cordeiro VSC[1], Valença SS[2], Soares de Moura R[1], Resende AC[1] [1]UERJ − Farmacologia e Psicobiologia, [2]UFRJ − Farmacologia

09.013
Gastroprotective effects of the trichloroethane fraction of *Piper tuberculatum* in rats. Burci LM[1], Pereira IT[1], da Silva LM[1], Baggio CH[1], Facundo VA[2], Rodrigues RV[2], Santos ARS[3], Marques MCA[1], Werner MFP[1] [1]UFPR − Farmacologia, [2]UNIR − Química, [3]UFSC − Ciências Fisiológicas

09.014
Analgesic and anti-inflammatory activity of *Anadenanthera macrocarpa* brenan. Silva, KO[1], Duarte JC[1], Souza EP[1], Cruz MP[1], Marques LM[1], Andrade MF[1], Dórea RSDM[1], Meireles VS[1], Yatsuda R[1], Napimoga MH[2], Clemente-Napimoga JT[2] [1]UFBA − Saúde, [2]UNIUBE − Saúde

09.015
Substances from the leaves of *Derris urucu* inhibit alpha-glucosidase. Pereira AC[1], Arruda MSP[2], Lemos VS[3], Côrtes SF[1] [1]UFMG − Farmacologia, [2]UFPA − Química, [3]UFMG − Fisiologia e Biofísica

09.016
Effects of an extract obtained from fruits of *Euterpe oleracea* mart. (Açaí) on experimental metabolic syndrome in C57BL/6 mice. Oliveira PRB[1], Costa CA[1], Bem GF[2], Cordeiro VSC[3], Carvalho LCRM[5], Souza MAV[5], Lemos Neto M[5], Soares de Moura R[2], Resende AC[1] [1]UERJ − Farmacologia e Psicobiologia, [2]UERJ − Farmacologia, [3]UERJ

09.017
The antinociceptive effect of triterpene 3beta, 6beta, 16beta-trihydroxylup-20(29)-ene against acute and chronic pain in mice: the involvement of glutamatergic system. Longhi-Balbinot DT[1], Lanznaster D[1], Martins DF[1], Villarinho JG[2], Ferreira J[2], Facundo VA[3], Santos ARS[1] [1]UFSC − Ciências Fisiológicas, [2]UFSM − Química, [3]UNIR − Química

09.018
Antiproliferative activity of extracts from leaves of fruit trees. Begnami AF[1], Figueira GM[2], Pereira B.[2], Ruiz ALTG[2], Carvalho JE[2], Rehder VLG[2] [1]FOP-UNICAMP, [2]CPQBA-UNICAMP

09.019
Gastric antisecretory activity of an ethanolic extract of *Arctium lappa* L. in rats. da Silva LM[1], Pereira IT[1], Mendes DAGB[1], Pizzolatti MG[2], Werner MFP[3], Andre E[4], Marques MCA[1] [1]UFPR − Farmacologia, [2]UFSC − Química, [3]UFSC − Farmacologia, [4]UFRN − Biofísica e Farmacologia

09.020
Gastroprotective and antioxidant effects of ethanolic extract of *Arctium lappa* L. on acetic acid-induced ulcers in rats. da Silva LM.[1], Crestani S[1], Burci LM[1], Pizzolatti MG[2], Werner MFP[1], Andre E[3], Marques MCA[1] [1]UFPR − Farmacologia, [2]UFSC − Química, [3]UFRN − Biofísica e Farmacologia

09.021
Crotalus durissus terrificus: hepatic effects of snake venom in rats. da Silva JG[1], Soley BS[1], Gris V.[1], Rocio AAP[2], Cadena SMSC[2], Eler GJ[3], Bracht A[3], Dalsenter PR[1], Acco A[1] [1]UFPR − Farmacologia, [2]UFPR − Bioquímica e Biologia Molecular, [3]UEM − Bioquímica

09.022
Lonomia obliqua venom-induced pro-inflammatory profile in endothelial cell *in vitro* and increased leukocyte trafficking *in vivo*. Nascimento-Silva V[1], Rodrigues GS[1], Moraes JA[1], Cyrino FZ[2], Bouskela E[2], Guimarães JA[3], Barja Fidalgo TC[1] [1]UERJ − Farmacologia, [2]UERJ − Fisiologia, [3]UFRGS − Farmacologia

09.023
Gastroprotective effect of *Terminalia fagifolia* ethanolic extract. Soares GFS, Sousa OT, Souza AES, Oliveira AC, Nunes PHM, Martins MCC UFPI − Biofísica e Fisiologia

09.024
Biological activity of hidroalcoholic fraction of *Herissantia crispa* (L.) Brizicky. Dias GEN, Mota KSL[1], Lima IO[1], Pereira, FO[1], Viana WP[1], Teles YCF[1], Lima EO[1], Diniz MFFM[1], Souza MFV[1], Batista LM[1] [1] DCF-UFPB

09.025
Effect of methanolic extract and fractions from *Davilla elliptica* leaves (Dilleniaceae) on MMPs in *Bothrops jararaca* envenomation and inflammation. Nishijima CM[1], Delella FK[2], Bruni FM[3], Rodrigues CM[4], Vilegas W[4], Lopes-Ferreira M[5], Felisbino S[6], Hiruma-Lima CA[7] [1]UNESP-Botucatu − Fisiologia, [2]UNESP-Botucatu − Morfologia, [3]IBu − Toxinologia Aplicada, [4]IQ-UNESP-Araraquara − Química Orgânica, [5]IBu − Imunopatologia, [6]UNESP, [7]UNESP-Botucatu

09.026
Evaluation of anti-inflammatory activity of butanolic fraction from *Dioscorea scabra* Humb. & Bonpl. ex Willd. Hank A[1], Beduschi MG[1], Darmarco ED[2], Sousa JMB[1], Magina MDA[3], Guimarães CL[5] [1]FURB − Medicina, [2]FURB − Farmácia, [3]FURB − Ciências Farmacêuticas

09.027
Antibacterial activity of the *Byrsonima gardneriana* A. Juss. Dias GEN, Leite ATJ, Pereira FO, Rolim TL, Lima EO, Tavares JF, Batista LM DCF-UFPB

09.028
Effect of sub-chronic treatment with psychollatine in the mice light/dark paradigm. Passos CS[1], Both FL[1], Steffen VM[1], Kerber VA[2], Henriques AT[1] [1]UFRGS − Ciências Farmacêuticas, [2]UFPR − Farmácia

09.029
Mechanisms underlying the diuretic effects of isoquercitrin − an active flavonoid of *Tropaeolum majus* L. Gasparotto Júnior A[1], Gasparotto, FM[2], Leme TSV[2], Lourenço EL[1], Stefanello MEA[3], Silva

Santos, JE[4], Kassuya CAL[5], Marques MCA[6] [1]UNIPAR/UFPR − Farmacologia, [2]UNIPAR − Farmacologia, [3]UFPR − Química, [4]UFSC − Farmacologia, [5]UFGD − Farmacologia, [6]UFPR − Farmacologia

09.030
Anti-nociceptive and antiedematogenic effect of *Argyrovernonia harleyi* (H. Rob) Macleish hydroalcoholic extract on writhing test. Silva, AAR[1], Val DR[2], Souza RB[2], Araújo EB[2], Ribeiro KA[2], Brayner MMB[3], Chaves HV[4], Maia MBS[5] [1]UFC-Sobral − Odontologia, [2]UFC-Sobral, [3]UFC − Fisiologia e Farmacologia, [4]UFC, [5]UFPE − Fisiologia e Farmacologia

09.031
Modulation of T lymphocyte and eosinophil functions *in vitro* by natural tetranortriterpenoids isolated from *Carapa guianensis* Aublet. Ferraris FK[1], Rodrigues R[2], Silva VP[2], Figueiredo MR[2], Penido C[1], Henriques MGMO[1] – [1]Farmanguinhos-FIOCRUZ − Farmacologia Aplicada, [2]Farmanguinhos-FIOCRUZ − Química de Produtos Naturais

09.032
Evaluation of the antiulcer activity of the extract obtained from rhizomes of *Typha domingesis* Pers (Typhaceae). Molina L[1], Ornelas FGI[1], Toma W[1] [1]UNISANTA − Farmácia

09.033
Comparative study of different portions and extract from *Byrsonima intermedia* (leaves) A. Juss against disturbances gastrointestinal in rodents. dos Santos RC[1], Sannomiya M[2], Rodrigues CM[2], Vilegas W[2], Hiruma-Lima CA[1] [1]IB-UNESP-Botucatu − Fisiologia, [2]IQ-UNESP-Araraquara − Química Orgânica

09.034
Croton grewioides Baill. shows antidiarrhoeal activity in mice. Silva ADS[1], Silva, KM[1], Lima LO[1], Silva-Junior V[2], Silva PCB[3], Medeiros VM[3], Costa VCO[3], Tavares JF[3], Silva MS[4], Cavalcante FA[1] [1]ICBS-UFAL, [2]UFAL − Nutrição, [3]LTF-UFPB, [4]UFPB − Química

09.035
Evaluation of the toxicity and gastroprotective activity of the ethanolic extract from leaves of *Xylopia langsdorffiana* A. St.-Hil. & Tul. (Annonaceae). Montenegro CA, Lima GRM, Pessoa DR, Viana WP, Castello Branco MVS, Tavares JF, Batista LM LTF-DCF-UFPB

11. Clinical Pharmacology, Pharmacokinetics, Pharmacogenomics and Preclinical Toxicology

11.001
The proper use of self medication and risks and consequences in the health area of universities. Naime ACA, Reis LM, Garcia JAD, Soares EA, Loyola YCS UNIFENAS − Farmácia

11.002
A functional matrix metalloproteinase (MMP)-9 polymorphism modifies plasma MMP-9 levels in subjects environmentally exposed to mercury. Jacob Ferreira ALB[1], Barbosa Jr F[2], Gerlach RF[3], Tanus-Santos JE[4] [1]FCM-UNICAMP − Farmacologia, [2]FCFRP-USP − Toxicologia, [3]FORP−USP − Morfologia, [4]FMRP-USP − Farmacologia

11.003
Endothelial nitric oxide synthase gene haplotypes affect nitrite levels in black subjects. Metzger IF[1], Ishizawa MH[1], Rios-Santos F[2], Carvalho WA[3], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]UESC − Saúde, [3]HSR − Patologia Clínica e Toxicologia

11.004
Interethnic diversity of NAT2 polymorphisms in Brazilian admixed populations. Talbot J[1], Magno LA[1], Santana CVN[1], Souza SMB[1], Melo PRS[1], Corrêa RX[1], Di Pietro G[2], Rios-Santos F[2] [1]DCB-UESC, [2]DCS-UESC

11.005
Pharmacokinetic evaluation of the anticancer candidate LaSOM 65 in rats. Torres B[1], Uchoa FDT[2], Canto RFS[1], Crestani A[3], Russowsky D[4], Eifler-Lima VL[1], Dalla Costa T[1] [1]UFRGS − Ciências Farmacêuticas, [2]FF-UFRGS – Medicamentos, [3]FF-UFRGS − Síntese Orgânica Medicinal, [4]UFRGS − Química

11.006
Pharmacoepidemiological evaluation of analgesic use for children and adolescents from a public school. Alves DS[1], Lacerda JSJ[1], Matias TC[1], Borlini PG[1], Brito BG[1], Almeida JM[1], Beijamini V[2] [1]UFES − Ciências da Saúde, [2]UFES − Ciências Farmacêuticas

11.007
Imbalanced matrix metalloproteinases levels in women with polycystic ovary. Gomes VA[1], Jacob Ferreira ALB[2], Belo VA[2], Vieira, CS[3], Fernandes JBF[3], Soares GM[3], Ferriani R[3], Tanus-Santos JE[4] [1]FCM-UNICAMP, [2]FCM-UNICAMP − Farmacologia, [3]FMRP-USP − Ginecologia e Obstetrícia, [4]FMRP-USP − Farmacologia

11.008
Histological changes in different tissues of non-pregnant and pregnant rats and their fetuses treated with statins. Oliveira LP, Ikeda CM, Maciel LIS, Pereira DA, Ferreira TMI, Melo R, Braga-Vilela AS UNIFENAS − Ciências Biomédicas

11.009
Influence of isotretinoin in liver transaminases and triglycerides plasma levels. Vieira AS[1], Beijamini V[2], Melchiors, AC[1] [1]UFES − Ciências da Saúde, [2]UFES − Ciências Farmacêuticas

11.010

Matrix metalloproteinase 9 gene polymorphisms affect left ventricular hypertrophy in hypertensive patients. Lacchini R[1], Jacob Ferreira ALB[2], Luizon MR[1], Coeli FB[3], Izidoro-Toledo TC[1], Gasparini G[4], Ferreira-Sae MC[4], Schreiber R[4], Nadruz Filho W[5], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]FCM-UNICAMP − Farmacologia, [3]FMRP-USP − Endocrinologia, [4]FCM-UNICAMP − Patologia, [5]FCM-UNICAMP − Clínica Médica

11.011

Endothelial nitric oxide synthase (ENOS) haplotypes associated with aura in women with migraine. Gonçalves FM[1], Oliveira AM[2], Speciali JG[3], Izidoro-Toledo TC[4], Silva PS[1], Dach F[3], Tanus-Santos JE[4] [1]UNICAMP − Farmacologia, [2]USP − Farmacologia, [3]FMRP − Neurologia, [4]FMRP-USP − Farmacologia

01. Cellular and Molecular Pharmacology

01.014
Modulation of VEGF effects by a synthetic analogue of 15-epi-lipoxins: involvement of the enzyme heme oxygenase-1. Vieira AM[1], Barja Fidalgo TC[2], Fierro IM[2] [1]DFP-UERJ, [2]UERJ – Farmacologia

01.015
ATL-1, a synthetic analog of 15-epi-lipoxin A4, modulates foam cells activation: a novel potential tool for atherosclerosis treatment. Niconi-de-Almeida Y[1], Simões RL[1], Barja Fidalgo TC[2], Fierro IM[2] [1]UERJ − Farmacologia e Psicobiologia, [2]UERJ − Farmacologia

01.016
Characterization of the antimuscarinic effect of LASSBio-767 in HT-29 cells. Gambôa NF[1], Pimentel LSB[1], Fraga CAM[2], Barreiro EJ[2], Bolzani V[3], Castro NG[1] [1]ICB-UFRJ Farmacologia Molecular, [2]FF-UFRJ − LASSBio, [3]UFRJ – LASSBio, UFRJ, [3]NuBBe-UNESP–Araraquara – Química Orgânica

01.017
In vitro characterization of six new 1,4-benzodiazepines compounds. Thibaut JPB[1], Vieira RO[1], Menezes CMS[2], Barreiro EJ[2], Lima LM[2], Noel F[1] [1]UFRJ − Farmacologia Básica e Clínica, [2]FF-UFRJ − LASSBio

01.018
Signaling function of Na/K-ATPase in ouabain-induced a decrease in LPS inflammation model *in vivo*. Kinoshita PF[1], Yshii LM[1], Sa Lima L[1], Davel APC[2], Rossoni LV[2], Kawamoto EM[1], Scavone C[1] [1]ICB-USP − Farmacologia, [2]ICB-USP − Fisiologia e Biofísica

01.019
Protective effects of resveratrol on hepatotoxicity induced by antituberculosis drugs. Nicoletti NF[1], Santos Jr AA[2], Rodrigues-Junior VS[2], Campos MM[3], Leite CE[4], Basso LA[2], Santos DS[5], Souto AA[6] [1]INCTTB-PUCRS − Biologia Molecular e Funcional, [2]INCTB-PUCRS, [3]PUCRS, [4]PUCRS − Toxicologia, [5]PUCRS − Farmácia, [6]PUCRS − Química

01.020
The time-points of *Bothrops lanceolatus* venom molecular effects on rat gastrocnemius. Barbosa-Souza V[1], Contin DK[1], Bonventi W[2], Lôbo de Araújo A[1], Irazusta SP[1], Cruz-Höfling MA[3] [1]UNICAMP − Farmacologia, [2]CEETEPS, [3]IB-UNICAMP − Biologia Celular e Estrutural

01.021
An autocrine/paracrine role for relaxin system in testis: Sertoli cell proliferation via activation of ERK1/2 pathway and a possible role in early steps of spermatogenesis. Nascimento AR, Pimenta MT, Royer C, Lucas TFG, Porto CS, Lazari MFM UNIFESP − Farmacologia

01.022
β-adrenoceptor modulates skeletal muscle contraction by coupling to both Gs and Gi proteins: a new concept to cAMP signaling pathway. Rodrigues FSM, Bergantin LB, Pires-Oliveira M, Andrade-Lopes AL, Godinho RO UNIFESP − Farmacologia

01.023
Role of $P2X_7$ receptor during *Mycobacterium tuberculosis*-infection in mice. Santos Jr AA[1], Rodrigues-Junior VS[2], Coutinho R[3], Santos DS[2], Campos MM[4], Morrone FB[1] [1]PUCRS − Biologia Celular e Molecular, [2]INCTTb-PUCRS − Biologia Molecular e Funcional, [3]IBCCF-UFRJ, [4]PUCRS – Odontologia

01.024
Creb response after caloric restriction in a LPS inflammation model in rat hippocampus. Vasconcelos AR, Sá Lima L, Kawamoto EM, Scavone C ICB–USP − Farmacologia

01.025
Ovariectomy does not modulate chronic unpredictable stress (CUS) potentiation of lipopolysaccharide-induced NF-kB activity in striatum of female Wistar rats. Sá Lima L[1], Porto CS[2], Scavone C[1], Carolina DM[1] [1]ICB–USP – Farmacologia, [2]UNIFESP – Farmacologia

01.026
Effect of N[1]-acetyl-N-formyl-5-methoxykynuramine (AFMK) on the production nitric oxide by cultured endothelial cells. Freitas AH, Tamura EK, Markus RP IB-USP − Fisiologia

01.027
Reactivity of endothelial cells in culture is conditioned by nocturnal melatonin surge in donor rats. Marçola M, Tamura EK, Fernandes PACM, Markus RP IB-USP − Fisiologia

02. Neuropharmacology

02.023
The selective A-type K^+ current blocker Tx3-1 rescues memory of mice submitted to a model of Alzheimer´s disease. Gomes GM[1], Dalmolin GD[2], Ferreira J[1], Gomez MV[2], Rubin MA[1] [1]UFSM – Química, [2]UFMG − Farmacologia

02.024
Ketamine/fentanyl administration in infant rats induces anxiolysis until adult life. Medeiros LF[1], Souza A[1], Rozisky JR[1], Santos VS[1], Netto CA[2], Battastini AMO[2], Torres ILS[1] [1]UFRGS – Farmacologia, [2]UFRGS − Bioquímica

02.025
Kinin B2 receptor can play a neuroprotective role in Alzheimer's disease. Caetano AL[1], Amaral FA[1],

Dong KE[1], Baraldi T[2], Viel TA[2], Buck HS[1] [1]FCMSCSP – Ciências Fisiológicas, [2]EACH-USP

02.026
Medial prefrontal cortex muscarinic receptors modulate the expression of contextual fear conditioning. Fedoce AG, Ferreira Junior NC, Reis DG, Corrêa FMA, Resstel LBM FMRP-USP – Farmacologia

02.027
Morphological changes in rat skeletal muscle during atrophy caused by amyotrophic lateral sclerosis. Figueiredo LB, Barnabe GF, Mello LE, Godinho RO UNIFESP – Farmacologia, [2]UNIFESP – Fisiologia

02.028
"Anxious" and "non-anxious" subgroups of rats selected in the elevated plus maze do not differ in the density of [^{3}H]-flunitrazepam binding in the hippocampus and limbic cortex. Carvalho JGB, Venditti MAC UNIFESP – Psicobiologia

02.029
Binding of [^{3}H]-flunitrazepam and [^{3}H]-MK-801 in brain regions of rats with different sensitivity to the convulsant effect of a benzodiazepine inverse agonist. Conto MB[1], Carvalho JGB, Venditti MAC UNIFESP – Psicobiologia

02.030
The paraventricular nucleus of the hypothalamus mediates pressor response to acute restraint stress in rats. Busnardo C[1], Tavares RF[1], Resstel LBM[1], Elias LLK[2], Corrêa FMA[1] [1]FMRP-USP – Pharmacology, [2]FMRP-USP – Physiology

02.031
Pre-synaptic nicotinic cholinergic receptor increases neurotransmitter release in cultured cells from the medulla oblongata. Matsumoto JPP, Martins EAC, Fior-Chadi DR IB-USP – Physiology

02.032
Facilitation of endocannabinoid-mediated neurotransmission in the dorsal hippocampus induces anxiolytic effects in rats submitted to the Vogel conflict test. Nejo P, Lisboa SF, Resstel LBM, Guimarães FS FMRP-USP

02.033
Possible role of the angiotensin (1-7) in the hippocampus in a model of epilepsy. Pereira MGAG[1], Souza LL[1], Becari C[2], Camacho F[1], Oliveira JAC[3], Salgado MCO[2], Garcia-Cairasco N[3], Costa-Neto CM[1] [1]FMRP-USP – Biochemistry and Immunology, [2]FMRP-USP – Pharmacology, [3]FMRP-USP – Physiology

02.034
Enriched environment stimulus improves spatial and aversive-related memory performance in an animal model of severe Alzheimer's disease. Schowe NM[1], Oliveira EM[1], Souza LHJ[1], Sousa AMA[2], Amaral FA[2], Lopes ASA[2], Caetano AL[2], Rocha MN[3], Buck HS[2], Viel TA[1] [1]EACH-USP, [2]FCMSCSP – Ciências Fisiológicas, [3]FCMSCSP – Medicina Molecular

02.035
Acute but not chronic administration of pioglitazone promoted behavioral and neurochemical protective effects in the MPTP model of Parkinson's disease. Barbiero JK, Santiago RM, Lima MMS, Ariza D, Morais LH, Andreatini R, Vital MABF UFPR – Farmacologia

02.036
The noradrenergic neurotransmission in the MeA modulates the cardiovascular responses to acute restraint stress in rats. Fortaleza EAT, Scopinho AA, Corrêa FMA FMRP-USP

02.037
Inhibition of spinal c-Jun-N-terminal kinase (JNK) after spinal cord injury improves locomotor performance. Martini AC[1], Forner S[1], Koepp J[2], Rae GA[1] [1]UFSC – Pharmacology, [2]UFSC – Chemical and Food Engineering

02.038
Medial prefrontal cortex NMDA-Nitric oxide pathway modulates anxiety-behavior in rats submitted to the Vogel conflict test. Resstel LBM, Lisboa SF, Guimarães FS FMRP-USP

02.039
Aged and young rats respond differently to permanent, 3-stage 4-vessel occlusion: An analysis of learning, neurodegeneration and β-APP expression. Ferreira EDF[1], Romanini CV[2], Albertin M[1], Mori MA[3], Oliveira RMW[1], Milani H[1] [1]UEM – Farmácia e Farmacologia, [2]UEL – Farmácia e Farmacologia, [3]UEM – Ciências Biológicas

02.040
Alpha2-adrenoceptors in the lateral septal area modulates cardiovascular responses evoked by restraint stress in rats. Scopinho AA, Reis DG, Resstel LBM, Corrêa FMA FMRP-USP

02.041
Involvement of glutamate AMPA receptors in the hypothalamic mechanisms triggered by paracetamol on the supression of LPS-induced fever. Campos EMB[1], Moraes TP[1], Kanashiro A[2], Malvar DC[3], Souza GEP[2] Iyomasa MM[1], Rosa ML[1] [1]FAMECA-FIPA – Neurociências, [2]FCF-USP – Física e Química, [3]UFRRJ – Ciências Fisiológicas

02.042
Comparison of cognitive stimulation during life-time and during the elderly: effects on spatial memory and on neuroplasticity of mice. Baraldi T[1], Amaral FA[2], Caetano AL[2], Albuquerque MS[1], Buck HS[1], Viel TA[1] [1]EACH-USP, [2]FCMSCSP – Ciências Fisiológicas

02.043
Characterization of glycinamide as a co-agonist of NMDA receptors. Montenegro VM[1], Setti-

Perdigão P, Guimarães MZP, Castro NG ICB-UFRJ − Farmacologia Molecular

02.044
The expression of mRNA encoding flip and flop isoforms of GLUR1 is increased in hippocampus of isolated young adult rats. Pereira MTR[1], Tonso VM[2], Limonte FH[2], Oliveira FS[3], Iyomasa MM[1], Rosa ML[1] [1]FAMECA-FIPA − Neurociências, [2]FAMECA-FIPA − Bioquímica, [3]USP – Farmacologia

03. Psychopharmacology

03.013
Effect of transient reversible inactivation of the ventral hippocampus in rats submitted to the forced swimming test. Diniz CRAF, Casarotto PC, Joca SRL FCFRP-USP − Física e Química

03.014
Involvement of local insular cortex neurotransmission on contextual fear conditioning. Alves FHF[1], Reis DG[2], Crestani CC[2], Corrêa FMA[3], Resstel LBM[3] [1]FMRP-USP – Farmacologia

03.015
DNA demethylating agents: new antidepressant drugs? Sales AJ[1], Biojone C[2], Gomes MVM[3], Joca SRL[1] [1]FCFRP-USP − Física e Química, [2]FMRP-USP − Farmacologia, [3]UNOPAR − Genética

03.016
New *N*-phenylpiperazine derivatives with antipsychotic-like activity in rodents bind to α_{1a} and α_{1b} receptors. Betti AH[1], Antonio CB[1], HASSE DR[2], Vieira RO[3], Martins TS[4], Barreiro EJ[4], Fraga CAM[4], Noel F[5], Rates SMK[1] [1]UFRGS – Ciências Farmacêuticas, [2]UFRGS – Psicofarmacologia Experimental, [3]UFRJ – Farmacologia Celular e Molecular, [4]FF-UFRJ – LASSBio, [5]UFRJ – Farmacologia Básica e Clínica

03.017
Acute MDMA (Ecstasy) treatment induces a persistent leukocyte distribution change and enhances susceptibility to infection. Ferraz-de-Paula V[1], Ribeiro A[1], Souza-Queiroz J[2], Torello CO[3], Queiroz MLS[4], Moreau RLM[5], Palermo-Neto J[6] [1]FMVZ-USP − Patologia, [2]IP-USP, [3]UNICAMP − Farmacologia, [4]UNICAMP – Farmacologia / Hemocentro, [5]FCF-USP − Análises Clínicas e Toxicológicas, [6]FMZV-USP – Neuroimuno-modulation

03.018
Evaluation of the antinociceptive effect of ethanolic extract of *Sida galheirensis* (Malvaceae). Salgado PRR, Fonsêca DV, Carvalho FL, Torres PA, Lima MRV, Morais LCSL, Almeida RN, Souza MFV UFPB − Pharmaceutical Technology

03.019
Evaluation of central activity of essential oil *Lippia microphylla* (Verbenaceae) in mice. Monte LO, Mota VG, Pinheiro LS, Timôteo RNPD, Tavares JF, Morais LCSL, Alencar JL, Almeida RN UFPB − Pharmaceutical Technology

03.020
Restraint stress changes temporal patterns of adenine nucleotides hydrolysis in rat's blood serum. Souza A[1], Detanico BC[1], Rozisky JR[1], Medeiros LF[2], Caumo W[2], Hidalgo MP[3], Battastini AMO[4], Torres ILS[2] [1]UFRGS − Farmacologia, [2]UFRGS − Anestesia, [3]UFRGS − Psiquiatria, [4]UFRGS − Bioquímica

03.021
Behavioral syndromes in experimental autoimmune encephalomyelitis. Rodrigues DH[1], Sousa LFC[1], Miranda AS[1], Lacerda-Queiroz N[1], Vilela MC[1], Campos RDL, Teixeira MM[1], Reis HJ[2], Teixeira AL[1] [1]UFMG − Imunofarmacologia, [2]UFMG − Neurofarmacologia

03.022
Chronic administration of medroxyprogesterone or clomifene, anti-estrogenic drugs, does not reproduce the antimanic-like effect of tamoxifen in an animal model of mania. Pereira M[1], Siba IP[1], Martynhak BJ[1], Correia D[1], Baretta IP[2], Andreatini R[1] [1]UFPR − Pharmacology, [2]UNIPar

03.023
Anxiolytic-like effect of benzodiazepine flurazepam is oestrous cycle-dependent. Silva Medeiros AG, Muniz GD, Figueiredo Neto JL, Arruda Junior WB, Costa RD, Carvalho MS, de Paula Soares V UFRN −. Biofísica e Farmacologia

03.024
Role of median raphe nucleus 5-HT1a receptors on behavioral despair. Trovo MC, Almeida PVG, Pereira DHS, Padovan CM FFCLRP-USP − Psicologia e Educação

04. Inflammation

04.047
Effect of repeated treatment with enalapril on the hepatotoxicity induced by acetaminophen in mice. Betto MRB[1], Lazarotto LF[2], Leite CE[3], Watanabe TTN[4], Driemeier D[5], Campos MM[6] [1]PUCRS − Biologia Celular e Molecular, [2]PUCRS – Farmácia, [3]PUCRS − Toxicologia, [4]UFRGS − Patologia e Clínica Veterinária, [5]UFRGS − Veterinária, [6]PUCRS − Cirurgia-Odontologia

04.048
A pharmacological approach to food allergy in mice: novel therapeutic targets. Pereira-Silva PEM, Amaral SS, Noviello MLM, Menezes GB, Cara DC ICB-UFMG − Morfologia

04.049
Quercetin inhibits neutrophil recruitment *in vivo* and *in vitro*: inhibition of actin polymerization. Zarpelon AC[1], Souto FO[2], Staurengo-Ferrari L[1],

Fattori V[1], Casagrande R[1], Fonseca MJ[3], Cunha TM[2], Ferreira SH[2], Cunha FQ[2], Verri Jr WA[1] [1]UEL − Ciências Patológicas, [2]FMRP-USP − Farmacologia, [3]FCFRP-USP − Ciências Farmacêuticas

04.050

Effects of mangiferin on allergic inflammation induced by ovalbumin in A/J mice. Coelho LP, Jurgilas PB, Serra MF, Pires ALA, Cruz CCD, Cordeiro RSB, Silva PMR, Martins MA IOC-FIOCRUZ − Fisiologia e Farmacodinâmica

04.051

Inhibition heme oxygenase increases neutrophil migration to the bronchoalveolar spaces and attenuates pulmonary mechanics changes during severe sepsis induced by pneumonia. Czaikoski PG[1], Nascimento DCB[2], Spiller F[1], Rocco PRM[3], Cunha FQ[1] [1]FMRP-USP − Pharmacology, [2]FMRP-USP − Immunology, [3]UFRJ Investigação Pulmonar

04.052

Effect of thoracic lymphatic duct ligation on the release of lung inflammatory mediators in the model of gut trauma in rats. Breithaupt-Faloppa AC, Vitoretti LB, de Assis Ramos MM, Cavriani G, Sudo-Hayashi LS, Oliveira-Filho RM, Vargaftig BB, Tavares de Lima W ICB-USP − Farmacologia

04.053

Eosinophils as novel cell source of prostaglandin D_2: autocrine activity and allergy-driven synthesis. Luna-Gomes T[1], Magalhães KG[2], Mesquita-Santos FP[2], Bakker-Abreu I[1], Samico RF[1], Bozza PT[2], Diaz BL[1], Bandeira-Melo C[1] [1]IBCCF-UFRJ, [2]IOC-FIOCRUZ

04.054

Kinetics of tissue response to orthodontic forces in mice: mechanical stimulation leads to bone remodeling through differential expression of osteoclast and osteoblast related factors. Garlet TP[1], Tadei SR[2], Silva TA[3], Garlet GP[4], Cunha FQ[1] [1]FMRP-USP, [2]ICB-UFMG, [3]UFMG − Patologia, [4]FOB-USP

04.055

Signaling transduction pathway involved in LPS-induced suppression of melatonin production by rat pineal gland. Cruz-Machado SS[1], Pinato, L[2], Carvalho-Sousa CE[1], Tamura EK[1], Ferreira ZS[1], Markus RP[1] [1]IB-USP − Fisiologia, [2]UNESP − Fonoaudiologia

04.056

Enhanced airway smooth muscle reactivity to cholinergic provocation is associated to mast cells in A/J mice. Anjos-Valotta EA, Farias-Filho FA, Serra MF, Cordeiro RSB, Silva PMR, Martins MA FIOCRUZ − Inflamação

04.057

Prior exposure to staphylococcal enterotoxin type B (SEB) potentiates the pulmonary eosinophil infiltration of allergic mice. Squebola Cola DM[1], Mello GC[1], Schenka A[2], Souza IA[1], Antunes E[1] [1]FCM-UNICAMP − Farmacologia, [2]FCM-UNICAMP − Patologia

04.058

Role of mast cells on the production of CINC-2, migration of neutrophils and bone resorption in SHR animals submitted to periodontal disease. Belini L[1], Salzedas LMP[2], Oliveira SHP[1] [1]FO-UNESP-Araçatuba − Ciências Básicas, [2]UNESP-Araçatuba − Radiologia

04.059

Modulation of FCgR-mediated phagocytosis in macrophages by TLRS agonists: involvement of 5-LO products. Pinheiro CS[1], Monteiro APT[2], Benjamim CF[3], Canetti C[1] [1]IBCCF-UFRJ, [2]UERJ − Farmacologia e Psicobiologia, [3]UFRJ − Farmacologia Básica e Clínica

04.060

Odontoblasts stimulated by lipopolysaccharide express SCF and FGF-2 via p42/44, p38 and PI3K. Santos VAC[1], Oliveira SHP[2] [1]FOA-UNESP − Odontologia Social e Preventiva e Ciências Básicas, [2]FOA-UNESP − Ciências Básicas

04.061

Role of *GILZ (glucocorticoid-induced leucine zipper*) on resolution of inflammation. Nogueira CRC[1], Tavares LP[1], Silva JPV[1], Queiroz ALL[1], Silva DM[1], Soriani FM[1], Russo RC[1], Garcia CC[1], Lopes F[2], Pinho V[1], Teixeira MM[1], Sousa LP[3] [1]UFMG − Bioquímica e Imunologia, [2]UFMG − Morfologia, [3]UFMG − Patologia Clínica − COLTEC

04.062

Evaluation of the anti-inflammatory effect of mycofenolate mofetil in mice LPS-induced pleurisy. Beduschi MG[1], Darmarco ED[3], Frode TS[2], Guimarães CL[4] [1]FURB − Medicina, [2]UFSC − Análises Clínicas, [3]FURB − Farmácia, [4]FURB − Ciências Farmacêuticas

04.063

CC chemokine receptors play different roles in the pathogenesis of dengue virus infection in mice. Guabiraba R[1], Pereira-Silva REM[1], Besnard AG[2], Souza DG[3], Ryffel B[2], Teixeira MM[1] [1]UFMG − Bioquímica e Imunologia, [2]CNRS-IEM, [3]UFMG − Microbiologia

04.064

Inhibition of guanylyl cyclase restores neutrophil migration and maintains bactericidal activity increasing survival in sepsis. Amêndola R[1], Neto H[2], Souto FO[3], Alves-Filho JC[3], Spiller F[3], Freitas A[3], Cunha FQ[3], Barja Fidalgo TC[2] [1]UERJ − Farmacologia e Psicobiologia, [2]UERJ − Farmacologia, [3]USP − Farmacologia e Dor

04.065

Anti-inflammatory effects of lovastatin on the tests of formalin and dextran-induced paw edema. Siqueira RMP[1], Gonçalves DO[1], Calou

IBF[2], Olinda TM[1], Figueiredo IST[1], Pinheiro CN[4], Melo TS[1], Cavalcante, ALC[5], Viana GSB[1] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Análises Clínicas, [4]FATECI − Biomedicina, [5]UFC − Ciências Médicas

04.066

Effect of the crude extract and the aerial parts fractions of *Sesbania virgata* on the inflammatory response in animals. Arruda LLM[1], Bonfim, N. M.[2], Kummer R[1], Souza, M. C.[3], Sarragioto MH[2], Baroni S[1], Grespan R[1], Bersani-Amado CA[1] [1]UEM − Farmácia e Farmacologia, [2]UEM − Química, [3]UEM − Biologia

04.067

Effects of resveratrol in the acute and chronic models of inflammation in rats. Silva RBM[1], Maciel IS[2], Souto AA[3], Morrone FB[4], Campos MM[5] [1]PUCRS − Farmacologia Aplicada, [2]PUCRS − Farmacologia, [3]PUCRS − Química, [4]PUCRS − Farmácia, [5]PUCRS − Cirurgia-Odontologia

04.068

Inflammatory response induced by carvacrol, a *Thymus vulgaris* essential oil constituent. Fachini FC, Kummer R, Ritter AMV, Anteguera AAC, Domiciano TP, Bersani-Amado CA, Cuman RKN UEM − Farmácia e Farmacologia

04.069

LTB4 as chemoattractant factor in the regulatory T cells migration. Pecli CP[1], Molinaro RC[2], Peters-Golden M[3], Kunkel SL[4], Canetti C[5], Benjamim CF[1] [1]ICB-UERJ, [2]IOC-FIOCRUZ, [3]University of Michigan − Pulmonary and Critical Care Medicine, [4]University of Michigan – Pathology [5]IBCCF-UFRJ

04.070

Ntipyretic effect of dipyrone is not related with the hypothalamic PGE2 synthesis inhibition in rats. Malvar DC, Figueiredo MM, Martins JM, Pessini AC, Soares DM, Souza GEP FCFRP-USP − Física e Química

04.071

The role of acid-induced laminin polymer in splenic dendritic cells. Ladislau L[1], Da-Fe AR[2], Coelho-Sampaio TL[3], Kunkel SL[4], Benjamim CF[5] [1]ICB-UFRJ, [2]UERJ − Farmacologia e Psicobiologia, [3]UFRJ − Histologia, [4]University of Michigan − Pathology, [5]UFRJ − Farmacologia Básica e Clínica

04.072

Clinical evaluation of the anti-inflammatory effect of *Baccharis dracunculifolia* propolis gel (patent PI 0904121-4) on cervicitis. Paulino N[1], Scremin Paulino A[2], Marcucci MC[1], Vautier P[1] 1UNIBAN − Farmácia, [2]UFSC − Farmácia

04.073

Activation of TLR9 in circulating neutrophils inhibits their migration to inflammatory site. Trevelin SC[1], Alves-Filho JC[1], Sônego F[1], Souto FO[2], Nascimento DCB[2], Turato W[2], Cunha TM[1], Gazzinelli RT[2], Cunha FQ[1] [1]FMRP-USP − Pharmacology, [2]FMRP-USP, Immunology and Biochemistry

04.074

Effects of eotaxin in the peritoneal migration of eosinophils and neutrophils, dependent on 5-lipoxygenase products. Lages PM1, Arcanjo LCG[2], Lopes RS[1], Silva CLCA[1], Luz RA[3], Elsas PPX[4], Elsas MICG[5] [1]IMPPG-UFRJ, [2]IFF-FIOCRUZ, [3]IFF-FIOCRUZ − Pediatria, [4]UFRJ, [5]FIOCRUZ

04.075

Adenosine and adenosine-monophosphate present into the *Phlebotomus papatasi* saliva block dendritic cell function and ammeliorate collagen-induced arthritis. Carregaro V[1], Sá-Nunes, A[2], Cunha, TM[3], Grespan R[4], Oliveira CJ[1], Lima-Jr DS[5], Costa DL[1], Milanezi CM[1], Verri Jr WA[5], Valenzuela JG[6], Silva JS[1], Ribeiro JM[6], Cunha FQ[3] [1]FMRP-USP − Biochemistry and Immunology, [2]ICB-USP − Immunology, [3]FMRP-USP − Pharmacology, [4]UEM − Farmácia e Farmacologia, [5]UEL − Pathology and Pharmacology, [6]NIAID/NIH − Vector Biology

04.076

Anti-inflammatory activity of crude extract and of flowers fractions from *Palicourea rigida* in mice. Arruda LLM[1], Rosa EA[2], Oliveira CMA[3], Fachini RF[2], Silva CC[2], Baroni S[1], Grespan R[1], Bersani-Amado CA[1] [1]UEM − Farmácia e Farmacologia, [2]UEM Química, [3]UFGO − Química

04.077

Short chain-fatty acid effects on acute gout: importance in induction and resolution of inflammatory responses. Vieira AT[1], Shim D[2], De-Leon E[2], Schilter HC[2], Amaral FA[3], Arruda MCC[3], Maslowski KM[2], Fagundes CT[3], Nicoli JR[4], Teixeira MM[3], Mackay CR[2] [1]Garvan Institute of Medical Research/UFMG − Arthritis and inflammation / Bioquímica e Imunologia, [2]Garvan Institute of Medical Research − Arthritis and Inflammation, [3]UFMG − Bioquímica e Imunologia, [4]UFMG − Microbiologia

04.078

In vivo hydroquinone exposure affects leukocyte recruitment and adhesion molecules expression on LPS inflamed lung. Ribeiro ALT[1], Shimada ALB[1], Hebeda CB[1], Bolonheis SM[1], Tavares de Lima W[2], Farsky S[1] [1]USP-Clínical and Toxicological Analyses, [2]ICB-USP – Pharmacology

04.079

Exogenous leptin modulates acute lung inflammation induced by LPS in mice. Landgraf MA[1], Silva RC[2], Hiyane M[3], Cunha CS[3], Vieira PMM[3], Cenedeze MA[2], Keller AC[4], Pacheco-Silva A[4], Araújo RC[6], Câmara NOS[1], Landgraf RG[4] [1]ICB-USP, [2]UNIFESP − Nefrologia, [3]ICB-USP − Imunologia, [4]UNIFESP-Diadema − Ciências Biológicas, [6]UNIFESP − Biofísica

04.080
Gastroprotection and healing activities of the seeds oil from *Carapa guianensis* Aubl. in mice. Gonçalves DO[1], Figueiredo IST[1], Osório CBH[1], Siqueira RMP[1], Oliveira RSB[2], Freitas LBN[1], Vieira IR[1], Vasconcelos MAM[3], Alencar NMN[1] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Bioquímica e Biologia Molecular, [3]EMBRAPA − Agroindústria

04.081
ADP effect on skin wound healing on diabetic mice. Branco AMC[1], Brogliato AR[1], Figueiredo JB[2], Melo PA[3], Benjamim CF[3] [1]UFRJ − Farmacologia, [2]ICB-UFRJ, [3]UFRJ − Farmacologia Básica e Clínica

04.082
Inflammasome activation and IL-1b and IL-18 production are essential for host resistance to dengue virus primary infection. Fagundes CT[1], Ávila TV[2], Costa VV[2], Silveira KD[3], Cisalpino D[2], Valadão DF[2], Tavares LD[2], Morcatty TQ[2], Santos AG[2], Souza RS[2], Vieira LQ[3], Zamboni DS[4], Souza DG[2] Teixeira MM[3] [1]UFMG − Bioquímica e Imunologia/Microbiologia, [2]UFMG – Microbiologia, [3]UFMG − Bioquímica e Imunologia, [4]FMRP-USP − Biologia Celular, Molecular e Bioagentes Patogênicos

04.083
Membrane TNF-α is essential for the pathogeneses of gouty arthritis. Tavares LD[1], Amaral FA[1], Costa VV[1], Fagundes CT[2], Quesniaux V[3], Ryffel B[4], Teixeira MM[2], Souza DG[1] [1]UFMG − Microbiologia, [2]UFMG − Bioquímica e Imunologia, [3]CNRS − Molecular Immunology and Embryology, [4]IEM-CNRS

04.084
ATL-1, a synthetic analog of 15-Epi-lipoxin A4, promotes changes in dendritic cells phenotype and function. Da-Fe AR[1], Ladislau L[2], Kunkel SL[3], Benjamim CF[4], Fierro IM[5] [1]UERJ − Farmacologia e Psicobiologia, [2]ICB-UFRJ, [3]University of Michigan − Pathology, [4]UFRJ − Farmacologia Básica e Clínica, [5]UERJ − Farmacologia

04.085
Platelet-Activating Factor (PAF) contributes to the neuroinflammatory process involved in the *Plasmodium berghei* ANKA infection. Lacerda-Queiroz N[1], Rodrigues DH[1], Miranda AS[2], Vilela MC[3], Teixeira MM[4], Teixeira AL[5] [1]UFMG − Biologia Celular, [2]UFMG − Medicina Tropical, [3]UFMG − Neurociências, [4]UFMG, [5]UFMG − Medicina

04.086
iNOS-derived nitric oxide modulates bone loss from ligature induced periodontitis by inhibiting osteoclast differentiation and activity. Herrera BS[1], Martins Porto R[2], Costa SKP[2], Spolidório LC[1], Van Dyke TE[3], Gyurko R[3], Muscará MN[2] [1]FOAR-UNESP − Physiology and Pathology, [2]ICB-USP − Pharmacology, [3]Boston University − Periodontology and Oral Biology

04.087
HQ impairs nitric oxide synthesis in neutrophils via post transcriptional modifications. Hebeda CB[1], Bolonheis SM[1], Pinedo F[1], Teixeira SA[2], Muscará MN[2], Farsky S[1] [1]USP − Análises Clínicas e Toxicológicas, [2]USP − Farmacologia

04.088
Melatonin inhibits adhesion of neutrophils induced by lipopolysaccharide (LPS) in endothelial cells culture. Abrantes-Lima KD, Tamura EK, Markus RP IB-USP − Fisiologia

04.089
Interaction of the anti-inflammatory annexin A1 protein and tacrolimus immunosuppressant in the renal function of rats. Truzzi RR[1], Araújo LP[2], Oliani SM[1] [1]UNESP − Biology, [2]UNIFESP − Morphology

04.090
Crosstalk of TLR2/CD36 with PPARg in lipid metabolism and inflammatory response during infection by *Mycobacterium bovis* BCG: role of rafts. Almeida PE[1], Antunes KM[2], Maya-Monteiro CM[1], Almeida CJ[1], Silva AR[1], Castro-Faria-Neto HC[1], Bozza PT[1] [1]FIOCRUZ − Fisiologia Farmacodinâmica, [2]FIOCRUZ − Microbiologia

04.091
Role of PPARγ in macrophage activation but not in neutrophil recruitment during *Mycobacterium bovis* BCG infection *in vivo*. Sette-Martins R, Almeida PE, Roque NR, Bozza PT IOC-FIOCRUZ − Imunofarmacologia

04.092
Role of substance P in different endogenous pyrogen-induced fever. Brito HO, Reis RC, Zampronio AR UFPR − Farmacologia

05. Pain and Nociception

05.027
Resistance exercise induces antinociception in rats with participation of nitric oxide/$_{C}$GMP/K_{ATP} pathway. Galdino GS, Silva GC, Almeida RT, Duarte ID, Perez AC UFMG − Farmacologia

05.028
Peripheral sensitization increases opioid receptor activation and expression in both dorsal root ganglia and nerve paw of rats. Zambelli VO[1], Gutierrez VP[1], Fernandes ACO[1], Parada CA[2], Cury Y[1] [1]IBu − Dor e Sinalização, [2]UNICAMP − Farmacologia

05.029
The sesquiterpene lactone, budlein A, inhibits antigen induced-arthritis inflammation in mice. Zarpelon AC[1], Pinto LG[2], Souto FO[2], Turato W[3], Arakawa NS[4], Da Costa FB[4], Cunha TM[2], Ferreira SH[2], Cunha FQ[2], Silva JS[5], Verri Jr WA[1] [1]UEL − Ciências Patológicas, [2]FMRP-USP − Farmacologia, [3]FCFRP-USP − Análises Clínicas,

Toxicológicas e Bromatológicas, [4]FCFRP−USP, [5]FMRP-USP − Imunologia

05.030

LASSBio-294 has partial agonist and antagonistic actions on TRPV1. Munaro DV[1], Barreiro EJ[2], Fraga CAM[2], Castro NG[3], Guimarães MZP[4] [1]UFRJ − Biofísica, [2]FF-UFRJ − LASSBio, UFRJ, [3]UFRJ − Farmacologia Molecular, [4]UFRJ − Farmacologia Básica e Clínica

05.031

Study of anti-inflammatory and antinociceptive properties of new derivatives rationally designed as PPAR agonists. Santos BLR, Lima CKF, D´Andrea ED, Lima LM, Barreiro EJ, Miranda ALP FF-UFRJ − LASSBio

05.032

Ketamine/fentanyl administration in infant rats promotes analgesia associated with increased hydrolysis of nucleotides. Medeiros LF[1], Souza A[2], Rozisky JR[1], Santos VS[1], Netto CA[2], Battastini AMO[2], Torres ILS[1] [1]UFRGS − Farmacologia, [2]UFRGS − Bioquímica

05.033

Evaluation of some mechanisms involved in antinociceptive effect of (-)epicatechin obtained from *Combretum leprosum* Mart. & Eicher (Combretaceae) in models of acute pain. Lopes LS[1], Pereira SS[1], Marques RB[1], Ayres MCC[2], Chaves MH[2], Almeida FRC[3] [1]NPPM-CCS-UFPI, [2]UFPI − Chemistry, [3]UFPI − Biochemistry and Pharmacology

05.034

Preliminary studies of possible mechanisms involved in the antinociception presented by α-terpineol, a major constituent of essential oil from *Protium heptaphyllum* March. resin. Marques RB[1], Lopes LS[1], Fernandes, HB[1], Pereira SS[1], Chaves MH[2], Oliveira, FA[1], Almeida FRC[3] [1]NPPM-CCS-UFPI, [2]UFPI − Chemistry, [3]UFPI − Biochemistry and Pharmacology

05.035

Antinociceptive effect of (-) epicatechin obtained from *Combretum leprosum* Mart. & Eicher (Combretaceae) in models of acute pain. Lopes LS[1], Fernandes, HB[1], Pereira SS[1], Marques, RB[1], Ayres MCC[2], Chaves MH[2], Almeida FRC[1] [1]NPPM-CCS-UFPI, [2]CCN-UFPI − Química

05.036

Antinociceptive activity of new isatin derivatives. Figueiredo GSM[1], Zardo RS[1], Silva BV[2], Matheus ME[1], Pinto AC[3], Fernandes PD[1] [1]UFRJ − Farmacologia Básica e Clínica, [2]IQ-UFRJ − Química Orgânica, [3]UFRJ − Química

05.037

Involvement of adenosinergic system in the antinociceptive effect of ethanolic extract of *Cipura paludosa* Aubl. in mice. Macedo Junior SJ[1], Lucena GMRS[2], Nascimento FP[3], Cerutti M[3], Santos ARS[1] [1]UFSC − Ciências Fisiológicas, [2]UnB − Ciências da Saúde, [3]UFSC − Farmacologia

05.038

Direct blockade of inflammatory hypernociception by peripheral activation of the A1 adenosine receptor: involvement of the NO/cGMP/PKG/KATP signaling pathway. Cunha TM[1], Lima FO[1], Souza GR[1], Verri Jr WA[2], Parada CA[3], Ferreira SH[1], Cunha FQ[1] [1]FMRP-USP − Farmacologia, [2]UEL − Ciências Patológicas, [3]UNICAMP − Farmacologia, [5]FMRP-USP

05.039

Ropivacaine gel for topical anesthesia: *in vitro* permeation skin and cytotoxic effects. Stoco SM[1], Grillo R[2], Mello NFS[2], Guilherme VA[1], Franz-Montan M[1], Tófoli GR[3], Fraceto LF[2], de Paula E[1], de Araújo DR[4] [1]UNICAMP − Bioquímica, [2]UNESP – Engenharia Ambiental, [3]UNIFAG − Farmacologia Clínica, [4]CCNH-UFABC − Farmacologia

05.040

The armed spider toxin TX3-3 restores the analgesic effect of morphine in neuropathic and opioid-tolerant mice. Dalmolin GD[1], Rigo FK[1], Silva CR[2], Gomez MV[1], Ferreira J[2] [1]UFMG − Farmacologia, [2]UFSM − Química

05.041

Celecoxib induces analgesia by release of B-Endorphin in rat paws. Paiva-Lima P[1], Queiroz Junior CM[1], Rezende RM[2], Machado-Silva LDF[2], Caliari MV[3], Bakhle YS[4], Francischi JN[1] [1]UFMG − Farmacologia, [2]UFMG − Fisiologia e Farmacologia, [3]UFMG − Patologia, [4]Imperial College − Leukocyte Biology

05.042

Antinociceptive and anti-inflammatory effect of electroacupuncture in zymosan-induced arthritis in the rat temporomandibular joint. Gondim DV[1], Chaves, HV[1], Costa JL[2], Rocha SS[2], Brito GAC[3], Vale ML[2] [1]UFC − Medicina Clínica, [2]UFC − Fisiologia e Farmacologia, [3]UFC − Morfologia

05.043

Macrophage Migration Inhibitory Factor (MIF) is involved in a cascade of events leading to inflammatory hypernociception in mice. Costa VV[1], Amaral FA[2], Sachs D[3], Tavares LD[4], Scopa IP[2], Morcatty TQ[1], Teixeira MM[1], Souza DG[2] [1]UFMG − Bioquímica e Imunologia, [2]UFMG − Microbiologia, [3]FMRP-USP − Farmacologia, [4]UFMG − Fisiologia e Farmacologia

05.044

Role of TRPV1 and NK1 receptors on nociception and edema induced by monosodium urate crystals in rats (MSU). Trevisan G, Rossato M, Hoffmeister C, Ferreira J UFSM − Química

05.045
The antinociception observed during glycogen-induced inflammation in rat paws is mediated by neutrophil migration and is independent of opioid peptides. Nogueira TO[1], Spadacci-Morena DD[1], Santoro ML[1], Pagano RL[2], Giorgi R[1] [1]IBu – Fisiopatologia, [2]IEP-HSL

05.046
Ropivacaine gel for topical anesthesia: *in vitro* permeation skin and cytotoxic effects. Stoco SM[1], Grillo R[2], Mello NFS[2], Guilherme VA[1], Franz-Montan M[1], Tófoli GR[3], Fraceto LF[2], de Paula E[1], de Araújo DR[4] – [1]UNICAMP – Bioquímica, [2]UNESP – Engenharia Ambiental, [3]UNIFAG – Farmacologia, [4]CCNH-UFABC – Farmacologia

05.047
Cannabinoid and opioid receptors activation induces peripheral antinociception by noradrenaline release and α_{2C} adrenoceptor interaction. Romero TRL[1], Duarte IDG[2] [1]UFMG – Fisiologia e Farmacologia, [2]UFMG – Farmacologia

05.048
Antinociceptive effects of *Parkia platycephala* Benth in diabetic rats. Amorim VR[1], Brito SRMC[2], Sales Filho HLA[3], Piauilino, CA[4], Chaves MH[5], Bezerra RDS[5] [1]UFPI – Farmacologia, [2]UFPI – Bioquímica e Farmacologia, [3]UFPI – Farmacologia, [4]UFPI-NPPM-UFPI, [5]UFPI – Química

05.049
Effects of thalamic nucleus submedius inhibition on the stimulation-induced antinociception in rats Reis GM, Rossaneis AC, Fais RS, Prado WA FMRP-USP – Farmacologia

05.050
Amitriptyline increases the duration of the antinociceptive effect produced by 2 Hz electroacupuncture in rats. Fais RS, Reis GM[2], Dias QM[1], Silveira JWS[1], Prado WA[3] FMRP-USP – Farmacologia

05.051
Role of IL-33 / ST2 in carrageenin-induced innate inflammatory hypernociception in mice. Zarpelon AC[1], Cunha, TM[2], Xu D[3], Alves-Filho JC[3], Liew FY[3], Ferreira SH[2], Cunha FQ[2], Verri Jr WA[1] [1]UEL – Patologia, [2]FMRP-USP – Pharmacology, [3]University of Glasgow – Immunology, Infection and Inflammation

05.052
Antinociceptive property of selenothiazolidines administered by oral route in mice. Frasson NR[1], Donato F[1], Schneider PH[2], Savegnago L[1] [1]UNIPAMPA – Farmacologia e Toxicologia, [2]UFRGS – Química

06. Cardiovascular and Renal Pharmacology

06.027
Cardiac dysfunction in experimental sepsis as assessed by the isolated and perfused mouse heart. Bóf ER, DalBó S, Ramos GC, Assreuy J UFSC – Pharmacology

06.028
Sepsis-induced renal impairment to a second renal insult. Portella VG[1], Silva-Filho JL[1], de Rico TB[2], Landgraf SS[1], Vieira MAR[3], Takiya CM[4], Benjamim CF[2], Canetti C[1], Pinheiro AAS[1], Caruso-Neves C[1] [1]IBCCF-UFRJ – Ciências da Saúde, [2]ICB-UFRJ – Farmacologia, [3]ICB-UFMG – Fisiologia e Biofísica, [4]ICB-UFRJ – Anatomia e Histologia

06.029
Effects of antioxidants treatment on cardiac dysfunction and MMP-2 levels in renovascular hypertension. Rizzi E[1], Castro MM[1], Ceron CS[1], Neto-Neves EM[1], Tanus-Santos JE[1], Gerlach RF[2] [1]FMRP-USP – Farmacologia, [2]FORP-USP – Morfologia

06.030
Role of potassium channels in endothelium-dependent vasodilation in experimental periodontits in rats. Olchanheski Junior LR[1], Santos FA[2], Fernandes D[1] [1]UEPG – Ciências Farmacêuticas, [2]UEPG – Odontologia

06.031
Inhibition of MMP-mediated vascular changes in 2K1C hypertension by doxycycline is dose-dependent. Guimarães DA[1], Rizzi E[1], Ceron CS[1], Oliveira AM[2], Marçal DMO[5], Tirapelli CR[5], Gerlach RF[6], Tanus-Santos JE[1] [1]FMRP-USP – Farmacologia, [2]FCFRP-USP – Farmacologia, [5]EERP-USP – Farmacologia, [6]FORP-USP – Morfologia

06.032
New nitrite-pro-drug releases nitric oxide in a tissue and enzyme-dependent way. Pereira AC[1], Lunardi CN[2], Biazzotto JC[1], Silva RS[1], Bendhack LM[1] [1]FCFRP-USP, [2]UnB

06.033
Effect of rosmarinic acid on the inhibition of angiotensin converting enzyme in normotensive and hypertensive rats. Ferreira LG, Celotto AC, Capellini VK, Albuquerque AAS, Evora PRB FMRP-USP – Cirurgia e Anatomia

06.034
Modulation of cardiac and renal P-type ATPases in diet-induced atherosclerosis. Balter AS, Marques EB[1], Motta NAV[1], Brito FCF[1], Scaramello C[1] [1]UFF – Farmacologia Experimental

06.035
A new vasodilator compound (DCBPY-NO) presents cyclic activity in releasing nitric oxide by nitrite. Rodrigues GJ[1], Cicillini SA[2], Silva RS[2], Bendhack LM[2] [1]FMRP USP − Farmacologia, [2]FCFRP−USP

06.036
The renal effects of l-amino acid oxidase from *Bothrops leucurus* venom in the rat perfused kidneys. Morais ICO[1], Marinho AD[2], Menezes RRPPB[3], Dantas RT[1], Torres AFC[2], Lopes KS[2], Meneses GC[2], Costa MFB[2], Jorge RJB[1], Alves RS[1], Toyama MH[4], Monteiro HSA[1], Martins AMC[3] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Farmácia, [3]UFC − Análises Clínicas e Toxicológicas, [4]IB-UNICAMP

06.037
Tempol attenuates the hemodynamic changes associated with acute pulmonary embolism. Santos Sousa O[1], Neto-Neves EM[1], Ferraz KC[2], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]UFJF − Farmacologia

06.038
Impaired cardiovascular responsiveness to isoprenaline in rats subjected to high-salt intake. Crestani S[1], de Souza P[1], Bóf ER[2], Guarido KL[2], Assreuy J[2], Marques MCA[1], da Silva-Santos JE[2] [1]UFPR − Farmacologia, [2]UFSC − Farmacologia

06.039
High salt-intake increases the activity of angiotensin-converting enzyme in rats. Crestani S[1], Gasparotto Junior A[2], Marques MCA[1], da Silva-Santos JE[3] [1]UFPR − Farmacologia, [2]UNIPAR/UFPR − Farmacologia, [3]UFSC − Farmacologia

06.040
Quercetin produces beneficial effects in renovascular hypertension. Neto-Neves EM, Montenegro MF, Ceron CS, Dias-Junior CAC, Castro MM, Tanus-Santos JE FMRP-USP − Farmacologia

06.041
Neonatal hyperleptinaemia possibly modulates cardiac function. Marques EB[1], Balter AS[1], Pereira-Toste F[2], Raimundo JM[3], Sudo RT[3], Zapata-Sudo G[3], Marques SA[4], Vieyra A[5], Scaramello C[1] [1]UFF − Farmacologia Experimental, [2]UFF − Ciências do Exercício, [3]UFRJ − Farmacologia Básica e Clínica, [4]UFRJ − Histologia e Embriologia, [5]IBCCF-UFRJ

06.042
High-salt intake impairs the involvement of Rho-A/Rho-kinase and intracellular calcium in contractile responses of rat aortic rings. Crestani S[1], Marques MCA[1], da Silva-Santos JE[2] [1]UFPR − Farmacologia, [2]UFSC − Farmacologia

06.043
A sulfonamide compound attenuates vascular smooth muscle contraction and lowers arterial pressure of normotensive and spontaneously hypertensive rats. Pontes LB[1], Raimundo JM[1], Sudo RT[1], Lima LM[2], Barreiro EJ[2], Zapata-Sudo G[1] [1]UFRJ − Farmacologia Básica e Clínica, [2]FF-UFRJ − LASSBio

06.044
New NO donor induces relaxation of mesenteric resistance arteries and reduces resistance of mesenteric bed of normotensive and 2K-1C hypertensive rats. Araújo AV[1], Rodrigues GJ[1], Vercesi JA[2], Biazzotto JC[2], Bonagamba LGH[3], Machado BH[3], Silva RS[2], Bendhack LM[2] [1]FMRP-USP − Farmacologia, [2]FCFRP-USP − Física e Química, [3]FMRP-USP − Fisiologia

06.045
Vasodilation induced by atrial natriuretic peptide (ANP) involves K_{ATP} channels activation in rat aorta. Andrade FA[1], Bendhack LM[2] [1]FMRP-USP, [2]FCFRP-USP

06.046
Cardiovascular hyporresponsiveness in severe sepsis is related with augment of G-protein receptor kinase (GRK)-2 expression via a nitric oxide-dependent mechanism. Dal-Secco D[1], Olivon VC[2], Corrêa T[1], Celes MRN[3], Abreu A[3], Rossi MA[3], Oliveira AM[2], Cunha FQ[4], Assreuy J[1] [1]UFSC − Farmacologia, [2]FCFRP-USP − Física e Química, [3]FMRP-USP − Patologia, [4]FMRP-USP – Farmacologia

06.047
Vasorelaxant effect of Isotirumalin, a dihydroflavonol from *Derris urucu*, on rat aorta. Mendes LJ[1], Capettini LSA[2], Arruda MSP[3], Lemos VS[2], Côrtes SF[1] [1]UFMG − Farmacologia, [2]ICB-UFMG − Fisiologia e Biofísica, [3]UFPA − Química

06.048
Echocardiography aspects of structural changes in different morphological and functional models of hypertension in SHR. Pereira DJ[1], Gazzoto AF[1], Pires NF[1], Moreira MM[1], Santos RC[1], Ludovico ND[1], Quinaglia TSS[1], Renno AL[2], Figueiredo VN[1], Moreno Junior H[1] [1]UNICAMP − Farmacologia cardiovascular, [2]UNICAMP − Farmacologia Bioquímica

06.049
Development and validation of analytical method for quantification of arsenic and antimony in liposomes. Reis PG, Souza J, Teixeira MC, Grabe-Guimarães A, Silva-Barcellos NM UFOP − Farmácia

06.050
Spontaneously hypertensive rats (SHR) under chronic treatment with sodium fluoride showed reduced fluoride and calcium concentrations in plasma and saliva. Picco DCR[1], Delbem ACB[1],

Antoniali C[2] [1]FOA-UNESP − Odontologia Infantil e Social, [2]UNESP-Araçatuba − Ciências Básicas

06.051

H_2O_2 -induced vasodilatation through neuronal nitric oxide synthase activation by a natural xanthone. Capettini LSA[1], Silva JF[1], Dos Santos MH[2], Nagem TJ[3], Côrtes SF[4], Lemos VS[1] [1]ICB-UFMG Fisiologia e Biofísica, [2]UNIFAL-MG − Farmácia, [3]UFOP − Química, [4]UFMG − Farmacologia

06.052

Consequences of acute or chronic stress on relaxation induced by angiotensin 1-7 in rat carotid. Banin TM[1], Olivon VC[1], Ramalho L[2], de Oliveira AM[1] [1]USP − Física e Química, [2]USP − Patologia

09. Natural Products and Toxinology

09.036

Investigation of gastroprotective effect and 50 lethal concentration of the ethanolic extract from *Combretum duarteanum cambess* (Combretaceae). Lima GRM, Montenegro CA, Almeida CLF, Pessoa DR, Moreira MMB, Castello Branco MVS, Tavares JF, Batista LM LTF-DCF-UFPB

09.037

Mechanisms involved in the antinociceptive effect of the ethanolic extract from the leaves of C*eltis iguanaea* (jacq.) Sargent (Ulmaceae). Nascimento MVM[1], Lino RC[1], Sousa BF[1], Florentino FI[1], Galdino PM[1], Couto, RO[2], Paula JR[2], Costa EA[1] [1]ICB-UFG, [2]UFG − Farmácia

09.038

Effects of amblyomin-X on tumor growth, endothelial cell migration, adhesion and secretion. Dias RYS[1], Drewes CC[1], Hebeda CB[1], Simons SM[2], Chudzinski-Tavassi AM[2], Farsky S[1] [1]FCF-USP Análises Clínicas e Toxicológicas, [2]IBu − Laboratório de Bioquímica

09.039

Healing properties of bark extract of *Tabebuia avellanedae* in chronic gastric ulcer induced by acetic acid in rats. Pereira IT[1], Burci LM[1], da Silva LM[1], Baggio CH[1], Andre E[2], Pizzolatti MG[3], Marques MCA[1], Werner MFP[4] [1]UFPR – Farmacologia, [2]UFRN − Farmacologia, [3]UFSC – Química, [4]UFSC − Farmacologia

09.040

Subfraction of *Pterodon pubescens* seeds oil induces apoptosis of leukemic cells by inducing Apaf-1 gene expression. Martino T, Pereira MF, Dalmau SR, Silva MCC, Coelho MGP, Sabino KCC UERJ − Bioquímica

09.041

Antinociceptive effects of (1→3),(1→6)-linked b-glucan isolated from *Pleurotus pulmonarius* in models of acute and neuropathic pain in mice. Baggio CH[1], Freitas CS[1], Werner MFP[2], Martins DF[3], Mazzardo L[3], Smiderle FR[4], Sassaki GL[4], Iacomini M[4], Marques MCA[1], Santos ARS[5] [1]UFPR − Farmacologia, [2]UFSC − Farmacologia, [3]UFSC − Fisiologia, [4]UFPR − Bioquímica, [5]UFSC − Ciências Fisiológicas

09.042

Effect of p-cymene obtained from *Citrus latifolia* Tanaka essential oil on *in vitro* leukocytes chemotaxis. Kummer R[1], Fachini FC[1], Silva CFE[1], Freitag A[1], Silva EL[2], Grespan R[1], Bersani-Amado CA[1], Cuman RKN[1] [1]UEM − Farmácia e Farmacologia, [2]UEM − Química

09.043

Effect of eugenol treatment on renal parameters after renal ischemia and reperfusion in mice. Damião MJ[1], Victor ML[1], Fonseca JP[1], Bersani-Amado CA[1], Rilson JP[1], Giannocco G[2], Câmara NOS[3], Cuman RKN[1] [1]UEM − Farmácia e Farmacologia, [2]UNIFESP − Endocrinologia, [3]ICB-USP

09.044

Cardiovascular activity *Hancornia speciosa* ethanolic extract in a model of hypertension induced by nitric oxide synthesis inhibition in rats. Silva MDA[1], Serra CP[2], Grabe-Guimarães A[2], Guimarães HN[3], Braga FC[4] [1]CiPharma-UFOP, [2]DEFAR-UFOP, [3]UFMG − Engenharia Elétrica, [4]FaFar -UFMG

09.045

Investigation of toxic and antidiarrhoeal activities of ethanol extract of aerial parts from *Xylopia langsdorffiana* A. St. Hil. & Tul. (Annonaceae) in mice. Silva KM[1], Silva ADS[1], Lima LO[1], Clementino-Neto J[1], Silva PCB[2], Medeiros VM[2], Costa VCO[2], Tavares JF[2], Silva MS[2], Cavalcante FA[1] [1]ICBS-UFAL, [2]LTF-UFPB

09.046

Investigation of topical anti-inflammatory activity of *Vochysia bifalcata*. Silva CD[1], Mendes DAGB[1], Soley BS[1], Ferreira BGA[2], Zuffellato-Ribas KC[2], Otuki MF[1], Cabrini DA[1] [1]UFPR − Farmacologia, [2]UFPR − Botânica

09.047

Anti-inflammatory effect of latex proteins (LP) isolated from *calotropis procera* in 5-fluorouracil-induced oral mucositis in hamsters. Freitas APF[1], Almeida RA[2], Cerqueira GS[2], Alencar NMN[2], Brito GAC[3], Ribeiro RA[2], Ramos MV[4], Vale ML[2] [1]UFC − Medicina Clínica, [2]UFC − Fisiologia e Farmacologia, [3]UFC − Morfologia, [4]UFC − Bioquímica

09.048

Evaluation of antimicrobial activity and preliminary phytochemical profile of *Hyptis suaveolens* L. Poit. Jesus NZT[1], Mota FD[2], Silva Junior IF[3], Tavares JF[4], Batista LM[5] [1]UNIC-UFPB-LTF, [2]UNIC − Farmácia, [3]UFMT − Farmacologia, [4]UFPB − Tecnologia Farmacêutica, [5]UFPB − Ciências Farmacêuticas

09.049

Acute toxicity and gastric cytoprotective effect of *Argyrovernonia harleyi* (H. ROB) Macleish in mice. Silva AAR[1], Bezerra MM[3], Aguiar, JA[2], Chaves, HV[4], Ribeiro, KA[5], Pereira, KMA[4], Maia, MBS[6] [1]UFC − Pharmacology Laboratory, [2]UFC − Medicine, [3]UFC − Biotechnology, [4]UFC − Pharmacology Laboratory, [5]UVA − Pharmacology, [6]UFPE − Pharmacology and Toxicology of Bioactive Products

09.050

(+)-cordiaquinone J triggers both death receptor-dependent apoptosis and necrosis by oxidative stress pathway in leukemia cells. Marinho-Filho JDB[1], Araújo AJ[1], Bezerra DP[2], Montenegro RC[3], Pessoa C[1], Diniz J[3], Viana FA[4], Pessoa ODL[4], Silveira ER[4], Moraes MO[1], Costa-Lotufo LV[1] [1]UFC − Fisiologia e Farmacologia, [2]UFAL, [3]UERN − Química, [4]UFC − Química Orgânica e Inorgânica

09.051

Anti-inflammatory activity of anethole obtained from *Foeniculum vulgare* Miller var. *vulgare* Miller essential oil. Domiciano TP[1], Ritter AMV[1], Silva EL[2], Dantas JA[1], Caparroz-Assef SM[3], Cuman RKN[1], Bersani-Amado CA[1] [1]UEM − Farmácia e Farmacologia, [2]UEM − Química, [3]UEM − Inflamação

09.052

Evaluation of antimicrobial activity of ethanol extract of the aerial parts of *Nanuza plicata* (Mart.) L. B. Smith & Ayensu. Tenório JAB[1], Mendes JM[1], Falcão HS[1], Lima EO[2], Marculino DMM[1], Tavares JF[1], Batista LM[1], Montes RC[1] [1]UFPB − Ciências Farmacêuticas, [2]UFPB − Micologia

09.053

Evaluation of the effects of jatobá juice concentrating on the glucemic control, lipid profile and liver function of diabetic rats. Almeida IP[1], Damasceno DCF[1], Sales ALCC[2], Teixeira JMR[1], Soares LFM[1], Santos Júnior JC[1], Amorim VR[3], Silva AFS[3], Assis RC[3], Martins MCC[1] [1]UFPI − Biophysics and Physiology, [2]UFPI − Nutrition, [3]UFPI − Biochemistry and Pharmacology

09.054

Inhibitory effects of ginger (*Zingiber officinale* Roscoe) essential oil on *in vivo* and *in vitro* leukocytes migration. Grespan R, Nogueira de Melo GA, Fonseca JP, Farinha TO, Dantas JA, Miranda CR, Bersani-Amado CA, Cuman RKN UEM − Pharmacy and Pharmacology

09.055

Hypoglycemic effect of *Terminalia catappa* Linn. in alloxan-induced diabetic rats. Ferreira AKB[1], Costa DL[2], Tenório EP[1], Oliveira DA[1], Santana AEG[2], Humberto MMS[2], Grillo LAM[1], Ribeiro EAN[1] [1]ESENFAR-UFAL, [2]IQB-UFAL

09.056

Fraction from *Calotropis procera* Latex shows anti-inflammatory effects on the pathogenesis of irinotecan-induced intestinal mucositis in mice. Alverne SM[1], Bitencourt FS[1], Figueiredo JG[2], Luz PB[1], Lima-Júnior RCP[1], Ramos MV[3], Cunha FQ[4], Ribeiro RA[1], Alencar NMN[1] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Bioquímica e Biologia Molecular, [3]UFC − Bioquímica, [4]FMRP-USP

09.057

Ability of fucosylated chondroitin sulfate to inhibit muscle damage induced by *Bothrops jararacussu* crude snake venom. Machado MM[1], Strauch MA[1], Tomaz MA[1], Cons BL[1], Branco AMC[1], Martins VV[1], Mourão PAS[2], Melo PA[1] [1]UFRJ − Farmacologia Básica e Clínica, [2]UFRJ − Bioquímica Médica

09.058

Evaluation of the toxicity of the ethanol extract of aerial parts of *Nanuza plicata* (Mart.) L. B. Smith & Ayensu.Tenório JAB[1], Falcão HS[1], Viana WP[2], Dias GEN[1], Batista LM[1], Diniz MFFM[1], Tavares JF[1] [1]LTF-UFPB − Ciências Farmacêuticas, [2]UFPB − Ciências da Saúde

09.059

Fish oil supplementation on motor and cognitive side effects of typical antipsychotics in psychiatric patients. Bürger ME[1], Cardoso PM[2], Reckziegel P[2], Pase CS[2], Emanuelli, T[3], Santos DB[4], Cunha A[5], Rocha JBT[4] [1]UFSM − Farmacologia, [2]UFSM − Fisiologia e Farmacologia, [3]UFSM − Tecnologia e Ciência dos Alimentos, [4]UFSM − Química, [5]UFSM − Neuropsiquiatria

09.060

Anti-inflammatory activity of the hydroalcoholic extract and fractions from *Gochnatia polymorpha ssp floccosa* in mouse air pouch model. Piornedo RR[1], Kassuya CAL[2], Zampronio AR[1], Stefanello MEA[3], Strapasson RLB[3] [1]UFPR − Farmacologia, [2]UFGD − Ciências da Saúde, [3]UFPR − Química

09.061

In vitro chlorogenic acid inhibits adhesion molecules expression and inflammatory mediators secretion in neutrophils. Bolonheis SM[1], Hebeda CB[1], Belinati KD[1], Lopes NP[2], Farsky S[1] [1]FCF-USP − Análises Clínicas e Toxicológicas, [2]FCFRP−USP − Física e Química

09.062

Topical effect of crude hydroalcoholic extract from *Psychotria nuda* (Cham. & Schltdl.) Wawra leaves in skin inflammation model. Mendes DAGB[1], Soley BS[1], Ferreira BGA[2], Zuffellato-Ribas KC[2], Otuki MF[1], Cabrini DA[1] [1]UFPR − Farmacologia, [2]UFPR − Botânica

09.063

Ethanol crude extract of *Erythroxylum caatingae* induces relaxant effect in the guinea-pig trachea. Santos HAS[1], Oliveira SL[2], Tavares JF[2], Ribeiro LAA[3], Lima JT[3] [1]UNIVASF − Medicina, [2]UFPB −

Tecnologia Farmacêutica, [3]UNIVASF − Ciências Farmacêuticas

09.064

Effect of LED treatment on muscular edema and mionecrose induced by *Bothrops jararaca* venom. Bulgarelli[1], Barbosa AM[2], Lima CJ[3], Zamuner SR[4] [1]UNIVAP − Fisiologia e Inflamação, [2]UNIVAP − Pesquisa e Desenvolvimento, [3]UNICASTELO − Instrumentação Optobiomédica, [4]UNINOVE − Ciências da Reabilitação

09.065

Modulation of gene expression in melanoma cells by treatment with crotamine. Moura AB[1], Yonamine CM[1], Pellegrino R[2], Oliveira EB[3], Yamane T[4], Lapa AJ[1], Hayashi MA[1] [1]UNIFESP − Farmacologia, [2]UNIFESP − Psicobiologia, [3]FMRP-USP − Biochemistry and Immunology, [4]CBA − Bioquímica e Biologia Molecular

09.066

Preliminary biochemical and pharmacological characterizations of *Rhinela icterica* toad venom. Pesamosca ME, Freitas TC, Franco JL, Dal Belo CA UNIPAMPA

09.067

Evaluation of antinociceptive property of ethanolic extract of *Sidastrum micranthum* (A. St.-Hil.) Fryxell. Villa JKD[1], Marinho DG[2], Dias DM[1], Ramiro JB[1], Scherrer JV[1], Faccim AG[1], Almança CCJ[3], Marinho BG[1] [1]UFES − Medicina Veterinária, [2]ICB-UFRJ − Farmacologia e Química Medicinal, [3]FAFIA − Farmácia

09.068

Endothelium-dependent and independent relaxation of rat aortic ring by crude extracts and fractions from *Scutia buxifolia*. Silva RCMVAF[1], Crestani S[1], Boligon AA[2], Athayde ML[2], Santos ARS[3], Marques MCA[1], Kassuya CAL[1], da Silva-Santos JE[4] [1]UFPR − Farmacologia, [2]UFSM − Farmácia Industrial, [3]UFSC − Ciências Fisiológicas, [4]UFPA − Farmacologia Experimental e Pré-clínica

09.069

Anti-inflammatory and antinociceptive activity of the ethanolic extract from *Sinningia leucotricha*. Botelho A[1], Verdan ML[2], Stefanello MEA[2], Kassuya CAL[3], Zampronio AR[1] [1]UFPR − Farmacologia, [2]UFPR − Química, [3]UFGD − Ciências da Saúde

09.070

Preliminary evaluation of wound healing activity of the aqueous extract from stem bark of *Bowdichia virgilioides*. Agra IKR, Santos TC, Smaniotto S, Barreto E UFAL − Biologia Celular

11. Clinical Pharmacology, Pharmacokinetics, Pharmacogenomics and Preclinical Toxicology

11.012

Efficacy evaluation of the therapeutic protocols used to treatment of TMDR patients in a respiratory diseases reference centre, in Salvador, Bahia, Brazil. Pitanga QML[1], Santos CBS[1], Pitta LB[1], Pinheiro CG[1], Carvalho JSM.[2], Carvalho FLDQ[1] [1]UNEB − Ciências da Vida, [2]UNIJORGE − Saúde

11.013

Quantification of cyproheptadine in human plasma by high-performance liquid chromatography coupled to electrospray tandem mass spectrometry in a bioequivalence study. Arruda AMM[1], Mendes GD[1], Chen LS[2], Nucci G[1] [1]FCM-UNICAMP − Farmacologia/FCM, [2]Galeno Research Unit − Bioequivalence

11.014

Multi-drug resistance tuberculosis: association between comorbidities, drug resistance and treatment. Santos CBS[1], Pitanga QML[1], Pitta, LB[2], Pinheiro CG[2], Carvalho JSM[2], Carvalho FLDQ[1] [1]UNEB − Ciências da Vida, [2]UNIJORGE − Saúde

11.015

Specific matrix metalloproteinase-9 (MMP-9) genotype and haplotype in obese children and adolescents. Belo VA[1], Souza-Costa DC[2], Carneiro PC[3], Lanna CM[4], Izidoro-Toledo TC[3], Gerlach RF[5], Tanus-Santos JE[3] [1]UNICAMP − Farmacologia, [2]UFJF − Farmacologia, [3]FMRP-USP − Farmacologia, [4]UFJF − Fisiologia, [5]FORP-USP − Morfologia

11.016

Adverse reactions to chemotherapy for breast cancer: influence of clinical and histopathologic variables. Índio-do-Brasil V[1], Telles C[1], Vianna-Jorge R[2], Koifman S[3] [1]INCa − Farmacologia, [2]UFRJ − Farmacologia Básica e Clínica, [3]ENSP-FIOCRUZ − Saúde Pública e Meio Ambiente

11.017

Evaluation of the toxicity of the ethanolic extract of *Gracilaria ferox* J. Agardh (Gracilariaceae) leaves. Almeida CLF, Falcão HS, Montenegro CA, Lima GRM, Ramirez RRA, Souza MFV, Batista LM LTF-DCF-UFPB

11.018

Adverse reactions to chemotherapy for breast cancer and impact of genetic polymorphisms. Martins CL[1], Índio-do-Brasil V[1], Telles C[1], Vianna-Jorge R[2], Koifman S[3] [1]INCa − Farmacologia, [2]UFRJ − Farmacologia Básica e Clínica [3]ENSP-FIOCRUZ –Saúde Pública e Meio Ambiente

11.019
Pre-clinical pharmacokinetic study of LASSBio-468: a new achiral thalidomide analogue. Kaiser M[1], Haas SE[2], Azeredo FJ[2], Torres B[2], Brum Junior L[2], Uchôa FDT[1], Contri, RV[2], Lima LM[3], Barreiro EJ[3], Dalla Costa T[2] [1]UFRGS − Medicamentos, [2]UFRGS − Ciências Farmacêuticas, [3]UFRJ − LASSBio

11.020
Assessment of the pharmaceutical equivalence of captopril and propranolol hydrochloride tablets sold in the popular pharmacy program in Brazil. Pontes AV, Pimenta Costa CS, Nascimento DF, Leite ALAS, Capistrano Júnior VL, Rocha MBS, Moraes RA, Frota Bezerra FA, Moraes MEA, Moraes MO UFC − Fisiologia e Farmacologia

11.021
Reduction of nitric oxide levels after intervention with sitagliptin in type 2 diabetic patients. Capistrano Júnior VL[1], Tagliapietra JI[1], Oliveira JC[1], Pimenta Costa CS[1], Souza MHLP[1], Montenegro Jr RM[2], Leite ALAS[1], Frota Bezerra FA[1], Vale OC[1], Moraes MEA[1] [1]UFC − Fisiologia e Farmacologia, [2]HU-UFC − Endocrinologia e Metabologia

11.022
Learning and teaching pharmacology: a case study in Rio de Janeiro, Brazil. Fidalgo-Neto AA, Lopes RM, Alves LA IOC-FIOCRUZ − Comunicação Celular

01. Cellular and Molecular Pharmacology

01.028
The C-Rel subunit from nuclear factor kappa B (NFKB) family may play a differential role in peripheral melatonin synthesis. Lapa MAPC, Ferreira ZS, Markus RP [1]IB-USP − Fisiologia

01.029
NADPH oxidase mediates heme-induced cytoskeletal alterations, endothelial permeability and increased expression of adhesion molecules in HUVEC. Nascimento-Silva V[1], Morandi V[2], Barja Fidalgo TC[1], Arruda MA[3] [1]UERJ − Farmacologia, [2]UERJ − Biologia Celular, [3]FIOCRUZ − Farmanguinhos

01.030
Testosterone induces VSMC proliferation via P38-COX2-dependent, NFKB- independent pathways. Chignalia AZ[1], Munhoz CD[1], Yogi A[1], Camargo LL[1], Oliveira MA[1], Lopes LR[1], Rossoni LV[2], Carvalho MHC[1], Fortes ZB[1], Tostes RCA[1] [1]ICB-USP − Farmacologia, [2]ICB-USP − Fisiologia e Biofisica

01.031
Modulation of gastrointestinal epithelial cells activation by heme. Barcellos-de-Souza P[1], Nasciutti LE[2], Barja Fidalgo TC[1], Arruda MA[3] [1]UERJ − Farmacologia, [2]UFRJ − Histologia e Embriologia, [3]FIOCRUZ − Farmanguinhos

01.032
Schild analysis of the self cancelling effects of tricyclic antidepressants on alpha-1 adrenoceptor mediated responses. Nojimoto FD, Pupo AS UNESP − Farmacologia

01.033
MicroRNA *let-7b* targets AKT-1 and regulates skeletal muscle atrophy in diabetic rats. Sousa TA[1], Kato M[2], Paula-Gomes, S.[1], Silva VAO[1], Tragante V[1], Zanon NM[1], Wang M[2], Kettelhut IC[1], Natarajan R[2], De Lucca FL[1] [1]FMRP-USP – Biochemistry and Immunology, [2]Beckman Research Institute − Gonda Diabetes Center

01.034
Expression of MicroRNAS in skeletal muscle atrophy induced by fasting in rats. Tragante V, Sousa TA, Silva VAO, Zanon NM, Kettelhut IC, De Lucca FL FMRP-USP – Biochemistry and Immunology

01.035
Evidence of a regulatory role of dystrophin on the release of acetylcholine (ACh) in the mouse brain. Della Colleta E, Nogueira FM, Campos DV, Lima-Landman MTR, Lapa AJ, Souccar C UNIFESP − Farmacologia

01.036
Facilitatory action of a quaternary derivate of l-hyoscyamine on acetylcholine (ACh) release in rat cortex synaptosomes. Analysis of the mechanisms involved. Nogueira FM, Della Colleta E, Lima-Landman MTR, Lapa AJ, Souccar C UNIFESP/EPM − Farmacologia

01.037
Treatment effect of acute *in vivo* ethanol on adrenergic neurotransmission in the smooth muscle of periadolescent rats vas deferens. Silva Junior ED, Jurkiewicz A, Jurkiewicz NH UNIFESP − Farmacologia

01.038
Characterization of signaling pathways of Angiotensin I-converting enzyme in mesangial cells of spontaneously hypertensive rats (SHR). Reis RI[1], Parreiras-e-Silva LT[2], Becari C[3], De Andrade MCC[4], Salgado MCO[3], Costa-Neto CM[2], Casarini DE[5] [1]UNIFESP − Rim e Hormônios, [2]FMRP-USP − Bioquímica e Imunologia, [3]FMRP–USP − Farmacologia, [4]UNIFESP − Nefrologia, [5]UNIFESP − Medicina

01.039
Estrogen attenuates cellular death induced by H_2O_2 in C6 cells: a role for ESR and GPER receptors. Franco LAM, Yshii LM, Lopes DCF, Sá Lima L, Scavone C, Munhoz CD ICB-USP − Farmacologia

01.040
P2Y1 receptors stimulation on rat pineal glands: effects on nuclear factor kappa B pathway (NFKB) and inducible nitric oxide synthase (iNOS). Petrilli CL, Carvalho-Sousa CE, Muxel SM, Markus RP, Ferreira ZS IB-USP − Fisiologia

02. Neuropharmacology

2.045
Imipramine facilitates adaptation to chronic stress in animals with lesions of serotonergic neurons of the median raphe nucleus. Silva K, Padovan D, Padovan CM FFCLRP-USP − Psicologia e Educação

02.046
Allopregnanolone antidepressive and stress activity evaluation after nucleus *accumbens* administration in rats. Ferri MK[1], Dalpra WL[1], Azeredo LA[1], Couto-Pereira N[2], Nin MS[1], Gomez R[1], Barros HMT[1] [1]UFCSPA − Farmacologia, [2]UFCSPA − Ciências Fisiológicas

02.047
The expression of mRNAs encoding the flip isoforms of GLuR1 and GLuR2 are decreased in hippocampus of rats reared in isolation from weaning. Trindade LB[1], Sestito RS[1], Kerbauy LN[1], de Souza RG[1], Limonte FH[1], Iyomasa MM[2], Rosa ML[2] [1]FAMECA − Bioquímica, [2]FAMECA-FIPA − Neurociências

02.048
Neuroprotective effect of propofol in model of hippocampal ischemia in rats. Binda NS[1], Pessoa FLC[2], Pinheiro ACN[1], Silva JF[3], Lavor MSL[4], Gomez RS[6], Gomez MV[3] [1]UFMG − Farmacologia, [2]UFMG − Medicina, [3]UFMG − Farmacologia Bioquímica e Molecular, [4]UFMG− Clínica e Cirurgia Veterinária, [5]UFMG − Cirurgia

02.049
Glial cells are important in protecting neurons after ischemia induced by glucose deprivation. Lopes DCF, Matsubara CS, Franco LAM, Sá Lima L, Scavone C, Munhoz CD ICB-USP − Farmacologia

02.050
Interaction between 5-HT_{1A} receptor and a nitric oxide donor, sin-1, ON locomotor activity of rats. Gualda LBS[1], Martins GG[2], Guimarães FS[3], Oliveira RMMW[1] [1]UEL − Farmácia e Farmacologia, [2]UEM − Farmácia e Farmacologia, [3]FMRP-USP

02.051
Glutamatergic neurotransmission within the hypothalamic paraventricular nucleus is involved in the cardiovascular response evoked by noradrenaline microinjected into the dorsal periaqueductal gray area of rats. Pelosi GG[1], Busnardo C[2], Tavares RF[2], Corrêa FMA[2] [1]UEL − Farmacologia, [2]FMRP-USP − Farmacologia

02.052
Relationship of long-term memory evocation and cholinergic markers in hippocampus, along the aging process of rats. Oliveira EM[1], Souza LHJ[1], Schowe NM[1], Albuquerque MS[1], Baraldi T[1], Chambergo FS[1], Pina dos Santos VP[2], Araújo MS[2], Buck HS[3], Viel TA[1] [1]EACH-USP, [2]UNIFESP − Bioquímica, [3]FCMSCSP − Ciências Fisiológicas

02.053
Effects of isolation rearing on the expression of B-amyloid precursor proteins, isoforms 695 and 751/770, in rat hippocampus. Kerbauy LN[1], de Souza RG[2], Trindade LB[1], Sestito RS[1], Limonte FH[2], Iyomasa MM[2], Rosa ML[2] [1]FAMECA-FIPA − Bioquímica, [2]FAMECA−FIPA Neurociências

02.054
Influence of glucocorticoids in the increase of CRF_2 mRNA levels in the lateral septal nucleus of rats submitted to chronic unpredictable stress. Malta MB[1], Sita LV[2], Silva JM[2], Bittencourt JC[2], Scavone C[1], Munhoz CD[1] [1]ICB-USP − Farmacologia, [2]ICB-USP − Anatomia

02.055
Overactive bladder induced by spinal cord injury: implication of TRPA1 receptor. Andrade EL[1], Forner S[1], Bento AF[1], Leite DFP[1], Dias MA[2], Leal PC[3], Koepp J[4], Calixto JB[1] [1]UFSC − Farmacologia, [2]ISCAL − Neurocirurgia, [3]UFSC − Química, [4]UFSC − Engenharia Química

02.056
Neonatal morphine exposure alters E-NTPDase activity and gene expression pattern in spinal cord and cerebral cortex of rats. Rozisky JR[1], Silva, RS[2], Adachi LNS[1], Bogo MR[2], Bonan CD[2], Torres ILS[1] [1]UFRGS − Farmacologia, [2]PUCRS − Biologia Celular e Molecular

02.057
Fish oil prevents orofacial dyskinesia, memory loss and lipid peroxidation induced by haloperidol in rats. Bürger ME[1], Barcelos RCS[2], Benvegnú DM[1], Müller LG[2], Reckziegel P[2], Pase C[2], Emanuelli T[3], Boufleur N[2] [1]UFSM − Farmacologia, [2]UFSM − Fisiologia e Farmacologia, [3]UFSM − Tecnologia dos Alimentos

03. Psychopharmacology

03.025
Nitric oxide modulates glutamatergic control of sensorimotor gating in rats. Henriques-Santos NF[1], Brosco MC[1], Del Bel EA[2], Salum C[1] [1]UFABC − Matemática, Computação e Cognição, [2]FORP-USP − Morfologia, Estomatologia e Fisiologia

03.026
Effects of nNOS Inhibition on forced swimming-induced FOS expression in the rat brain. Silva M, Aguiar DC, Guimarães FS, Joca SRL FMRP-USP − Farmacologia

03.027
Antidepressant-like activity of *Hypericum caprifoliatum* Cham. & Schltdl (Guttifereae) is not due to NMDA receptor activation neither to neuronal glutamate transport. Centurião FB[1], Stein AC[1], Gay BM[2], Prigol M[2], Viana AF[3], Nogueira CW[2], Rates SMK[3] [1]UFRGS − Farmácia, [2]UFSM − Química, [3]UFRGS − Ciências Fisiológicas

03.028
Repeated morphine administration in early life promotes anxiolytic effect on elevated plus-maze. Nonose Y[1], Rozisky JR[1], Santos VS[1], Medeiros LF[1], Souza A[2], Caumo W[3], Torres ILS[1] [1]UFRGS − Farmacologia, [2]UFRGS − Bioquímica, [3]UFRGS − Anestesia

03.029
Evaluation of intestinal motility tolerance after repeated diethylpropion administration in rats. Dalpra WL[1], Caletti, G[1], Olguins, DB[2], Barros HMT[1], Gomez, R[1] [1]UFCSPA − Farmacologia, [2]IPA − Farmacologia

03.030
Blockade of glutamate NMDA receptors in the rat ventromedial prefrontal cortex (vMPFC) induces antidepressant-like effects. Pereira VS[1], Joca SRL[2] [1]FMRP-USP − Farmacologia, [2]FCFRP-USP − Física e Química

03.031
Ayahuasca repeated treatment inhibits behavioral sensitization previously developed to ethanol in mice. Marinho EAV[1], Gerardi-Junior CA[2], Santos R[3], Baldaia MA[4], Oliveira-Lima AJ[5], Wuo-Silva R[6], Hollais AW[7], Malpezzi-Marinho ELA[2], Fernandes HA[8], Frussa-Filho R[5] [1]UBC/UNIFESP − Ciências da Saúde / Farmacologia, [2]UBC − Ciências da Saúde, [3]UBC − Farmácia, [4]UBC/UNIFESP − Farmacologia, [5]UNIFESP − Farmacologia, [6]UBC − Fisiologia

03.032
Acute effect of *Dioclea violacea* M. (aqueous extract) on general activity observed in the rats on elevated plusmaze and its dyskinetics movements. Gemignani S, Silva FO, Biscaro MDA, Santos NSS, Rassam E, Gonçalves RB, Pedroso-Mariani SR FMJ − Farmacologia

03.033
Role of L-arginine-nitric oxide pathway and possible implications for cardiovascular disease in depressed patients. Pinto VLM[1], Fontoura PCS[1], Brunini T[2], Mendes Ribeiro AC[1] [1]UERJ − Farmacologia e Psicobiologia, [2]UERJ − Farmacologia

03.034
Chronic imipramine treatment enhances the panicolytic-like effect caused by the stimulation of 5-HT_{1A} and 5-HT_{2A} receptors in the dorsomedial hypothalamic nucleus. de Bortoli VC, Zangrossi Jr H FMRP-USP − Farmacologia

03.035
Bipolar disorder and cardiovascular disease. Fontoura PCS[1], Pinto VLM[1], Cheniaux Jr E[2], da Silva O[1], Brunini T[3], Mendes Ribeiro AC[1] [1]UERJ − Farmacologia e Psicobiologia, [2]IPUB-UFRJ − Psiquiatria, [3]UERJ − Farmacologia

03.036
Memory impairment is associated with inflammatory changes in the hippocampus of DENV-3 infected mice. Campos RDL[1], Amaral DCG[2], Cisalpino D[3], Vilela MC[4], Rodrigues DH[5], Miranda AS[1], Lacerda-Queiroz N[5], Souza KPR[3], Kroon EG[3], Reis HJ[6], Teixeira, AL[2] [1]UFMG − Bioquímica e Imunologia, [2]UFMG − Clínica Médica, [3]UFMG − Microbiologia, [4]UFMG − Neurociências, [5]UFMG − Biologia Celular, [6]ICB-UFMG − Farmacologia

04. Inflammation

04.093
Pineal gland is instrumented to be an integral player of innate immune response. Carvalho-Sousa CE[1], Cruz-Machado SS[1], Fernandes PACM[1], Tamura EK[1], Pinato, L[2], Petrilli CL[1], Markus RP[1] [1]IB-USP − Fisiologia, [2]UNESP − Fonoaudiologia

04.094
The intestinal ischemia/reperfusion in rats promotes changes in the lung depending on the time of reperfusion. Vitoretti LB, Breithaupt-Faloppa AC, Gimenes-Junior JA, Domingos HV, Sudo-Hayashi LS, Oliveira-Filho RM, Tavares de Lima W ICB-USP − Pharmacology

04.095
Saccharomyces boulardii prevents the inflammatory response in intestinal mucositis induced by 5-fluorouracil in mice. Justino PFC[1], Silva LMN[1], Melo LFM[1], Costa JVG[1], Nogueira AF[1], Ribeiro RA[1], Souza MHLP[1], Soares PMG[2] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Morfologia

04.096
Effect of protease-activated receptor-2 activating peptide on B1 cell spreading and its modulation by the C-terminus of the calcium binding protein S100A9. Moraes NF[1], Sampaio SC[1], Freitas JD[1], Pagano RL[2], Giorgi R[1] [1]IBu − Fisiopatologia, [2]IEP − Neuromodulação e Dor Experimental

04.097
Effects of the treatment with an inhibitor of CCL2 synthesis, in acute diet-induced adiposity in mice. Lima RL[1], Menezes Z[1], Santos MCC[1], Guglielmotti A[2], Teixeira MM[3], Ferreira AVM[4], Souza DG[1] [1]UFMG − Microbiologia, [2]ACRAF − Pharmacology, [3]UFMG − Imunofarmacologia, [4]UFMG − Enfermagem Básica

04.098
Hydrogen sulfide and antioxidant enzyme activities in allergic mice lungs. Campos D[1], Benetti LR[1], Nogueira JS[1], Gurgueira SA[2], Vercesi AE[3], Ferreira HHA[1] [1]USF − Inflamação, [2]FCM-UNICAMP − Bioenergética, [3]FCM-UNICAMP − Patologia Clínica

04.099
Expression of adhesion molecules in vessels of the microcirculation affected by different metalloproteases isolated from *Bothrops* venoms. Zychar BC[1], Baldo C[2], Clissa, PB[2], Alves AS[3], Britto LRG[3] [1]IBu − Fisiopatologia, [2]IBu − Imunopatologia, [3]USP − Fisiologia e Biofísica

04.100
Effects of the cystein proteinase obtained from *C. candamarcensis* P1G10-treatment on eosinophil recruitment in allergic mice pleurisy. Miwa MY[1], Lopes MTP[1], Ferreira RG[2], Gomides LF[1], Salas CE[3], Klein A[2] [1]UFMG − Farmacologia, [2]UFMG − Fisiologia/Farmacologia, [3]UFMG − Bioquímica e Imunologia

04.101
Aprotinin potentiates carrageneen edema formation: evidences for prostaglandin participation. Ferreira RG[1], Godin AM[2], Matsui TC[3], Coelho MM[4], Klein A[3] [1]UFMG − Fisiologia/Farmacologia, [2]FF-UFMG − Produtos Farmacêuticos, [3]UFMG − Farmacologia, [4]UFMG

04.102
Methotrexate effects on systemic and adipose tissue alterations induced by obesity in mice. De Oliveira CC, Acedo SC, Gotardo EMF, Gambero A USF − Farmacologia e Gastroenterologia

04.103
Crohn´s experimental model decreased the mechanical inflammatory hypernoception in rats- role of NO/cGMP/ KATP pathway. Barbosa ALR[1], Sousa RB[2], Torres JNL[2], Lucetti LT[2], Cunha, TM[3], Cunha FQ[3], Ribeiro RA[2], Vale ML[2], Souza MHLP[2] [1]UFPI − Fisiologia e Farmacologia / UFC, [2]UFC − Fisiologia e Farmacologia, [3]FMRP-USP

04.104
Effects of hydrogen sulfide on leukocyte migration and protein tyrosine nitration in airways of allergic mice. Benetti LR[1], Teixeira SA[2], Campos D[3], Silva AA[3], Costa SKP[2], Muscará MN[2], Ferreira HHA[3] [1]USF − Farmacologia, [2]USP − Farmacologia, [3]USF − Inflamação

04.105
Characterization of allergic lung inflammation in genetically obese mice. Lintomen L, Calixto MC, Schenka A, Antunes E UNICAMP − Farmacologia

04.106
Mycobacterium bovis BCG infection activates a rapamycin-sensitive mTOR pathway: involvement in the lipid body formation and inflammatory response. D´Ávila H[1], Lage SL[2], Roque NR[3], Maya-Monteiro CM[3], Almeida PE[3], Melo RCN[4], Castro-Faria-Neto HC[3], Bozza PT[3] [1]UFJF − Biologia Celular, [2]USP − Imunologia, [3]FIOCRUZ − Imunofarmacologia, [4]FIOCRUZ − Biologia Celular

04.107
Contribution of reactive-oxygen species to the enhancement of platelet aggregation in high-fat fed rats. Monteiro PF[1], Prada Morganti R[1], Delbin MA[2], Pires MEL[1], Priviero FBM[1], Marcondes S[1], Zanesco A[2], Antunes E[1] [1]UNICAMP − Farmacologia, [2]UNESP − Educação Física

04.108
Staphylococcal enterotoxin A (SEA) inhibits human eosinophil migration *in vitro*. Mello GC, Squebola Cola DM, Souza IA, Antunes E FCM-UNICAMP − Farmacologia

04.109
Effect of a combination of medium chain triglycerides, linoleic acid, soy lecithin and vitamins A and E on wound healing in rats. Magalhães MS[1], Moraes MEA[1], Fechine FV[1], Nascimento DF[1], Macedo RN[1], Monteiro DLS[2], Oliveira CC[1], Linhares JH[3], Leite ALAS[1], Moraes MO[1] [1]UFC − Fisiologia e Farmacologia, [2]FAMED-UFC − Fisiologia e Farmacologia, [3]UFC − Cirurgia

04.110
Leukotriene B4 mediates γδ T lymphocyte migration in response to diverse stimuli. Souza-Martins R[1], Costa MFS[1], Souza M[1], Piva B[1], Diaz BL[3], Peters-Golden M[4], Henriques MGMO[1], Canetti C[1], Penido C[1] [1]Farmanguinhos-FIOCRUZ − Farmacologia Aplicada, [2]IBCCF-UFRJ, [3]IBCCF-UFRJ − Imunobiologia, [4]University of Michigan − Pulmonary and Critical Care Medicine

04.111
Adhesion molecules and chemokines receptor expression in bone marrow eosinophils of obese mice. Calixto MC[1], Lintomen L[1], Thomé R[2], Tamashiro WMSC[2], Antunes E[2] [1]UNICAMP − Farmacologia, [2]UNICAMP − Imunologia e Microbiologia

04.112
Antinociceptive and anti-inflammatory effect of heme oxygenase-1 / biliverdin / carbon monoxide pathways in temporomandibular joint arthritis induced by zymosan. Chaves HV[1], Filgueira AA[2], Ribeiro KA[3], Silva AAR[2], Souza MHLP[4], Ribeiro RA[4], Bezerra MM[5], Brito GAC[6] [1]UFC − Ciências Médicas, [2]UFC-Sobral − Odontologia, [3]UVA − Biologia, [4]UFC − Fisiologia e Farmacologia, [5]UFC-Sobral − Medicina, [6]UFC − Morfologia

04.113
Antipruritic activity of the ethanol extract from *Lecythis pisonis* Camb. (Lecythidaceae) leaves in mice. Silva LL[1], Gomes BS[1], Silva AMS[1], Oliveira JPC[2], Chaves MH[2], Oliveira FA[1] [1]UFPI − NPPM, [2]UFPI − Química

04.114
Effects of acute treatment with 1-(3-chlorophenyl) piperazine (mCPP) in the leukocyte traffic in rats. Lombardi L, Hebeda CB, Farsky S, Moreau RLM USP − Análises Clínicas e Toxicológicas

04.115
Lipoxin A4 plays a protective role in experimental dengue disease. Cisalpino D[1], Fagundes CT[2], Costa VV[2], Guabiraba R[2], Souza DG[1], Teixeira MM[2] [1]UFMG − Microbiologia, [2]UFMG − Bioquímica e Imunologia

04.116
Lipopolysaccharide *in vivo* increases the production of reactive oxygen species in rat platelet mainly through activation of NADPH-oxidase system. Pires MEL, Cardelli NJA, Anhê GF, Antunes E, Marcondes S UNICAMP − Farmacologia

04.117
Synthesis and evaluation of activity of 4-antihipernociceptiva aminochalconas. Sonza DR, Buzzi FC, Rodrigues C, Corrêa R, Souza PS, Quintão NLM [1]NIQFAR-UNIVALI − Farmacêuticas

04.118
Mechanisms of the inflammatory response induced by topical application of cinnamaldehyde in mice. Silva CR[1], Oliveira SM[1], Rossato MF[1], Guerra GP[1], Dalmolin GD[2], Prudente AS[3], Cabrini DA[3], Otuki MF[3], Ferreira J[1] [1]UFSM − Química, [2]UFMG − Farmacologia, [3]UFPR − Farmacologia

04.119
IFN-g limits antigen-induced arthritis severity by inducing IDO/GCN2 kinase pathway. Lemos HP[1], Mellor AL[2], Chandler PR[3], Vieira SM[4], Grespan R[5], Cunha FQ[1] [1]FMRP-USP − Farmacologia, [2]Medical College of Georgia − Molecular Medicine and Genetics, [3]Medical College of Georgia − Immunotherapy, [4]COPE-INPA, [5]UEM − Farmácia e Farmacologia

04.120
IL-1 receptor antagonist (IL-1RA) Prevents hemorrhage, inflammation, nociception and bladder dysfunction in ifosfamide-induced hemorrhagic cystitis. Leite CAVG[1], Alencar VTL[1], Lima-Júnior RCP[1], Mourão LTC[1], Wong DVT[1], Melo DLR[1], Magalhães PJC[1], Santos AA[1], Brito GAC[2], Cunha FQ[3], Ribeiro RA[1] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Morfologia, [3]FMRP-USP

04.121
Involvement of nitric oxide on the pathogenesis of irinotecan-induced intestinal mucositis: role of cytokines on inducible nitric oxide synthase activation. Leite CAVG[1], Lima-Júnior RCP[1], Wong DVT[1], Oriá RB[2], Brito GAC[2], Souza MHLP[1], Cunha FQ[3], Ribeiro RA[1] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Morfologia, [3]FMRP-USP

04.122
IL-6/ IL-23/ IL-17/ IL-22 axis mediates the inflammatory response in antigen-induced arthritis in mice. Pinto LG[1], Talbot J[1], Vieira SM[2], Verri Jr WA[3], Cunha, TM[1], Cunha FQ[1], Ferreira SH[1] [1]FMRP-USP − Farmacologia, [2]COPE-INPA, [3]UEL − Pathology and Pharmacology

04.123
Nitric oxide, carbon moxide and guanylate cyclase modulate remote ischemic preconditioning: participation of adhesion molecules in inhibition of neutrophils migration. Simão AFL[1], Souza-Filho MVP[2], Souto FO[3], Simão AAL[4], Cunha FQ[5], Ribeiro RA[5] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Cirurgia, [3]FMRP-USP − Surgery and Anatomy, [4]UFC − Farmacologia, [5]FMRP-USP

04.124
Lipid droplet in pulmonary dendritic cells during severe sepsis. Molinaro RC[1], Vieira-de-Abreu A[1], Silva AR[1], Castro-Faria-Neto HC[1], Benjamim CF[2], Bozza PT[1] [1]DFF-FIOCRUZ, [2]UFRJ − Farmacologia Básica e Clínica

04.125
Intraperitonial injection of *S. aureus* induces fever accompanied by an increase of prostaglandin E2 (PGE2) in the CSF and hypothalamus in rats. Martins JM[1], Soares DM[2], Malvar DC[1], Figueiredo MJ[1], Souza GEP[2] [1]FMRP-USP − Farmacologia, [2]FCFRP-USP − Física e Química

04.126
Reduction of the expression of membrane CXCR2 and BLT1 receptors on neutrophils related to increased mortality of septic patients in emergency department. Sousa RB[1], Souto FO[2], Spiller F[2], Turato W[3], Lobo RR[1], Mendonça PR[1], Cunha FQ[2], Pazin A[1] [1]FMRP-USP − Clínica Médica, [2]FMRP-USP − Pharmacology, [3]FCFRP-USP − Análises Clínicas, Toxicológicas e Bromatológicas

04.127
JM 25-1, a non anesthetic lidocaine derivative, decreased the expression of transcription factor GATA-3 in a murine model of acute allergic inflammation. Couto GC[1], Serra MF[1], Cotias AC[1], Anjos-Valotta EA[2], Jurgilas PB[1], Ferreira TPT[1], Costa JCS[1], Pires ALA[1], Cordeiro RSB[1], Silva PMR[1], Martins MA[1] [1]IOC-FIOCRUZ − Fisiologia e Farmacodinâmica, [2]ICB-USP

04.128
Lidocaine inhibits airway inflammation, peribronchial fibrosis and mucus production in a murine model of asthma. Serra MF[1], Cotias AC[1], Anjos-Valotta EA[2], Couto GC[1], Pão CRR[1], Jurgilas PB[1], Ferreira TPT[1], Pires ALA[1], Arantes ACS[1], Cordeiro RSB[1], Silva PMR[1], Martins MA[1] FIOCRUZ − Fisiologia e Farmacodinâmica, [2]ICB-USP

04.129
Role of arylhidrocarbon Receptor (AhR) in antigen-induced arthritis. Talbot J[1], Pinto LG[1], Alves-Filho JC[2], Vieira SM[3], Ferreira SH[1], Cunha TM[1], Louzada Jr P[4], Cunha FQ[1] [1]FMRP-USP − Farmacologia, [2]University of Glasgow − Immunology, Infection and Inflammation, [3]COPE-INPA, [4]FMRP-USP − Clínica Médica

04.130
Chemokine decoy receptor D6 deficiency protects against bleomycin-induced pulmonary inflammation and fibrosis in mice. Russo RC[1], Savino B[1], Mirolo M[1], Buracchi C[2], Anselmo A[2], Zammataro L[2], Pasqualini F[2], Germano G[2], Nebuloni M[3], Mantovani A[3], Teixeira MM[1], Locati M[2] [1]UFMG − Bioquímica e Imunologia, [2]Istituto Clinico Humanitas, Leukocyte Biology, [3]University of Milan − Pathology

04.131
Early-lifetime exposure to 1,2-naphthoquinone (1,2-NQ) interact to increase asthma susceptibility and behavior changes in juvenile mice. Florenzano J, Santos KT, Teixeira SA,

Barreto MAA, Muscará MN, Camarini R, Costa SKP ICB-USP − Farmacologia

04.132

Pharmacological blockade of the CXCL-ELR+ chemokine / receptor cxcr2 axis accelerates wound closure in mice. Castro TBR[1], Canesso MCC[1], Almeida BG[1], Colotta F[2], Bertini R[2], Proudfoot AEI[3], Andrade SP[1], Teixeira MM[4], Barcelos L[1] [1]ICB-UFMG − Physiology and Biophysics, [2]Dompé Research and Development, [3]Serono Pharmaceutical Research Institute, [4]ICB-UFMG − Biochemistry and Immunology

04.133

Absence of $P2X_7$ purinergic receptors in the hemorrhagic cystitis induced by cyclophosphamide in mice. Martins JP[1], Silva RBM[2], Santos Jr AA[3], Coutinho R[4], Battastini AMO[5], Santos DS[6], Morrone FB[6], Campos MM[7] [1]PUCRS − Medicina, [2]PUCRS − Farmacologia Aplicada, [3]INCTb-PUCRS − Biologia Molecular e Funcional, [4]IBCCF-UFRJ, [5]UFRGS − Bioquímica, [6]PUCRS − Farmácia, [7]PUCRS − Cirurgia-Odontologia

04.134

Characterization of the inflammatory process in epididymal fat tissues in mice submitted to a palatable diet. Bernardes PTT[1], Rezende B[2], Castor MGM[3], Ferreira AVM[4], Teixeira MM[5], Pinho V[5] [1]UFMG − Morfologia e Bioquímica, [2]UFMG − Bioquímica e Imunologia e Morfologia, [3]UFMG − Fisiologia e Farmacologia, [4]UFMG − Fisiologia e Biofísica, [5]UFMG − Bioquímica e Imunologia

04.135

In vitro-differentiated mouse eosinophils as a new tool for the study of eosinophil biology. Samico RF[1], Luna-Gomes T[1], Mesquita-Santos FP[2], Bakker-Abreu I[1], Diaz BL[3], Bandeira-Melo C[2] [1]IBCCF-UFRJ, [2]FIOCRUZ − Fisiologia e Farmacodinâmica, [3]IBCCF-UFRJ − Imunobiologia

04.136

Behavioral changes are associated with central nervous system inflammation in an experimental model of malaria. Miranda AS[1], Lacerda-Queiroz N[2], Vilela MC[3], Rodrigues DH[2], Reis HJ[4], Teixeira MM[1], Rachid M[6], Teixeira, AL[7] [1]UFMG − Bioquímica e Imunologia, [2]UFMG − Biologia Celular, [3]UFMG − Neurociências, [4]UFMG − Farmacologia, [6]UFMG − Patologia, [7]UFMG − Clínica Médica

04.137

Hydrogen Peroxide is associated to neutrophil clearance in model of arthritis induced in mice. Lopes F[1], Sousa LP[2], Coelho FM[3], Costa VV[3], Gonçalves W[1], Teixeira MM[3], Pinho V[3] [1]UFMG − Morfologia, [2]UFMG − Patologia Clínica e Bioquímica e Imunologia, [3]UFMG − Bioquímica e Imunologia

04.138

Role of transient receptor potential vanilloid 4 (TRPV4) in joint inflammation. Denadai-Souza A[1], Vergnolle N[2], Martin L[2], Muscará MN[1], Cenac N[2] [1]ICB-USP − Pharmacology, [2]INSERM

05. Pain and Nociception

05.053

Evaluation of the antinociceptive profile of a series of *N*-acylhydrazones derivatives modified from the prototype LASSBio-294. Veloso RR, Nogueira MCO, Maia RC, Lima ML, Barreiro EJ, Miranda ALP FF-UFRJ- LASSBio

05.054

Effects of methysergide injected into the anterior pretectal nucleus on the stimulation-induced antinociception from the retrosplenial cortex. Rossaneis AC, Reis GM, Prado WA FMRP USP − Farmacologia

05.055

Crotalphine induces a long-lasting and opioid-mediated antinociceptive effect in an experimental model of bone cancer pain. Gutierrez VP[2], Brigatte P[1], Zambelli VO[2], Carvalho JS[2], Picolo G[2], Radin A[3], Marques FLN[3], Cury Y[2] [1]CEIS-UNESP, [2]IBu − Pain and Signaling, [3]FM-USP − Oncology

05.056

Aspirin-triggered resolvin d1, AT-RVD1, and its precursor, possess anti-hyperalgesic properties in a model of arthritis in rats. Lima-Garcia JF[1], Motta EM[1], Campos MM[2], Calixto JB[1] [1]UFSC − Farmacologia, [2]PUCRS − Cirurgia-Odontologia

05.057

Endothelin-1 induces both itch and pain in the mouse cheek. Oliveira L, Hara DB, Rae GA UFSC − Farmacologia

05.058

The involvement of the transient receptor potential A1 (TRPA1) on norepinephrine-induced nociception in neuropathic mice. Pinheiro FV[1], Silva CR[2], Oliveira SM[2], Villarinho JG[3], Andre E[3], Ferreira J[2] [1]UFSM − Fisiologia e Farmacologia, [2]UFSM − Química, [3]UFRN − Biofisica e Farmacologia

05.059

Peripheral antinociceptive effect of inosine depends of the A1 adenosine receptor but not of receptors A2A and A3. Cerutti ML[1], Nascimento FP[2], Macedo Junior SJ[1], Santos ARS[1] [1]UFSC − Ciências Fisiológicas, [2]UFSC − Farmacologia

05.060

Analgesic effects of ethanolic extract from *Sinningia aggregata* tubers and from the isolated compound 3-prenyl-4-oxo-3´-methylnaphtho [1,2b] oxepin-1,3´(4H)-diol. Simas AS[1], Verdan ML[2], Kassuya CAL[3], Stefanello MEA[2], Zampronio AR[1] [1]UFPR − Farmacologia, [2]UFPR − Química, [3]UFGD − Ciências da Saúde

05.061
Cytokines and chemokines participate in the genesis of post-incisional pain. Carreira EU, Cunha FQ, Ferreira SH, Cunha TM FMRP-USP − Farmacologia

05.062
Effect of Oligopeptidases B from *Trypanosoma cruzi and Trypanosoma brucei* in an experimental model of pain in mice. Abrahão RQ[1], Juliano MA[2], Juliano L[2], Giorgi R[3], Dale CS[4] [1]UNIFESP − Biofísica, [2]UNIFESP − Farmacologia, [3]IBu − Fisiopatologia, [4]IEP-HSL

05.063
Central antinociceptive effects of ethanolic extract from the bark of *Pithecellobium cochliocarpum* on mice. Souza IA[1], Jesus RPFS[2], Bastos IVGA[2], Rodrigues GCR[2] [1]UFPE − Antibióticos

05.064
Role of NMDA receptors in carrageenin-induced hypernociception in rat temporomandibular joint: magnesium chloride modulator effect. Cavalcante ALC[1], Siqueira RMP[2], Colares MT[1], Chaves HV[1], Vale ML[2] [1]UFC − Ciências Médicas, [2]UFC − Fisiologia e Farmacologia

05.065
Involvement of B1 and B2 kinin receptors in painful neuropathy induced by paclitaxel in mice. Tamiozzo LLR[1], Dalmolin GD[2], Rigo FK[2], Ferreira J[1] [1]UFSM − Química, [2]UFMG − Farmacologia

05.066
Mechanism of peripheral antinociceptive effect of 15D-PGJ_2 on rheumatoid arthritis into the TMJ of rats. Quinteiro MS[1], Napimoga MH[2], Clemente-Napimoga JT[1] [1]UNIUBE − Biologia Molecular, [2]UNIUBE − Biologia Celular e Molecular

05.067
Involvement of kinin receptors in pronociceptive action of dynorphin A (1-17) in a mouse model of orofacial neuropathy. Schroeder SD[1], Luiz AP[2], Chichorro JG[3], Rae GA[1] [1]UFSC − Farmacologia, [2]UFSC − Ciências Fisiológicas, [3]UFPR − Farmacologia

05.068
Analgesic effect of a novel allosteric antagonist of C5a receptors. Carneiro VL[1], Bertini R[2], Cunha FQ[1], Ferreira SH[1], Teixeira MM[5] [1]FMRP-USP − Farmacologia, [2]Dompé Research and Development

05.069
Hyponociceptive effect of H1 and H2 receptors in a model of articular inflammation induced by formalin. Souza-Silva E, Mascarin LZ, Eto C, Oliveira D, Tonussi CR UFSC − Farmacologia

05.070
Role of nitric oxide in nociception, edema and plasma leakage induced by intra-articular formalin. Eto C, Souza-Silva E, Mascarin LZ, Tonussi CR UFSC − Farmacologia

05.071
Cafestol evokes peripheral antinociception via activation of α_{2C} adrenoceptors. Guzzo LS, Perez AC, Duarte IDG UFMG − Farmacologia

05.072
Antinociceptive and anti-inflammatory activity of ethanol extract of *Polygala sabulosa* in mice. Borges FR, Pierosan L, Silva MD, Córdova MM, Santos ARS CFS-UFSC

05.073
Visceral antinociceptive effect of HC-030031, an antagonist of TRPA1 ion channel, is independent of inflammatory cells and nitric oxide. Pereira LMS[1], Sá LG[1], Wong DVT[1], Callado RB[1], Teixeira CCG[1], Bem AXC[1], Larsen GR[2], Lima-Júnior RCP[1], Ribeiro RA[1] [1]UFC − Fisiologia e Farmacologia, [2]Hydra Biosciences Biopharmaceutical Co − Biopharmacology

05.074
Synthesis and evaluation of antihyperalgesic activity of benzofuranone derived of xanthoxylin: hypothesis of the possible mechanism of action. Souza JP[1], Quintão NLM[1], Sonza DR[2], De Campos-Buzzi F[2], Niero R[2] [1]UNIVALI − Ciências Farmacêuticas, [2]NIQFAR-CCS-UNIVALI

05.075
Evaluation of antinociceptive spinal activity of TX3-4, a toxin peptide purified from the spider *Phoneutria nigriventer* venom, in mice. Silva JF[1], Fontanini CEM[2], Lavor, MSL[3], de Souza AH[4], Pessoa FLC[2], Pinheiro ACN[4], Binda NS[4], Ferreira J[5], Gomez MV[4] [1]UFMG − Farmacologia Bioquímica e Molecular, [2]UFMG − Medicina, [3]UFMG − Clínica e Cirúrgica Veterinária, [4]UFMG − Farmacologia, [5]UFSM − Química

05.076
Is there an involvement of endocannabinoid system in the peripheral antinociception of NSAID? Resende LC, Duarte IDG UFMG − Farmacologia

05.077
Antinociceptive effect of Riparin II in mice. Carvalho AMR[1], Leite CP[1], Moura BA[3], Vasconcelos LF[1], Melo TV[1], Bastos MVR[1], Barbosa Filho JM[2], Sousa FCF[2] [1]UFC − Fisiologia e Farmacologia, [2]UFPB − Tecnologia Farmacêutica

05.078
Crotalphine reduces peripheral sensitization evoked by activation of TRPV1 receptor in mice. Motta EM[1], Machado FC[2], Gutierrez VP[2], Lira[2], Gandra[2], Picolo G[3], Cury Y[3] [1]UFSC − Dor e Sinalização, [2]IBu − Dor e Sinalização, [3]IBu − Laboratório de Fisiopatologia

05.079
Evidence for the Involvement of kinin B_1 and B_2 receptors in the neuropathic pain-like behavior after treatment with paclitaxel in mice. Motta EM[1], Costa R[2], Manjavachi MN[3], Dutra RC[2], Pesquero JB[3], Calixto JB[2] [1]UFSC − Dor e Sinalização, [2]UFSC − Farmacologia, [3]UNIFESP − Biofísica

06. Cardiovascular and Renal Pharmacology

06.053
Study of the cytotoxic effect of *Bothrops pauloensis* on MDCK cells. Jorge RJB[1], Marinho AD[2], Barbosa JPC[3], Abreu ML[1], Morais ICO[1], Menezes RRPPB[4], Lima Filho CF[5], Pessoa AWP[6], Santos LFL[6], Alves CD[3], Toyama MH[7], Martins AMC[4], Evangelista JSAM[6], Monteiro HSA[1], Moraes GB[6] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Farmácia, [3]UNIFOR − Fisiologia e Farmacologia, [4]UFC − Análises Clínicas e Toxicológicas, [5]UECE − Ciências Fisiológicas, [6]UECE − Veterinária, [7]IB-UNICAMP

06.054
Important changes in hematologic and cardiovascular parameters produced by chronic treatment with etoricoxib in normotensive and spontaneously hypertensive rats. Baracho NCV FMIT − Farmacologia e Bioquímica

06.055
Hypotensive and diuretic effect of *Achillea millefolium* L. (Asteraceae) in rats. de Souza P[1], Gasparotto Junior A[2], Boffo MA[3], Lourenço EL[4], Stefanello MEA[5], Marques MCA[1], da Silva-Santos JE[6], Kassuya CAL[7] [1]UFPR − Farmacologia, [2]UNIPAR/UFPR − Farmacologia, [3]UNIPAR − Farmacologia, [4]USP − Toxicologia e Análises Toxicológicas., [5]UFPR − Química, [6]UFPA − Farmacologia Experimental e Pré-clínica, [7]UFGD − Ciências da Saúde

06.056
Involvement of L-arginine/NO pathway on the vascular adaptive response of high fat-diet rats exposed or not to chronic stress. Bruder-Nascimento T[7], Campos DHJ[2], Cicogna AC[2], Cordellini S[1] [1]UNESP − Farmacologia, [2]UNESP − Clínica Médica

06.057
Comparative study of effects hemodynamic of diabetic cardiomyopathy is induced by L-name in rats. Gazzoto AF[1], Pereira DJ[1], Pires NF[1], Moreira MM[2], Santos RC[1], Figueiredo VN[2], Renno AL[3], Quinaglia TSS[2], Ludovico ND[2], Moreno Junior H[4] [1]UNICAMP − Farmacologia, [2]UNICAMP − Farmacologia cardiovascular, [3]UNICAMP − Farmacologia Bioquímica, [4]UNICAMP

06.058
Effect of potassium-sparing diuretics on rat corpus cavernosum smooth muscle reactivity. Claudino MA[1], Silva FH[1], Franco-Penteado CF[2], Takeshi FI[1], Lopes AG[3], Antunes E[1], Nucci G[1] [1]UNICAMP − Pharmacology, [2]UNICAMP − Hemocentro, [3]IBCCF-UFRJ − Fisiologia Renal

06.059
Cytotoxic effect of *Bothrops pirajai* on MDCK cells. Marinho AD[1], Jorge RJB[2], Barbosa JPC[3], Abreu ML[3], Rocha VHP[1], Morais ICO[2], Menezes RRPPB[4], Morais GB[5], Sampaio AM[8], Alves RS[2], Jorge ARC[2], Ximenes RM[2], Toyama MH[6], Martins AMC[4], Evangelista JSAM[7], Monteiro HSA[2] [1]UFC − Farmácia, [2]UFC − Fisiologia e Farmacologia, [3]UNIFOR − Fisiologia e Farmacologia, [4]UFC − Análises Clínicas e Toxicológicas, [5]UECE − Faculdade de Veterinária, [6]UNICAMP − Instituto de Biologia

06.060
Inhibition of MMP decreases mortality and right ventricular damage caused by acute pulmonary embolism in rats. Cau SBA[1], Barato RC[1], Gerlach RF[2], Tanus-Santos JE[1] − [1]FMRP-USP − Farmacologia, [2]FORP-USP − Morfologia

06.061
Angiotensin (1-12) metabolism in cardiac perfusate of Wistar and spontaneously hypertensive rats. Becari C[1], Pereira HJV[2], Mesquita Jr O[1], Oliveira EB[1], Salgado MCO[1] [1]FMRP-USP − Pharmacology, [2]FMRP-USP − Biochemistry and Immunology

06.062
Impaired Rho-A/Rho-kinase pathway contributes to vascular dysfunction in endotoxemic mouse. Corrêa T, da Silva-Santos JE UFSC − Farmacologia

06.063
Protein Disulfide Isomerase regulation of NADPH oxidase activity in vascular smooth muscle cells: effects on Angiotensin II redox signaling in hypertension. Camargo LL[1], Androwiki ACD[1], Ceravolo GS[1], Denadai-Souza A[1], Muscará MN[1], Carvalho MHC[1], Fortes ZB[1], Janiszewski M[2], Lopes LR[1] [1]USP − Farmacologia, [2]Hospital Israelita Albert Einstein − IEP

06.064
Endothelium potentiates vasodilator effect of nitric oxide donor with gold nanoparticles in aortas from normotensive but not from renal hypertensive rats. Silva BR[1], Vercesi J. A[2], Moraes JB[2], Silva RS[2], Bendhack LM[2] [1]FMRP-USP − Farmacologia, [2]FCFRP-USP − Física e Química

06.065
Vascular response in acute lung injury induced by paraquat. Aires RD[1], Capettini LSA[1], Pinho JF[2], Côrtes SF[3], Pinho V[4], Lemos VS[1] [1]UFMG − Fisiologia e Biofísica, [2]UFMG − Fisiologia e Farmacologia, [3]UFMG − Farmacologia, [4]UFMG − Bioquímica e Imunologia

06.066
Expression of protein disulfide isomerase is associated with increased reactive oxygen

species generation in target organs in hypertension: possible interaction with NADPH oxidase. Androwiki ACD[1], Camargo LL[1], Dias AA[1], Janiszewski M[2], Lopes LR[1] [1]ICB-USP − Farmacologia, [2]Hospital Israelita Albert Einstein − IEP

06.067

Comparison of the action mechanisms of sodium nitroprusside and $[Ru(terpy)(bdq)NO^{+}]^{3+}$ in normotensive and hypertensive rats (SHR). Munhoz, FC[1], Pereira AC[2], Bonaventura D[2], Bendhack LM[2], Antoniali C[1] [1]FOA-UNESP − Ciências Básicas, [2]FCFRP-USP − Física e Química

06.068

Protein disulfide isomerase regulates reactive oxygen species generation and vascular reactivity to angiotensin II in rat aorta. Dias AA[1], Camargo LL[2], Ceravolo GS[1], Androwiki ACD[1], Carvalho, MHC[1], Laurindo FRM[2], Janiszewski M[3], Lopes LR' [1]ICB-USP − Farmacologia, [2]InCor-HC-FMUSP, [3]Hospital Israelita Albert Einstein − IEP

06.069

Pharmacological characterization of the contractile response of the dorsal penile vein Carioletti GH[1], Linder AE[2] [1]UFSC − Farmacologia, Centro de ciências Biológicas, [2]UFSC − Farmacologia

06.070

The contribution of endothelial factors to serotonin-induced contraction in the rat jugular vein. Costa EB, Linder AE [1]UFSC – Farmacologia

06.071

Hypothalamic obese and non-obese rats express a similar functional muscarinic M_3 subtype in the conductance artery. Scolaro LL[1], Oliveira JC[2], Ambiel CR[3], Mathias PC[2], Alves-do-Prado W[1] [1]UEM − Farmacologia, [2]UEM − Biologia Celular e Genética, [3]UEM − Ciências Fisiológicas

06.072

Phenylephrine relaxes rat jugular vein. De Prá MA, Linder AE UFSC − Farmacologia

06.073

Blood renin activity and vasoactive peptides concentrations in rats with different angiotensin I converting enzyme (ACE) phenotypes. Peixoto HS, da Silva RM, Tanae MM, Souccar C, Lapa AJ, Lima-Landman MTR UNIFESP − Farmacologia

06.074

Atorvastatin seems inhibit the MMP-9 expression more pronouncedly than sinvastatin in human endothelial cell culture. Izidoro-Toledo TC[1], Guimarães DA[1], Gerlach RF[2], Tanus-Santos JE[1] [1]FMRP-USP − Pharmacology, [2]FORP-USP − Morphology

06.075

Hydrogen sulfide improves vascular hyporesponsiveness and survival in severe polymicrobial sepsis in mice. Balsanelli LS, Dal-Secco D, Assreuy J UFSC − Farmacologia

06.076

Changes in rat's vascular reactivity in response to neonatal induced hyperleptinaemia. Motta NAV[1], Marques EB[1], Louback LS[2], Miranda ALP[2], Scaramello C[1], Brito FCF[1] − [1]UFF − Fisiologia e Farmacologia, [2]FF-UFRJ − LASSBio

06.077

Temporal evaluation of 2K1C model of renovascular hypertension: metalloproteinases and oxidative stress. Ceron CS[1], Rizzi E[1], Oliveira AM[3], Guimarães DA[1], Cau SBA[1], Marçal DMO[1], Gerlach RF[1], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]FORP-USP − Morfologia

06.078

Evaluation of cilostazol effects in hypercholesterolemic diet fed rats. Motta NAV[1], Canal PF[1], Amorim LEO[1], Reis RC[1], Miranda ALP[2], Brito FCF[1] − [1]UFF − Fisiologia e Farmacologia, [2]FF-UFRJ − LASSBio

06.079

Cardiovascular and neuroendocrine changes after blood volume expansion with hydroxyethyl starch 450/0.7 during experimental septic shock induced by cecal ligation and perforation (CLP). Santiago MB[1], Andrade CAF[1], Antunes-Rodrigues J[2], Giusti-Paiva A[1] [1]UNIFAL − Ciências Biológicas, [2]FMRP-USP − Fisiologia

06.080

Pharmacological characterization of a new steroidal cardiotonic isolated from *Physalis angulata* leafs. Gomes VM[1], Pessoa ODL[2], Santos CF[3], Fonteles MC[4], Lessa LMA[5], Nascimento NRF[6] [1]UECE − Fisiologia, [2]UFC − Química Orgânica, [3]UECE − Medicina, [4]Mackenzie − Fisiologia e Farmacologia, [5]ISCB-UECE, [6]UECE − Medicina Veterinária

06.081

Blood arterial pressure and vasoactive peptides concentration after nephrectomy in rats with different ace phenotypes. da Silva RM, Tanae MM, Peixoto HS, Souccar C, Lapa AJ, Lima-Landman MTR UNIFESP − Farmacologia

06.082

Cardiovascular responses to bothropstoxin. Rodrigues MAP, Dias L, Smaal A, Rennó AL, da Silva DA, Lorenzetti R, Hyslop S, Hyslop S UNICAMP − Farmacologia

06.083

Cardiovascular alterations caused by *Bothrops alternatus* snake venom in anesthetized dogs. Rodrigues MAP[1], Dias L[1], Smaal A[1], Rennó AL[1], Moreno Junior H[2], Mello SM[3], Hyslop S[1] [1]UNICAMP − Farmacologia, [2]UNICAMP, [3]UNICAMP − Controle de Intoxicações

06.084
The participation of AT_1 receptor in the Ang II-increased contraction in the contralateral carotid artery after balloon catheter injury. Olivon VC[1], Mestriner FL[2], Cunha FQ[2], de Oliveira AM[1] [1]FCF-USP − Física e Química, [2]FMRP-USP

06.085
Renin inhibition with aliskiren did not prevent the vascular remodeling found in 2K1C hypertension. Oliveira AM[1], Castro MM[1], Marçal DMO[1], Rizzi E[1], Ceron CS[1], Guimarães DA[1], Gerlach RF[2], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]FORP-USP − Morfologia

06.086
Renal cyclooxygenase expression in rats treated with *Bothrops alternatus* venom. Rennó AL[1], Penteado CF[2], Linardi A[1], Hyslop S[1] [1]UNICAMP − Farmacologia, [2]UNICAMP − Hemocentro

06.087
Effects of high-fat diet during six week on biochemical parameters and arterial blood pressure of Wistar rats. Milet-Morais MM[1], Silva OA[1], Figueiredo TG[2], Guedes GS[2], Xavier FE[3], Rabelo LA[2], Pinto Duarte G[1] [1]UFPE − Fisiologia e Farmacologia, [2]UFAL − Fisiologia e Farmacologia, [3]UFES − Fisiologia

06.088
Cardiovascular effects produced by chronic treatment with L-arginine in hypertensive rats. Baracho NCV[1], Silva GF[2], Bernardes DSV[1], Oliveira RCS[1] [1]FMIT − Farmacologia e Bioquímica, [2]FMIT

06.089
Cardiovascular effects produced by chronic treatment of sodium cyclamate in hypertensive rats. Pereira AC, Zaroni ACE, Tavares JD, Furtado GS, Baracho NCV, Irulegui RSC FMIT

07. Endocrine and Gastrointestinal Pharmacology

07.001
Role of the cholinergic/NO pathway in delayed gastric emptying of liquids induced by α-terpineol in awake rats. Bento-Silva MT[1], Marques RB[2], Oliveira FGV[1], Silva LL[2], Piauilino CA[2], Oliveira IS[2], Pinheiro, ADN[1], Santos AA[3], Oliveira FA[2], Almeida FRC[4] [1]UFC − Fisiologia e Farmacologia, [2]NPPM-CCS-UFPI, [3]UFC − Medicina, [4]UFPI − Bioquímica e Farmacologia

07.002
Characterization of neuro-humoral pathways in increased gastric retention of liquids due to mechanical right atrium stretch in awake rats. Palheta Junior RC[1], Okoba W[1], Bento-Silva MT[1], Pinheiro ADN[1], Oliveira, FGV[1], Elias LLK[2], Rodrigues JA[2], Santos AA[1] [1]UFC − Fisiologia e Farmacologia, [2]FMRP-USP − Fisiologia

07.003
Hypoglycemic activity of new derivates sulfonilhidrazonics in the animal model of type 1 diabetes. Oliveira LGT[1], Kartnaller MA[1], D´Andrea ED[2], Lima ML[2], Barreiro EJ[2], Sudo RT[1], Zapata-Sudo G[1] [1]UFRJ − Desenvolvimento de Fármacos, [2]FF-UFRJ

07.004
Antiulcerogenic activity of ethanolic and aqueous extracts of the bark of *Terminalia catappa* in gastric ulcer induced by ethanol in *Rattus norvegicus*. Viana VSL, Brito-Júnior EC, Rabelo RS, Nunes-Filho DM, Maia EP, Martins MCC, Nunes PHM UFPI − Biofisica e Fisiologia

07.005
Nitric oxide (NO) pathway influence the gastroprotection induced by carbon monoxide (CO) against ethanol- induced gastric damage in mice. Lucetti LT, Medeiros J-VR, Gomes AS, Santana APM, Soares PMG, Ribeiro RA, Souza MHLP UFC − Fisiologia e Farmacologia

07.006
Role of nitric oxide synthase (NOS) in the gastroprotective effect of hydrogen sulphide (H_2S) in ethanol-induced gastric damage in mice. Lucetti LT[1], Medeiros J-VR[2], Gomes AS[1], Santana APM[1], Barbosa ALR[1], Soares PMG[3], Cunha FQ[4], Ribeiro RA[1], Souza MHLP[1] [1]UFC − Fisiologia e Farmacologia, [2]UFPI − Biologia, [3]UFC − Morfologia, [4]FMRP-USP

07.007
A possible role of leptin and adiponectin during differentiation of monocyte in macrophage *in vitro*. Acedo SC[1], Gambero S[2], Cunha FGP[2], Lorand-Metze I[2], Gambero A[1] [1]UNIFAG-USF, [2]UNICAMP − Hemocentro

07.008
Gastroprotective effect of nitrosyl-ruthenium against the ethanol-induced gastric damage in mice. Santana APM[1], Torres JNL[1], Medeiros J-VR[2], Lucetti LT[1], Soares PMG[3], Wong DVT[1], Tavares BM[1], Saraiva MIR[1], Lopes LGF[4], Souza MHLP[1] [1]UFC − Fisiologia e Farmacologia, [2]UFPI – Biologia, [3]UFC − Morfologia, [4]UFC − Química Orgânica e Inorgânica

07.009
Perinodal adipose tissue and mesenteric lymph node cells interactions during inflammatory intestinal response: adipocytokine and polyunsaturated fatty acids profile. Gotardo EMF[1], Acedo SC[1], De Oliveira CC[1], Carvalho, PO[2], Gambero A[1] [1]UNIFAG-USF, [2]USF − Multidisciplinar

07.010
Saccharomyces boulardii ameliorates gastric dysmotility and inflammation presents in intestinal mucositis induced by 5-fluorouracil in mice. Justino PFC[1], SILVA LM[1], Melo LFM[1], Costa JVG[1], Nogueira AF[1], Lucetti LT[1], Ribeiro RA[1], Souza MHLP[1], Soares PMG[2] [1]UFC −

Fisiologia e Farmacologia, [2]UFC – Morfologia/Fisiologia e Farmacologia

07.011
Intestinal permeability test as a useful tool to discriminate patterns of diarrhea due to cancer chemotherapy agents. Wong DVT[1], Bem AXC[1], Nunes LG[1], Leite LL[1], Noronha FJD[1], Barbosa CRN[1], Brito GAC[2], Souza MHLP[1], Lima AAM[1], Lima-Júnior RCP[1], Ribeiro RA[1] [1]UFC – Physiology and Pharmacology, [2]UFC – Morphology

07.012
Mechanisms involved in delayed gastric emptying induced by thermogenic supplement in female ovariectomized mice. Sousa, LN[1], Santos, RGS[1], Oliveira, FGV[2], Silveira, GL[2], Bento-Silva MT[2], Monteiro FMF[1] Santos AA[2], Palheta Junior RC[1] [1]UNIVASF – Veterinary Medicine, [2]UFC – Physiology and Pharmacology

07.013
Metyrapone reverses effects of LPS on neuroendocrine response and maternal behavior of lactating female rats. Vilela FC, Melo CM, Andrade CAF, Giusti-Paiva A ICB-UNIFAL

08. Respiratory, Urinary and Reproductive

08.001
NO association between vitamin D Receptor haplotype and preeclampsia in a Brazilian population. de Rezende V[1], Sandrim VC[1], Palei ACT[2], Cavalli RC[2], Luizon MR[1], Tanus-Santos JE[1] [1]FMRP-USP – Farmacologia, [2]FMRP-USP – Ginecologia e Obstetrícia

08.002
Effect of intrauterin undernutrition in rat vas deferens: altered Ca^{2+} homeostasis and implications in male fertility. Muzi-Filho H[1], Souza AM[1], Bezerra CGP[1], Boldrini LC[2], Takiya CM[2], Oliveira FL[2], El-Cheikh MC[2], Einicker-Lamas M[3], Vieyra A[3], Lara Morcillo LS[1], Cunha VMN[1] [1]ICB-UFRJ – Farmacologia Celular e Molecular, [2]ICB-UFRJ – Ciências Morfológicas, [3]IBCCF-UFRJ – Instituto de Biofisica Carlos Chagas Filho

08.003
Functional characterization of erectile dysfunction in middle-aged rats. Silva FH, Monica FZT, Priviero FBM, Flores Toque HA, Antunes E UNICAMP – Pharmacology

08.004
Pre-clinical evaluation of the isoniazid-derived compounds IQG-607 and IQG-639 in a mouse model of tuberculosis. Rodrigues-Junior VS[1], Santos Jr AA[1], Jader ABS[1], Souto AA[1], Calixto JB[2], Basso LA[1], Santos DS[1], Campos MM[1] [1]INCTTB-PUCRS, [2]UFSC – Farmacologia

08.005
Effect of chronic undernutrition in rat vas deferens: altered Ca^{2+} homeostasis and implications in male fertility. Muzi-Filho H[1], Bezerra CGP[1], Souza AM[1], Boldrini LC[2], Takiya CM[2], Oliveira FL[2], El-Cheikh MC[2], Einicker-Lamas M[3], Vieyra A[3], Lara Morcillo LS[1], Cunha VMN[1] – [1]ICB-UFRJ – Farmacologia Celular e Molecular, [2]ICB-UFRJ – Ciências Morfológicas, [3]IBCCF-UFRJ

08.006
Functional, molecular and morphological characterization of bladder dysfunction in diabetic mice. Leiria LOS[1], Carvalho FDGF[1], Franco-Penteado CF[2], Monica FZT[1], Claudino MA[1], Schenka A[1], Nucci G[1], Antunes E[1] [1]UNICAMP – Farmacologia, [2]UNICAMP – Hemocentro

08.007
Relaxation of airways smooth muscle induced by glucagon is dependent of epithelial-derived factor. Insuela DBR, Coelho LP, Cruz CCD, Serra MF, Cordeiro RSB, Silva PMR, Martins MA, Carvalho VF IOC-FIOCRUZ – Fisiologia e Farmacodinâmica

08.008
Role of CXCR2 in a model of pulmonary infection with *Klebsiella pneumoniae*. Paula TP[1], Arifa RDN[1], Ávila TV[2], Fagundes CT[3], Cruz RC[1], Werneck SMC[1], Karklin YC[1], Baltazar LM[1], Madeira MFM[1], Campi PS[1], Pinho V[3], Teixeira MM[3], Souza DG[3] [1]UFMG – Microbiologia, [2]UFPR – Farmacologia, [3]UFMG – Bioquímica e Imunologia

08.009
Alpha-1 adrenoceptor mediated contractions of the rat bladder neck. Pacini ESA, Pupo AS UNESP Botucatu – Farmacologia

09. Natural Products and Toxinology

09.071
Antioxidant activity of dichloromethane fraction of *Baccharis trimera* and its effects on murines macrophages. Freitas GM, Gayer CRM, Coelho MGP, Sabino KCC UERJ – Bioquímica

09.072
Pulsed therapeutic ultrasound effects on skeletal muscle damage induced by *Bothrops jararacussu* snake venom. Tomaz MA[1], Saturnino-Oliveira J[2], Machado MM[1], Cons BL[1], Calil-Elias S[3], Martinez AMB[4], Melo PA[1] [1]UFRJ – Farmacologia Básica e Clínica, [2]UESC – Microscopia Eletrônica, [3]UFF – Farmácia e Administração Farmacêutica, [4]UFRJ – Embriologia e Histologia

09.073
Study of the neuropharmacological activity of the compound GB-2a obtained from *Rheedia gardneriana*. Santos ECS[1], Marques de Carvalho RS[1], Cechinel-Filho V[2], De Lima TCM[1] [1]UFSC – Farmacologia, [2]NIQFAR-UNIVALI

09.074
Effects of chronic treatment with aqueous extract of *Cuphea balsamona* L. on the lipid profile of rats submitted to a high-cholesterol diet. Baracho NCV[1], Brügger PG[2], Camanducaia DSM[2], Sanches AIF[2], Sanches RS[2] [1]FMIT − Farmacologia e Bioquímica, [2]FMIT − Medicina

09.075
Evaluation of antinociceptive property of ethanolic extract of *Buddleja brasiliensis*. Cavallini OF[1], Marinho DG[2], Freitas GA[1], Carneiro LU[1], Contarato KS[1], Almança CCJ[3], Marinho BG[1] [1]UFES − Medicina Veterinária, [2]ICB-UFRJ − Farmacologia e Química Medicinal, [3]FAFIA − Farmácia

09.076
Antidiarrheic activity of hidroalcoholic extract from barks of *Astronium fraxinifolium* schott in mice. Serikava SMO[1], Kushima H[1], Hiruma-Lima CA[1], da Silva VC[2], Vilegas W[3] [1]IB-UNESP-Botucatu − Fisiologia, [2]IQ-UNESP − Química Orgânica, [3]UNESP-Araraquara − Química Orgânica

09.077
Evaluation of the anti inflammatory cutaneous effect of *Croton brasiliensis*. Silva MO[1], Prudente AS[1], Conserva LM[2], Cabrini DA[1], Otuki MF[3] [1]UFPR − Farmacologia, [2]IQB-UFAL, [3]UEPG − Ciências Farmacêuticas

09.078
Crotoxin modifies intracellular signaling involved in phagocytosis by neutrophils. Lima TS[1], Sampaio SC[1], Della-Casa MS[2], Cirillo MC[1] [1]IBu − Fisiopatologia, [2]IBu − Imunopatologia

09.079
Anxiogenic-like effect of repeated administration of *Passiflora alata* aqueous extract in rodents in the elevated plus maze. Braga A[1], Fenner R[1], Betti AH[1], Stolz ED[2], Hasse DR[3], Gosmann G[1], Rates SMK[1] [1]UFRGS − Ciências Farmacêuticas, [2]UFRGS − Neurociências, [3]UFRGS − Psicofarmacologia Experimental, [6]UFRGS − Ciências Farmacêuticas

09.080
Bioassay-guided fractionation of the marine sponge *Polymastia janeirensis* for anticancer and anticoagulant activity. Biegelmeyer R[1], da Frota Jr. MLC[2], Andrade JMM[1], Carraro JLF[3], Zanotto-Filho A[2], Lorenzi R[2], Mothes B[3], Moreira JCF[2], Henriques AT[1] [1]UFRGS − Farmacognosia, [2]UFRGS − Bioquímica, [3]Fundação Zoobotânica − Ciências Naturais

09.081
Biomonitoring of *Coutarea hexandra* in topic model of inflammation in mice. Prudente AS[1], Lima SF[2], Conserva LM[2], Cabrini DA[1], Otuki MF[3] [1]UFPR − Farmacologia, [2]IQB-UFAL, [3]UEPG − Ciências Farmacêuticas

09.082
Preliminary investigation on the pharmacological activities of *Araucaria angustifolia* hydroalcoholic extract in insects. Lucho APB[1], Corrêa MS[2], Franco J[3], Dal Belo CA[1] [1]UNIPAMPA − Toxinologia, [2]UNIPAMPA − Química, [3]UNIPAMPA − Bioquímica

09.083
Amblyomin-X impairs VEGF-induced new vessels formation by altering endothelial cell functions. Drewes CC[1], Dias RYS[1], Hebeda CB[1], Simons SM[2], Chudzinski-Tavassi AM[2], Farsky S[1] [1]FCF-USP − Análises Clínicas e Toxicológicas, [2]IBu − Bioquímica

09.084
Ability of suramin to antagonize permeability alterations induced by honey bee venom. El-Kik CZ, Fernandes FFA, Fonseca TF, Gaban GA, Branco AMC, Silva CLM, Melo PA UFRJ − Farmacologia Básica e Clínica

09.085
Purification and characterization of hyaluronidase from venom of the Brazilian spider *Vitalius dubius* (Araneae, Theraphosidae). Sutti R, Tamascia ML, Hyslop S UNICAMP − Farmacologia

09.086
Effect of oral treatment with crude extract of *Plectranthus neochilus* in models of nociception and injury of the stomach mucosa. Calheiros AS[1], Souza[1], Azeredo JA[2], Castro-Faria-Neto HC[2], Frutuoso VS.[2] [1]FIOCRUZ – Imunofarmacologia, [2]FIOCRUZ − Fisiologia e Farmacodinâmica

09.087
Hecogenin-induced gastroprotection against acute gastric lesions: role of prostaglandins and K^+-channels. Neves KRT[1], Cerqueira GS[1], Siqueira RMP[1], Rocha NFM[1], Freitas APF[2], Vasconcelos SMM[1], Leal LKAM[3], Rios ERV[1], Macedo DS[1], Viana GSB[1], Moura BA[1], Sampaio LRL[1], [1]UFC − Fisiologia e Farmacologia, [2]UFC − Ciências Médica, [3]UFC − Farmácia

09.088
Antinociceptive effect of the hydroalcoholic extract of *Salvia officinalis* in mice. Rodrigues MRA[1], Kanazawa LKS[1], dos Santos FC[1], Neves TLM[1], Pereira IT.[1], Burci LM[1], Santos ARS[2], Pizzolatti GM[3], Baggio CH[1], Werner MFP[1] [1]UFPR − Farmacologia, [2]UFSC − Ciências Fisiológicas, [3]UFSC − Química

09.089
Antiophidic activity of the Amazon plant *Humirianthera ampla* and its compounds lupeol and sitosterol. Strauch MA[1], Azevedo SM[2], Ricardo HD[3], Lemos BC[3], Tomaz MA[1], Machado MM[3], Melo PA[1] [1]UFRJ − Farmacologia Básica e Clínica, [2]UNIR − Química Orgânica, [3]UFRJ − Farmacologia e Química Medicinal

09.090
Evaluation of anti-inflammatory activity of crude extract from *Sapium glandulatum (Vell.)*. Soley BS[1], Mendes DAGB[1], Ferreira BGA[2], Zuffellato-Ribas KC[2], Otuki MF[3], Cabrini DA[1] [1]UFPR − Farmacologia, [2]UFPR − Botânica, [3]UEPG − Ciências Farmacêuticas

09.091
Hypericum polyanthemum and its main phloroglucinol derivative uliginosin B present synergistic effect with different antidepressant drugs in the forced swimming test in mice. Stein AC[1], Centurião FB[1], Haas JS[1], Viana AF[2], Do Rego JC[3], Costentin J[3], Von Poser GL[2], Rates SMK[2] [1]UFRJ − Farmácia, [2]UFRGS − Ciências Farmacêuticas, [3]Université de Rouen − Neuropsychopharmacologie Expérimentale

09.092
Cardiovascular effects of *Syzygium cumini* L. Skeels fruit extract in rats. Tenório EP[1], Ferreira AKB[1], Oliveira DA[1], Aquino PGV[2], Araújo-Júnior JX[1], Santana AEG[2], Ribeiro EAN[1] [1]ESENFAR-UFAL, [2]UFAL − Química e Biotecnologia

09.093
Subchronic toxicity of a proteolytic fraction from *C. candamarcensis* latex: qualitative histopathological analysis. Villalba MIC[1], Bilheiro RP[2], Salas CE[3], Cassali, G. D.[4], Vasconcelos A[4], Tagliati CA[5], Lopes MTP[2] [1]UFMG − Fisiologia e Farmacologia, [2]UFMG − Farmacologia, [3]UFMG − Bioquímica e Imunologia, [4]UFMG − Patologia Geral, [5]UFMG − Farmácia

09.094
Effect of salvia (*Salvia officinalis*) hydroalcoholic extract on the topic anti-inflammatory response in mice. Lopes VM, Fonseca JP, Melo GAN, Damião JM, Freitag A, Amado CAB, Cuman RKN UEM − Farmácia e Farmacologia

09.095
Spasmolytic action of *Solanum agrarium* sendtner (Solanaceae) involves blockade of L-type calcium channels on guinea-pig ileum. Oliveira GA[1], Correia ACC[1], Santos RF[1], Agra MF[1], Silva TMS[2], Silva BA[1] [1]LTF-CCS-UFPB − Ciências Farmacêuticas, [2]DQ-UFRPE

09.096
Hydroxydihydrocarvone, a monoterpene derivative, decreases carrageenan-induced inflammation in rodents. Camargo E, de Souza DP UFS− Fisiologia

09.097
Evaluation of anethole obtained from *Foeniculum vulgare Mill* essential oil on renal ischemia and reperfusion in mice. Fonseca JP[1], Lopes VM[1], Damião MJ[1], Pinheiro RJ[1], Giannocco G[2], Bersani-Amado CA[1], Cuman RKN[1] [1]UEM – Farmácia e Farmacologia, [2]USP − Fisiologia e Biofísica

09.098
Inhibitory effects of *Combretum leprosum* Mart. & Eicher, *Protium heptaphyllum* March and *Copernica prunifera* on glycated hemoglobin. Piauilino CA[1], Sales Filho HLA[1], Sousa VR[1], Ayres MCC[2], Carvalho AA[2], Chaves MH[2], Brito SMRC[1] [1]NPPM-UFPI, [2]UFPI − Química

09.099
Involvement of potassium channels and cyclic nucleotides in the tocolytic action of Labdane-302 on rat uterus. Travassos RA, Macedo CL, Santos RF, Oliveira GA, Silva ACL, Carreiro JN, Ferreira TF, Tavares JF, Silva BA LTF-UFPB − Ciências Farmacêuticas

09.100
Involvement of K^+ channels on spasmolytic effect of the fraction of the total alkaloids from *Solanum paludosum* Moric. root bark on guinea-pig ileum. Silva ACL[1], Monteiro FS[1], Martins IRR[2], Travassos RA[4], Santos RF[2], Agra MF[1], Basílio IJLD[2], Bhattacharyya J[2,] Silva BA[1], [1]LTF-UFPB − Ciências Farmacêuticas, [2]LTF-CCS-UFPB

09.101
Evaluation of the activity of the crude extract, fractions and isolated compounds obtained from the leaves of *Chrysophyllum cainito* against sensory changes present in experimental models of clinical pain in rodents. Meira NA[1], Quintão NLM[1], Cechinel Filho V[1], Klein Jr LC[2], Martin Z[3], Rodriguez LMP[3] [1]NIQFAR-UNIVALI – Ciências Farmacêuticas, [2]UNIVALI − Produtos Naturais e Substâncias Bioativas, [3]CICY − Biotecnologia

09.102
Gastroprotective activity of chloroform and aqueous fractions obtained of hydroalcoholic extracts of *Brassica oleracea* L. var. *acephala* DC. Lemos M, Santin JR, Oliveira AP, Klein LC, Niero R, Andrade SF NIQFAR-CCS-UNIVALI

09.103
Antinociceptive effects of *Rheedia longifolia* Planch & Triana aqueous extract and its fractions. Nascimento DD[1], Siqueira AM[1], Costa NF[1], Bérenger ALR[2], Castro-Faria-Neto HC[1], Figueiredo MR[2], Frutuoso VS[1] [1]IOC-FIOCRUZ − Imunofarmacologia, [2]Farmanguinhos-FIOCRUZ − Produtos Naturais

09.104
Hypertension and oxidative stress associated with development of fetal programming: influence of extract from *Vitis vinífera* grape skin. Emiliano da Silva AF[1], Costa CA[2], Bem G[2], Carvalho LCRM[3], Boaventura GT[4], Soares de Moura R[3], Resende AC[1] [1]UERJ − Farmacologia e Psicobiologia, [2]UERJ − Farmacologia e Psicobiologia, [3]UERJ − Farmacologia, [4]UFF − Nutrição Dietética

09.105
Comparative study between the effect of aqueous extract of B*ixa orellana L.* and sinvastatin on

lipidic profile and blood pressure of hypertensive rats and submitted to a high-cholesterol diet. Reis MLA, Baracho NCV, Ferreira MRC FMIT

09.106
Study of the acute toxicity of *Eugenia brasiliensis* Lamarck and *Eugenia beaurepaireana* (Kiaerskou) Legrand extracts on mice. Lemes EV[1], Cabrini DA[2], Otuki MF[2], Pizzolatti MG[3], Brighente IMC[3], Magina MDA[4], Beirith A[5] [1]FURB – Physiotherapy, [2]UFPR – Pharmacology, [3]UFSC – Chemistry, [4]FURB – Pharmaceutical Sciences, [5]FURB – Natural Sciences

09.107
Evaluation of the anti-inflammatory and antinociceptive activities of the leaf and stem of *Costus spiralis* (Jacq.) Roscoe (Costaceae). Campesatto-Mella E[1], Araújo MV[1], Silva AKD[1], Santos DLF[1], Delatorre P[2], Rocha BAM[3] [1]UFAL - Farmacologia, [2]UFPB - Biologia Celular, [3]UFC – Bioquímica.

10. Cancer and Cell Proliferation

10.001
Obesity and cancer development: effect of metformin. Fonseca EAI[1], Oliveira, M. A.[1], Tostes RCA[1], Colquhoun A[2], Carvalho MHC[1], Zyngier SZ[1], Fortes ZB[1] [1]ICB-USP – Farmacologia, [2]ICB-USP – Biologia Tecidual e do Desenvolvimento

10.002
Effects of $P2X_7$ receptor agonists on cell proliferation of human glioma cell lines U138-MG and M059J. Gehring MP[1], Campos MM[2], Battastini AMO[3], Morrone FB[4] [1]PUCRS – Farmacologia Aplicada, [2]PUCRS – Cirurgia-Odontologia, [3]UFRGS – Bioquímica, [4]PUCRS – Farmácia

10.003
Evaluation of butyrate and aqueous extract of the *Ilex paraguariensis* enemas in reducing the levels of malondialdehyde in exclusion of colitis. Silva CMG, Lameiro TMM, Marques LHS, Almeida MG, Cunha FL, Martinez CAR – Sao Francisco University – Cancer and cell proliferation

10.004
Glucose starvation induces melanogenesis in B16F10 murine melanoma cells through oxidative stress. Piva B[1], Diaz BL[2] IBCCF-UFRJ – Programa de Imunobiologia

10.005
Structure-related activity of a series of chalcones derived from quinoxaline on *in vitro* oral squamous cell carcinoma proliferation. Mielcke TR[1], Mascarello A[2], Calixto JB[3], Yunes RA[2], Leal PC[2], Morrone FB[4], Campos MM[5] [1]PUCRS – Farmacologia, [2]UFSC – Química, [3]UFSC – Farmacologia, [4]PUCRS – Farmácia, [5]PUCRS – Cirurgia-Odontologia

10.006
Stress-related neurohormonal mediators influence the human oral cancer cells behavior. Bernabé DG[1], Tamae AC[1], Miyahara GI[2], Biasoli ER[2], Oliveira SHP[1] [1]FOA-UNESP – Ciências Básicas, [2]FOA-UNESP – Ciências Básicas, [3]FOA-UNESP – Oncologia Bucal

10.007
Anticancer Activities of 2,2-Dimethyl-3-(3-nitrophenylamino)-2,3-dihydro-naphtho[1,2-*b*]furan-4,5-dione (1): Oxidative Stress based-apoptosis. Araújo AJ[1], Marinho-Filho JDB[1], Silva-Junior EN[2], Moura MABF[3], Goulart MOF[4], Ferreira VF[2], Pessoa C[1], Moraes MO[1], Costa-Lotufo LV[1], Montenegro RC[1] [1]UFC – Fisiologia e Farmacologia, [2]UFF – Química Orgânica, [3]UFAL – Química, [4]UFAL – Química e Biotecnologia

10.008
Effect of crotoxin, the main toxin of the rattlesnake C.*d. terrificus* venom, on secretory activity of peritoneal macrophages during tumor progression. Studies *in vivo* and *in vitro*. Costa ES[1], Faiad OJ[1], Curi R[2], Cury Y[1], Sampaio SC[1] [1]IBu – Fisiopatologia, [2]ICB-USP – Fisiologia e Biofísica

10.009
Cytotoxic activity of benzothiazole analogues. Vieira GC[1], Araújo AJ[1], Vasconcelos, TRA[2], Ferreira VF[2], Nogueira, AF[2], Pessoa CO[1], Costa-Lotufo LV[1], Montenegro RC[1], Moraes MO[1] [1]UFC – Fisiologia e Farmacologia, [2]UFF – Química Orgânica

10.010
Assessment of the cytotoxic, antiproliferative, genotoxic, mutagenic and pro-apoptotic activities of indirubin, directed for its use as an anticancer substance. Fogaça MV[1], Cardoso PF[1], Cândido PM[1], Varanda EA[2], Calvo TR[3], Vilegas W[3], Cólus, IMS[1] [1]UEL– Biologia Geral, [2]FCFAR-UNESP – Ciências Biológicas, [3]IQ-UNESP – Química

10.011
Characterization of cytotoxic activity induced by phospholipase A2 (PLA-LYS49) isolated from *Bothrops jararacussu* snake venom. Lino CNR[1], Sorgi CA[2], Cintra ACO[2], Sampaio SV[2], Faccioli LH[2], Nomizo A[2] [1]FFCLRP-USP – Biologia, [2]FCFRP-USP – Análises Clínicas, Toxicológicas e Bromatológicas

10.012
Involvement of the kinin B1 receptor in melanoma progression. Maria AG[1], Reis RI[1], Dillenburg-Pilla P[1], Pesquero JB[2], Costa-Neto CM[1] [1]FMRP-USP – Bioquímica e Imunologia, [2]UNIFESP – Biofísica

10.013
Overexpression of platelet-derived growth factor receptor-α in basal-like triple-negative breast cancer. Melo-Filho AF[1], Ribeiro RA[2], Rodrigues EJM[2], Magalhães HO[1], Soares FA[3], Chagas DWN[2], Lima VCC[4] – [1]ICC – Mastologia, [2]UFC –

Fisiologia e Farmacologia, [3]HCANC − Anatomia Patológica, [4]HCANC − Oncologia Clínica

10.014
Leptin activates the mTOR pathway in epithelial cells: roles in lipid metabolism, inflammatory mediator production and cell proliferation. Fazolini NPB FIOCRUZ − Fisiologia e Farmacodinâmica. Fazolini NPB[1], Viola JPB[2], Maya-Monteiro CM[1], Bozza PT[1]. [1]IOC-FIOCRUZ − Imunofarmacologia, [2]INCa – Cellular Biology

10.015
Investigation of cytotoxicity of 4-nerolidilcaterol and its synthetic derivatives. Cunha CRM[1], Menegatti R[2], Valadares MC[2] [1]UFG − Farmacologia e Toxicologia Celular, [2]UFG − Química Medicinal

10.016
Effects of tumor-derived extracellular matrix on endothelial cell functions: implications to tumor-associated angiogenesis. Brandão-Costa RM[1], Saldanha-Gama RF[2], Helal Neto E[2], Morandi V[3], Barja Fidalgo TC[1] [1]UERJ − Farmacologia, [2]UERJ − Farmacologia e Psicobiologia, [3]UERJ − Biologia Celular e Genética

10.017
Withaphysalin F induces apoptosis and necrosis in HER-2 overexpression breast cell line. Montenegro RC[1], Rocha DD[1], Rodrigues FAR[1], Lima PDL[4], Pessoa CO[2], Maia I[3], Pessoa ODL[3], Moraes MO[1] [1]UFC − Fisiologia e Farmacologia, [2]UFC − Oncologia Experimental, [3]UFC − Química Orgânica, [4]UEPA – CCBS

10.018
5-hydroxy-2-(4-methylphenylthio)-1,4-naphtho-quinone, a juglone derivative, induces apoptosis and necrosis in hl-60 cell line. Araújo AJ[1], Montenegro RC[1], Marinho-Filho JDB[1], Rocha DR[2], Souza ACG[3], Pessoa C[1], Costa-Lotufo LV[1], Ferreira VF[3], Santos WC[4], Moraes MO[1] [1]UFC – Fisiologia e Farmacologia, [2]UFF − Instituto de Química, [3]UFF − Química Orgânica, [4]UFF − Farmácia e Administração Farmacêutica

11. Clinical Pharmacology, Pharmaco-kinetics, Pharmacogenomics and Preclinical Toxicology

11.023
Plasma levels of matrix metalloproteinases -8 and -9 and their endogenous inhibitors TIMP-1 and TIMP-2 in untreated hypertensive patients. Fontana V[1], Silva PS[2], Belo VA[2], Biagi C[3], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]FCM-UNICAMP − Farmacologia, [3]Santa Casa de Araçatuba − Cardiologia

11.024
Efficacy and safety of the *Mentha crispa* in the treatment of trichomonas infections: a randomized, open and parallel study. Pimenta Costa CS, Cavalcanti PP, Cunha GH, Pontes AV, Fechine FV, Oliveira JC, Andrade WS, Moraes RA, Camarão GC, Moraes MEA UFC − Fisiologia e Farmacologia

11.025
Effects of sitagliptin on visual alterations diagnosed by visual evoked potential in type 2 diabetic patients. Capistrano Júnior, VL[1], Tagliapietra JI[1], Pontes AV[1], Cunha GH[1], Rocha MBS.[1], Frota Bezerra FA[1], Vale OC[1], Fernandes VO[2], Montenegro Jr RM[2], Moraes MEA[1] [1]UFC − Fisiologia e Farmacologia, [2]HU-UFC − Endocrinologia e Diabetes, [4]HU-UFC − Endocrinologia e Metabologia

11.026
NAT2, GSTT1 and GSTM1 genotypes and predisposition to adverse drug reaction (ADR) in tuberculosis patients. Costa GNO[1], Santana CVN[2], Konstantinovas C[1], Magno LA[3], Bastos-Rodrigues L[3], Miranda DM[4], Romano-Silva M[3], Marco LAC[5], Di Pietro G[1], Rios-Santos F[1] [1]LAFEM-UESC − Ciências da Saúde, [2]LAFEM-UESC − Ciências Biológicas, [3]UFMG − Saúde Mental, [4]UFMG − Pediatria, [5]UFMG − Cirurgia

11.027
Sulfadiazine determination in human plasma by high-performance liquid chromatography coupled to tandem mass spectrometry (HPLC-MS-MS): application to pharmacokinetic study. Nascimento DF, Oliveira JC, Pimenta Costa CS, Rocha MBS., Moraes RA, Cunha GH, Magalhães MS, Uchoa CRA, Moraes MO, Moraes MEA UFC − Fisiologia e Farmacologia

11.028
Susceptible NOS3 (endothelial nitric oxide synthase) gene haplotypes in hypertension and resistant hypertension. Luizon MR[1], Sandrim VC[2], Izidoro-Toledo TC[1], Coelho EB[3], Tanus-Santos JE[1] [1]FMRP-USP − Farmacologia, [2]Santa Casa de Belo Horizonte, [3]FMRP-USP − Clínica Médica

11.030
New educational strategies to improve pharmacology teaching: a interdisciplinary approach. Fidalgo-Neto AA, Lopes RM, Alves LA[2] IOC-FIOCRUZ Laboratório de Comunicação Celular

11.031
Interference of matrix metalloproteinase (MMP)-9 genotypes and haplotypes in the responsiveness to antihypertensive therapy of patients with preeclampsia or gestational hypertension. Palei ACT[1], Sandrim VC[2], Cavalli RC[3], Gerlach RF[4], Tanus-Santos JE[5] [1]FCM − UNICAMP − Farmacologia, [2]Santa Casa de Belo Horizonte − Farmacologia, [3]FMRP-USP − Ginecologia e Obstetrícia, [4]FORP-USP − Morfologia, [5]FMRP-USP – Farmacologia

11.032
Comorbidities and medication use in elderly women with vestibular disorders. Prezotto AO, Paulino CA, Onishi ET UNIBAN

11.033
Serum cortisol and IL-10 levels increase in chronic renal failure patients with cognitive deficit. Degaspari D[1], Stein G[2], Munhoz CD[1], Martins JPB[2], Ribeiro Junior E[2], Sá Lima L[1], Tzanno-Martins CB[1], Scavone C[1], Kawamoto EM[1] [1]ICB-USP – Farmacologia, [2]CEHUS-CINE

11.034
Changes in cardiovascular and biochemical parameters produced by chronic treatment with celecoxib in normotensive rats. Figueiredo LF, Baracho NCV FMIT

Conferences

Ligand-directed signaling bias and its potential for new therapeutics.

Roger J. Summers (Monash Institute, Australia).

Ligand-directed signaling bias describes the reversal of efficacy and/or potency observed for different signaling pathways by certain drugs acting at G protein-coupled receptors. Current evidence suggests that it may be explained in terms of the production or selection of unique receptor conformations by particular drugs. Several key factors have emerged for the identification of signaling bias. The bioassay critically affects the type and magnitude of drug efficacy observed and many drugs are now known that behave as classical antagonists for one signaling pathway and agonists at another. Efficacy therefore depends on the assay being used and a drug may be described as an antagonist for cAMP accumulation but an agonist for Erk1/2 phosphorylation, an effect termed pluridimensional efficacy (Galandrin & Bouvier, 2006). Recent studies with β_1- β_2- and β_3-ARs have shown that many compounds originally classified as antagonists for cAMP signaling have quite different efficacy at other signaling pathways and can act as agonists, neutral or inverse antagonists for MAP kinase signaling (Galandrin & Bouvier, 2006; Baker *et al.*, 2003; Sato *et al.*, 2007; Sato *et al.*, 2008; Galandrin *et al.*, 2008) raising the possibility that further development of these compounds will allow the selection of a profile which is useful therapeutically. This work is supported by a project grant 491190 (to R.J. Summers and B.A. Evans) and program grant 519461 (to P.M. Sexton, A. Christopoulos and R.J. Summers) from the National Health and Medical Research Council of Australia. Baker JG *et al.*, (2003) *Mol Pharmacol 64, 1357-1369*; Galandrin S & Bouvier M (2006) *Mol Pharmacol 70, 1575-1584*; Galandrin S *et al.*, (2008) *Mol Pharmacol 74, 162-172*; Sato M et al., (2007) *Mol Pharmacol 72, 1359-1368*; Sato M et al., (2008) *Mol Pharmacol 74, 1417-1428*

Anti-inflammatory GPCRs as targets for novel therapeutics.

Mauro Perretti (Queen Mary University of London, London, UK).

Cell trafficking in inflammation is tightly controlled by endogenous anti-inflammatory mediators. Their properties are mediated by specific GPCRs which activate anti-inflammatory circuits limiting leukocyte extravasation in a space and time-dependent fashion. Our work focuses on some of these GPCRs, namely FPR2, a receptor activated by annexin A1 (AnxA1) and lipoxin A4, as well as MC3, a receptor activated by natural and synthetic melanocortin peptides (e.g. ACTH). Our approach is, at the beginning, pharmacological, so that the properties of specific ligands (often peptides, more recently also NCEs) as assessed in models of acute inflammation. Then, antagonists and/or transgenic mouse colonies are used to pinpoint the receptor target(s) responsible for these anti-inflammatory effects. Such an approach is then expanded by determining, on one hand, the molecular events and cellular responses regulated by FPR2 activation (in the case of Annexin A1) and, on the other hand, the pathophysiological relevance of a given receptor. The latter aspect is addressed by using transgenic colonies, and it requires the study of the full time course: we use the mouse paw oedema as a prototype of an acute model of inflammation and the K/BxN inflammatory arthritis, to monitor the relevance of these pathways in more prolonged (chronic?) inflammatory experimental conditions. Exquisite control of the process of blood-borne cell extravasation is achieved by activation of these receptors, associated with multiple proresolving actions on other processes including osteoclastogenesis, chondrocyte activation and phagocytosis of apoptotic leukocytes. Novel concepts in the field are linked to modulation of the expression of anti-inflammatory

GPCRs and to the exploitation of this approach for innovative drug discovery programs. Determining how FPR2 (and MC3) expression changes during an on-going inflammatory reaction can allow prediction of efficacy of new selective agonists for these receptors. Similarly, monitoring their expression in human cells and tissue samples would predict efficacy in human chronic inflammatory conditions. We are working on samples taken from patients suffering from rheumatoid arthritis and giant cell arteritis. In either case, treatment of patients with glucocorticoids, at doses able to control disease progression, would augment expression of FPR2 in circulating PMN and monocytes. Finally, the overall aim of this research is to develop selective agonists to either FPR2 or MC3 as novel anti-inflammatory therapeutics. We propose that these new drugs would produce very little, if any, side effects, as they will be operating in the same way our body disposes of an inflammatory episode. Such novel anti-inflammatory therapeutics would represent a fresh change to the drug discovery programs applied, and developed, so far in the pharmaceutical world. Selected References: Perretti M, Chiang N, et al. Endogenous lipid- and peptide-derived anti-inflammatory pathways generated with glucocorticoid and aspirin treatment activates the lipoxin A4 receptor. *Nature Med* 8: 1296-1302, 2002. Serhan CN, et al. Resolution of inflammation: state of the art, definitions and

terms. *FASEB J* 21:325-32, 2007. Perretti M, D'Acquisto. Annexin A1 and glucocorticoids as effectors of the resolution of inflammation. *Nat Rev Immunol* 9: 62-70, 2009.

Toll-like receptors: emerging roles in reproductive physiology and therapeutics.

Mark Hedger (Monash Institute, Australia)

Interactions between the immune and reproductive systems have important consequences for fertility and reproductive health. There is increasing evidence that many of these interactions involve pattern recognition receptors, such as the Toll-like receptors (TLRs), which recognize microbial products or "danger" signals, called pathogen associated molecular patterns. Although abundantly expressed by macrophages, TLRs are also widely distributed, particularly in epithelia. The TLRs are important in providing protection against infection in the reproductive tract, but there is increasing evidence for involvement of these receptors in more basic pathology and physiology. In the female, TLRs have been implicated in normal ovarian, endometrial and placental function, as well as in ovarian cancer, pelvic inflammatory disease, intrauterine growth restriction, pre-eclampsia and pre-term birth. In the male, TLRs appear to play a role in prostatitis and prostatic cancer, but also in the control of testicular steroidogenesis and spermatogenesis. Spermatogenesis is a complex, organized and highly regulated process involving intimate interactions between the developing germ cells and their supporting Sertoli cells. Sertoli cells express several TLRs and respond to TLR ligands by producing a number of inflammatory cytokines and mediators. These products regulate spermatogonial / spermatocyte development and many critical supportive functions of the Sertoli cells, and their production varies across the cycle of the seminiferous epithelium, with significant changes in expression coinciding with key events within the cycle. Such relationships between inflammatory signaling and spermatogenesis provide a potential mechanism to account for the link between infection/inflammation and testicular dysfunction. At least some of the negative effects of inflammation on spermatogenesis may be attributed to elevated production of inflammatory mediators that may exert disruptive effects on germ cell development and survival, as well as Sertoli cells support. Further investigation of these interactions may be applicable to improving fertility in men with disordered spermatogenesis. Finally, it is also expected that these studies may create new opportunities for modulating fertility by targeting spermatogonial renewal and meiosis as a safe, effective and reversible male contraceptive. This work has been supported by grants from the National Health and Medical Research Council and the Australian Research Council.

Indoleamine 2,3 dioxygenase (IDO) inhibitors: from bench to bedside.

Andrew L Mellor Immunotherapy Center, Medical College of Georgia, Augusta GA. USA

Indoleamine 2,3 dioxygenase (IDO) is an intracellular enzyme that catabolizes tryptophan (1). IDO is induced in settings of chronic inflammation associated with many diseases including cancer and infectious diseases. In 1998 we reported that pharmacologic inhibition of IDO during pregnancy induced rejection of fetal tissues by maternal T cells (2). This pioneering study identified IDO as a pivotal regulator of adaptive immunity, and suggested that IDO inhibitors might be effective as immune modulators to treat clinical syndromes in which immune system hyporeactivity contributed to disease progression. Immune hypo-reactivity is the hallmark of tumors and pathogens that cause persistent infections. IDO activity at sites of tumor growth and infection creates immune privilege, allowing these agents of disease to persist (3). Consistent with this notion IDO inhibitors – especially when combined with methods to incite immunity - enhance immunity to tumors and infected cells (4-6). In my presentation I will describe the current state of knowledge regarding the role of IDO in cancer and infectious diseases, and summarize progress in testing IDO inhibitors as cancer vaccine adjuvants in ongoing experimental clinical trials. This research is funded by grants from the NIH (AI063402, AI075165, AI083005). References: 1. Huang L, Baban B, Johnson BA, & Mellor AL (2010) Dendritic cells, IDO and acquired immune privilege. *Int. Rev. of Immunology* 29:133-155. 2. Munn DH, *et al.* (1998) Prevention of allogeneic fetal rejection by tryptophan catabolism. *Science* 281:1191-1193. 3. Mellor AL & Munn DH (2008) Creating immune privilege: active local suppression that benefits friends, but protects foes. *Nat Rev Immunol* 8:74-80. 4. Muller AJ, *et al.* (2005) Inhibition of IDO, an immunoregulatory target of the cancer suppression gene Bin1, potentiates cancer chemotherapy. *Nat Med* 11:312-319. 5. Potula R, *et al.* (2005) Inhibition of IDO enhances elimination of virus-infected macrophages in an animal model of HIV-1 encephalitis. *Blood* 106:2382-2390. 6. van der Sluijs KF, *et al.* (2006) Influenza-induced expression of IDO enhances IL-10 production and bacterial outgrowth during secondary pneumococcal pneumonia. *J Infect Dis* 193:214-222.

The stressed CNS: when glucocorticoids aggravate inflammation.

Javier R. Caso. Department of Biology, Stanford University, California-USA

Glucocorticoids (GCs) are hormones released during the stress response that are well known for their immune-suppressive and

anti-inflammatory properties; however, recent advances have uncovered situations wherein they have effects in the opposite direction, challenging the classic view of the GCs actions at a variety of levels. It was first observed that under some circumstances, acute GC exposure could have pro-inflammatory effects on the peripheral immune response. More recently, chronic exposure to GCs has been found to have pro-inflammatory effects on the specialized immune response to injury in the central nervous system. The central nervous system (CNS) is a particularly interesting example, both because of its unique immune environment, and because GCs affect immune responses differently in different brain regions. Thus, it has been shown that in some cases, glucocorticoids can increase pro-inflammatory cell migration, cytokine production, and even transcription factor activity in the brain.
Here, we discuss the contexts wherein GCs increase CNS inflammation and point out directions for future investigation as well as the considerable clinical implications of these findings.

Genetic modeling of PI3K inhibition.
Emilio Hirsch (University of Turin, Italy)
Phosphoinositide 3-kinases (PI3K) are crucial elements needed for receptor-mediated signal transduction and modification of PI3K signaling is emerging as a key element in cancer, inflammation, metabolic disorders and cardiovascular diseases. PI3K consist of heterodimers of a 110 kD catalytic (p110) as well as a regulatory/adapter subunit and are required for the production of a membrane bound phosphorylated lipid (PIP3) that acts as a critical secondary messenger molecule. Class I p110s (p110α, β, γ and δ) share significant homology but studies using genetically engineered mice show that they all play non-redundant roles. Interestingly, modeling by genetic means of PI3K inhibition revealed that different isoforms can be distinctly involved in different pathologies. For example, we recently demonstrated that, while PI3Kγ is crucially involved in the establishment of inflammatory responses, PI3Kβ is a key determinant in the development of Erbb2-driven mammary gland cancer. In addition, we also recently found that PI3Kγ signaling also occurs in the heart where it can modulate the contractile response and contribute to the development of heart failure. While these genetic studies recently provided support for PI3K catalytic activity as a promising drug target they also unexpectedly revealed that these proteins not only work as kinases but also as scaffolds for protein-protein interactions. Despite this complex regulation, genetic modeling of PI3K inhibition clearly supports that selective targeting of different PI3K isoforms can represent a promising strategy to improve efficacy and reduce side effects. Efforts to produce and test such drugs are under way and clinical trials are foreseen for the next future. This work is supported by Regione Piemonte, AIRC, Leducq Foundation, European Union FP6.

Chemokines and their receptors: the Nexus of neurobiology and immunobiology
Richard Ransohoff (Cleveland Clinic, USA)
Chemokines comprise a family of peptides that act through G protein-couple receptors (GPCRs) to regulate leukocyte migration throughout all tissues, in an exquisitely specific and flexible fashion. Initial studies related to neuroinflammation asked how chemokines and chemokine receptors governed inflammatory cell recruitment to the CNS during immune-mediated or virus-induced inflammation. More recently, it has become clear that the CNS complement of constitutive chemokines supports developmental and neurophysiological functions as well as regulating the behavior of both macroglia and microglia. Inflammation of the central nervous system (CNS) entails the activation of resident microglia and macroglia, as well as canonical events including recruitment of hematogenous leukocytes and degradation of blood-brain barrier function. Thus defined, inflammation accompanies most neurological disorders, including multiple sclerosis (MS), stroke, neoplasia, trauma and HIV-1-associated dementia, as well as Alzheimer's disease and other primary neurodegenerations. Because GPCRs can serve as drug targets, these results have implications for the understanding and treatment of disease by neurologists and neuroscientists.

Courses

Pharmacology of learning and memory formation.
Hudson de Sousa Buck (FCMSCSP)
Memory is the ability of humans and other animals to retain and subsequently retrieve information. Basically, the mnemonic process comprises three main stages: Encoding or registration (processing and combining of received information); Storage (creation of a permanent record of the encoded information), and; Retrieval or recall (calling back the stored information in response to some cue for use in a process or activity). Another important component of the mnemonic process that may occur is the forgetting of the information. This is a normal process that occurs all the time, but age, stress, emotions, anxiety, high blood pressure are examples of conditions that may lead to abnormal memory loss, resulting in manifestation of dementias like Alzheimer's disease. The neural mechanisms of memory are not totally determined. It is considered that the information is stored in several cortical areas (motor memory/motor cortex; visual memory/visual cortex; etc), hippocampus, dorsal striatum, parahippocampal region,

basal forebrain and cerebellum. The hippocampus is located deep inside the temporal lobe, and it receives inputs from virtually all association areas in the neocortex, including those in the parietal, temporal and frontal lobes, via the adjacent parahippocampal gyrus and entorhinal cortex. Additional inputs come from the amygdala and via a separate pathway, from the cholinergic and other regulatory systems. The dorsal striatum plays a vital role not only in learning new response strategies but also in the inhibition of preexisting strategies when a shift in strategy is required. This system is called as a declarative or relational memory system and appears to be essential for processing information about flexible utilization of the relationship between multiple external cues and events. The dorsal striatum is necessary for the mediation of stimulus response learning. The parahippocampal region receives inputs from widespread secondary or "association" cortical regions and provides the major conduct for hippocampal outputs to the same cortical association areas. This region can play a critical role in recognition memory. Various ions, neurotransmitters and messengers are associated with memory. The ion calcium participates in control of the formation and development of neural structures. Temporal and spatial control of calcium signaling through the neural circuitry involved in learning and memory are fundamental for cognitive capacities. The ion tassium can contribute to learning and memory through the slow after hyperpolarization (sAHP) and A-type potassium channel modification in hippocampal pyramidal neurons. The sAHP amplitude in hippocampal pyramidal neurons can be reduced by signaling pathways triggered by a variety of neurotransmitters, like Acetylcholine which have been implicated in learning and memory. Several findings showed that the neurotransmitters glutamate, γ-aminobutyric acid (GABA), acetylcholine, serotonin and dopamine can play a key role in the mnemonic process alone or by association among them. Glutamate, the major excitatory neurotransmitter in the brain, is associated to the modulation of cognitive process by acting on metabotropic glutamate (mGlu) receptors. Also, the positive modulation of AMPA (α-amino-3-hydroxyl-5-methyl-4-isoxazole-propionic acid)-type glutamate receptors can potentially enhance cognition by decrease the losses of glutamatergic synapses, promoting synaptic plasticity and increasing the production of tropic factors. Induction of long term potentiation (LTP), a presumed substrate of memory, requires intense depolarization of spine heads which could be induced by the activation of AMPA receptors. The γ-aminobutyric acid (GABA) is a central inhibitory neurotransmitter and the GABA system is a target for a variety of central pharmacological agents including sedatives, analgesics and anticonvulsants. Studies suggest that GABAergic drugs might impair memory formation through effects on cholinergic systems. Conversely, other findings showed that the GABA receptor agonists muscimol and baclofen enhance memory. Pharmacological data showed that the activation of muscarinic and nicotinic acetylcholine receptors have a role in the encoding of new memories. Acetylcholine might enhance the encoding of memory by increasing glutamate neurotransmission and, in this way, stimulating the LTP. Formation of LTP requires a persistent increase in neuronal stimulation with gene transcription and formation of new proteins, which leads to increases in post synaptic density. Acetylcholine might also enhance encoding through its role in increasing theta rhythm oscillations within the hippocampal formation. Learning is enhanced when stimuli are presented during periods of theta rhythmicity. Serotonin (5-HT) is another neurotransmitter that affects neuronal communication in hippocampus. 5-HT exerts a direct hyperpolarizing influence on principal cells, via 5-HT1 receptors, and indirectly, it facilitates GABA release from local interneurons through 5-HT3 receptors. Dopamine also has a great impact on cognitive processes. In the field of memory and learning studies the mesolimbic dopaminergic and especially the mesolimbic system clearly have received most of the scientific attention. These areas are known to play a crucial role in various cognitive processes. D1, D2, D3 and D4 receptors are all implicated in learning and consolidation of memory. Together with these neurotransmitters, a great variety of neuromodulators are implicated in memory formation. According to this, various attempts to characterize a proper memory pharmacology that could reverse memory decline have been done. The efficacy of memory therapeutics, however, depends on our understanding of the basic mechanisms that characterize memory itself, once memories are thought to be due to lasting synaptic modifications in the brain. Suggested literature: Roberto Lent, Cem Bilhões de Neurônios, 2005; The *Internet J of Pharmacol 7(1)*, 2009.

Neuropharmacological changes along the aging process.

Tânia Araújo Viel USP

Memory is the capacity of learn, consolidate and retrieve information that are acquired along life-time. The biological basis for memory formation requires proteins that are common to many living species. What really makes the difference between individuals is all experiences that one lives and which mobilizes those proteins to crate memory traces that are unique to each individual. During the aging process (since conception until elderly) the central nervous system develops in such a manner that

until a determined moment, the organism can increase neuroplasticity with great strength and velocity. After a point (still not determined, and that surely is different among persons), these features declines and the capacity of learning new information as well as the recall of consolidated memory starts to decline. However, there are certain types of memory that show no apparent deficit during normal aging. For example, normal elderly do not forget how to write, drive a car, or make a cup of tea, and vocabulary actually increases throughout life. There are some theories that try to explain these phenomena and these theories are related to formation of long term poten-tiation (LTP). LTP is a specific form of plasticity that normally leftovers for a long time, but there is large evidence showing that manipulations to disrupt LTP interfere with memory process and, in this way, it is reasonable to accept that age-related deficits in LTP would contribute to memory deficits along aging. LTP induced neuroplasticity depends on the integrity of neurons rather than their quantity. There is a common misconception that a significant neuronal loss is associated with aging. In fact, careful anatomical studies in humans, monkeys, rats and mice showed that there is no significant loss in the hippocampus areas during normal aging. So, the loss in neuroplasticity is likely to be related to the decline in functionality of LTP. Forma-tion and maintenance of LTP requires cellular and molecular events resulting in induction, expression and consolidation of neuronal plasticity. Induction requires cell depolarization that can be reproduced experi-mentally using theta burst stimulation (TBS, 100 Hz pulses separated by 200 ms intervals). This depolarization engages pro-teins from the cytoesqueleton such as the integrin-actin system. Alterations in actin cytoesqueleton modify spine and postsynaptic density. A constant release event would result in stable changes of the cytoesqueleton (consolidation), viewed as cross-linking of actin filaments. After that, increases in receptors densities or mobilization of receptors from extra-synaptic areas occur (mainly glutamate receptors). As a result, a stability of excitatory postsynaptic currents is installed. Actin network is constantly modulated by endogenous factors that posi-tively or negatively affect pro-duction and maintenance of stable LTP. Among the positive modulators are the neurotrophins brain-derived neurotrophic factor (BDNF) and nerve growth factor(NGF) and also some neurotransmitters such as serotonin, endorphins, canabinoids, corticosteroids, and acetylcholine, which also plays a direct role in the formation of memory. In opposite, adenosine was described as a potent negative modulator of LTP. In the elderly, the functionality of LTP can be disrupted as a result of interferences in the induction, expression or consolidation of the process and also as a result in decline of positive modulators or increase in negative modula-tors. However, there is no defined mechanism for these neuropharmacological changes. For instance, a decline in the clearance of extracellular adeno-sine was observed in aged rats which could account for the decline in LTP. Moreover, there is no consensus in the literature about the maintenance or the decrease in the quantity of the neurotrophins BDNF and NGF in the aged brain. In the same way, alteration in neurotransmitter or neuromodulator systems can also influence LTP maintenance. In a recent work we observed an increase in memory retention together with an increase in the density of α7 nicotinic acetylcholine receptors in hippocampus of rats with 6 and 12 months old, when compared to 3 month-old animals. In addition, decreases in memory and in density of those receptors were observed in 18 and 22 month-old rats. Once these receptors are involved with LTP in the hippocampus, these results also suggest an involvement of the cholinergic system with the modulation of memory along the aging process. The degree of LTP disruption during elderly can also be influenced by the quantity and quality of stimulus that is applied to the brain during aging process. In fact, people that are submitted to intellectual activities during their life have less probability to develop dementias. In the same way, young animals that are submitted to environmental enrichment present significant morphological and functional changes in the central nervous system. According to this, in another set of experiments we showed that C57Bl/6 mice submitted to environmental enrichment (boxes containing toys, activity wheels, wood objects, etc) during 15 months (from 2 to 17 months of age) showed increase in spatial memory evaluated in Barnes maze, when compared with their age-matched control. Surprisingly, mice submitted to the same protocol, but only in more advanced age (from 15 to 17 months of age), also presented improve in spatial memory when compared to non-stimulated aged-matched animals. These observations suggest that the stimulus applied during elderly is as significant and effective as those applied during the youth. Moreover, we can propose that deficits in LTP functionality, i.e., alterations in LTP and its positive or negative modulators, can be reversed by improving life quality with increases in social interactions, physical practices and changes in nutritional intake. Suggested literature: *Experim Gerontol 38*: 61-9, 2003; *Ageing Res Rev 5*:255-80, 2006; The *Internet J of Pharmacol 7(1)*, 2009; *J Neurosci 29(35)*:10883-9, 2009

Pharmacological modulation of memory process in neurodegenerative diseases.

Marielza Andrade Nunes (FCMSCSP)

Along aging, the cognitive processes naturally suffer impairments, varying from mild to aggressive forms.

The neurodegenerative diseases have some common symptoms like motor and memory deficits. In general, there are no diagnostic tests that could clarify the presence, absence or the type of the degenerative disease and frequently, the diagnoses are based on clinical symptoms. In addition, brain pathological lesions that could be characteristic for certain diagnosis can be found in different syndromes with cognitive and motor alterations related to elderly. As an example, one of the pathological characteristics of the Alzheimer's disease (AD), the senile plaques, can also be found in Parkinson's disease, although both pathological features have distinct components. In the same way, the presence of Lewy's bodies (consisted of α-synucleins) can be found in Parkinson's disease and also in another kind of dementia (dementia of Lewi's bodies). So, different types of neurodegenerative diseases may show similar histopathological characteristics.

From all the dementias, AD is surely one of the most devastating. The basic ultra-structural lesions of Alzheimer's disease are amyloid-β (Aβ) aggregates around the neuronal cell bodies and neurofibrillary tangles inside the cell bodies. Aβ peptide derives from amyloid precursor protein which starts an amyloid cascade leading to biochemical alterations that promote the aggregates. Inflammation and glial reaction follows those deposits and also alterations in calcium homeostasis, activation of the glycogen synthase kinase-3 (GSK3) α and β and finally inhibition of cell growth and neuronal death. Some recent works also describe a subsequent activation of caspases and apoptosis and also oxidative stress. Chemical shifts initiated by ultrastructural alterations in brains of AD patients leads to a significant loss in cholinergic neurons. In this way, concentration of the neurotransmitter acetylcholine and also function of cholinergic receptors (even muscarinic and nicotinic) are markedly decreased in AD, which has serious consequences to stability of long term potentiation (LTP) and, in this way, to maintenance of memory. Glutamate receptors, which are essential for LTP, are superactivated in this situation because of the low retrieval of glutamate by glial cells at the synaptic gap.

Parkinson's disease is the second more common neurodegenerative illness and is characterized by rest muscle tremor and rigidity. There are losses of domapinergic neurons in the substantia nigra and the presence of Lewy's bodies in those neurons. There is also loss in the density of presenilin, a transmembrane protein, alteration in glutamate release and alteration in intracellular calcium metabolism.

Other common dementias in the elderly include: Huntington's disease, where an error in DNA replication results in hyper excitability of glutamate transmission and neuronal death; Frontotemporal dementia, where mutations in the tau protein linked to chromosome 17 leads to neurofibrilary tangles inside neurons and glial cells with no senile plaques; Degenerative disease of Purkinje cells that produce antibodies for cdr2 cytoplasmatic protein, resulting in cellular death; Diffuse disease of Lewy's bodies, where these Lewy's bodies are widely expressed in subpopulations of cortical neurons and lead to the same alterations observed in Parkinson's disease.

Some researchers argued that neurodegenerative diseases characterized by abnormal deposits of proteins should be evaluated together with a continuum of symptoms and not only as individualized entities, once there are common chemical reactions for cell death in these illnesses. In addition, the modified genes in neurodegenerative diseases codify mutant proteins that lead to molecular and physiological abnormalities or discrete but progressive structural alterations that can disrupt the transmission in the neuronal circuitry, increasing the vulnerability of neuronal cells.

A better knowledge of the consequences of the chemical alterations for memory and other cognitive functions, in these diseases, may contribute to a more precise diagnosis and to the development of more specific and effective therapies. Suggested literature: Roberto Lent, Cem Bilhões de Neurônios, 2005; *J Alzheimer's Dis.* 2009;18(2):381-400.

Writing a Scientific Paper: theory and practice.

Y. S. Bakhle (Imperial College, UK)

Good scientific results deserve and need good communication. Scientific research has always depended on telling others about your results and you basing experiments on the results of others. So presenting your results in a form that makes them more readily acceptable to Journals and their readers is an essential part of advancing scientific knowledge.

This Course starts with a lecture on scientific writing which is the "Theory" followed by "practical" sessions. In these, MSS supplied by the "students" are discussed in some detail in terms of each component (Title, Abstract, Introduction, Figures), applying the principles and methods outlined in the lecture. I show the scripts marked up with the edits so it is clear what has been changed and then explain and comment on the edit (why was is necessary and how it is "corrected").

These practical sessions *must* be interactive. Students are en-

couraged and *need* to ask questions about the points raised, the stylistic and language editing and the editing of Figures and Tables. The MSS should be in an advanced state of preparation, i.e. ready to be submitted and some, on past occasions, have been submitted and already rejected. The latter class of MS together with the reviewers' comments can provide an excellent basis of discussing "what went wrong" with the MS. Even factual / data "errors and deficits" can be generated by inadequate explanation or description of experimental work. The topics of the MSS are not important in this context - good communication is as important for explaining ecstasy-induced hyperthermia as it is for relating CYP polymorphism to efficacy of anticancer drugs. The support of the British Pharmacological Society is gratefully acknowledged.

Quantitative and qualitative analysis of drug-receptor interactions.

André Sampaio Pupo (UNESP-Botucatu)

Pharmacologists are constantly required to present in understandable terms the effects of drugs on physiologic systems. This is usually accomplished by applying relatively simple mathematical transformations of drug's doses/concentrations and effects, and confronting them against theoretical backgrounds. The non compliance of the experimental findings to these theoretical backgrounds has led to important advances in the understanding of both the mechanisms of actions of drugs and the physiologic systems upon which the effects of these drugs are investigated. In this context, drugs are valuable tools to elucidate physiologic processes, provided that the effects of these drugs are appropriately interpreted and that all possible mechanisms of actions of these drugs are known. Therefore, misinterpretation of drugs effects or the incomplete knowledge of its mechanisms of actions may be deceiving.

The first class of this course will address theoretical and practical aspects of dose-response curves plotting, such as curve adjustment, requirements of dependent and independent variables, raw versus transformed data and its statistical analysis.

The second class of the course will discuss the actions of competitive antagonists focusing on the importance of the Schild analysis for the appropriate study of this class of drugs. Special attention will be dedicated to the fundamental criteria that must be fulfilled in the construction of Schild's plots and also to operational aspects of the calculation of the slope parameter. In addition, the pharmacological interpretation of slope values different from the predicted theoretical unity will be discussed in light of experimental results.

The final class of the course will focus on the functional analysis of dose-response curves in complex contexts where the pharmacological responses result from multiple mechanisms of actions and its consequences on the estimates of curves slopes and drug's potencies and efficacies.

The proteins kinase and their role in the cellular immune response.

Aristóbolo M. Silva (UFMG)

Protein kinases are enzymes that phosphorylate certain amino acid residues in specific proteins. They are key regulators of cell function by driving the activity, physical interaction and cellular localization of many proteins. The activity of protein kinases is tightly regulated by a number of molecular events such as the turning on or off by phosphorylation, and binding of activator proteins or small molecules. Protein kinases are involved in a myriad of cellular processes, including the immune cell function. In this module, I will present the basic concepts in the protein kinases such as the phosphorylation of amino acids and proteins by protein kinases, how does phosphorylation regulate activity of proteins, and how the domains within protein structures regulate cell function. Yet, considerable attention will be given to specific protein kinases or groups constituted by them whose activities will determine the major outcomes in the cellular immune response. Activation of immune cells in the immune response is triggered upon detection of stress stimuli that the organism receives. The outcome of these stimuli will be the activation of specific signaling pathways that control the expression regulation of inflammatory genes that are critical to cellular immune response. Therefore, protein kinases are essential cellular components required to modulate these pathways. Among these include the one which leads to the activation of the kinases responsible for nuclear factor kB (NF-kB) transcription factor activity regulation. This pathway involves the formation of NF-kB dimmers as a result of phosphorylation-induced proteolysis mediated by the kinase responsible for NF-kB activation, IkB kinase (IKK). Other important protein kinases constitute a group called a mitogen-activated protein kinases (MAPKs). MAPK pathway is classically composed by a cascade of three kinases: (1) a MAPK that is responsible for the effector functions, (2) the kinase that activates the MAPK by phosphorylation (MAP2K or MKK), and (3) the kinase that activates the MKK (MAP3K or MEKK), which provides specificity in response to cell surface receptors upon stress recognition. Within the MAPKs family, the stress-activated protein kinase (SAPK) group has been defined as a group of kinases that are activated by stimuli that cause cell stress such as inflammation. SAPKs control the expression of some proinflammatory genes such as those encoding cytokines. Finally, the function of a protein kinase with

a role in viral immune responses will also be presented. Recently, emerging role for this protein kinase has been observed in bacterial and parasitic infections. The double-stranded RNA dependent protein kinase PKR is a host defense enzyme whose expression is up-regulated in response to Interferons (IFNs) and during viral infections. Increased levels of PKR result in its activation by auto-phosphorylation, which in turn phosphorylates the alpha-subunit of eukaryotic translation initiation factor 2 (eIF2-alpha) resulting in the inhibition of global cellular protein synthesis. In summary, the basic concepts in the protein kinases and what is currently known about the kinases aforementioned in their overall contribution to the immune response will be discussed.

Pi3kinase and inflammation.
Remo de Castro Russo (UFMG)
Phosphorylated lipids are produced at cellular membranes during signaling events and contribute to the recruitment and activation of various signaling components. The role of phosphoinositide 3-kinase (PI3K) is to catalyze the production of phosphatidylinositol-3,4,5-trisphosphate in cell survival pathways, regulating gene expression, cell metabolism and highlighting cytoskeletal rearrangements. The PI3K pathway is implicated in human pathophysiologies including chronic inflammatory diseases and cancer; understanding the intricacies of this pathway may provide new direction for therapeutic intervention.
Based on their primary sequences, the PI3K family is divided into three classes, in which the class I PI3K has been studied most extensively and is the focus of this course. The expression patterns and mode of regulation of class II and III PI3Ks are less understood mechanisms of regulation and substrate specificities. Inside the class I PI3K, class IA subtypes are heterodimers that consist of a catalytic subunit (p110) and a regulatory subunit (p85). These subtypes are thought to be the major *in vivo* source of PIP3 upon activation of the receptors possessing protein– tyrosine kinase activity or the receptors coupling to Src-type protein–tyrosine kinases.
In mammals, there are multiple isoforms of class IA PI3K. Different genes encode class IA catalytic subunits, referred to as p110α, p110β and p1110δ, while other genes encode the associating regulatory subunits, referred to as p85, represented by five species (p85α, p85β, p55α, p55γ and p50α). p85 has two Src homology 2 (SH2) domains, which link the p85–p110 PI3K enzyme complex to tyrosine kinase signaling pathways. Class IA PI3K is activated by most receptors that trigger tyrosine kinase activity. In lymphocytes, this includes antigen receptors, co-stimulatory molecules, adhesion molecules, Toll-like receptors and cytokine receptors. By contrast, the class IB catalytic subunit p110γ binds to one of the two non-p85 regulatory subunits, called p101 and p84, and mediates PI3K activity upon G protein-coupled receptors (GPCRs) stimulation. Class IB PI3K is activated by GPCRs including chemokine receptors, bradykinin receptors and sphingosine-1-phosphate (S1P) receptors.
The acute phosphorylation of Phosphatidylinositol lipids at inositol ring D-3 position in response to cell stimulation by growth factors, hormones and chemokines sets in motion are a coordinated set of events leading to cell cycle entry, growth, migration and survival. How does lipid phosphorylation coordinate such complex behavior? Various signaling proteins, including the mitogen-transducing signal proteins (protein kinase C, phosphoinositide-dependent kinases, small G-proteins, MAP - mitogen activated protein- kinases), are activated either via their interaction with lipid products of PI3K as through PI3K-dependent phosphorylation of proteins, which have domains that specifically bind to D-3 phosphorylated phosphoinositides. These proteins are located in the cytosol of unstimulated cells, but in response to lipid phosphorylation, accumulate at the plasma membrane because of their ability to associate with the newly formed phosphoinositides. At the membrane, these proteins become activated and initiate various local responses, including polymerization of actin, assembly of signaling complexes and priming of protein kinase cascades.
Most PI3K subunits seem to have a broad tissue distribution, including endothelial cells, fibroblasts and tumor cells, with p110γ and p110δ being highly enriched in leukocytes, playing important roles in chemotaxis, antigen recognition, leukocyte activation and survival. Many chronic inflammatory diseases are associated with deregulated intracellular signal transduction pathways. Resultant pathogenic interactions between immune and stromal cells lead to changes in cell activation, proliferation, migratory capacity and cell survival, contributing to inflammation. Increasing efforts are now being made in the design of novel therapeutic compounds to interfere with signaling pathways in inflammatory diseases like rheumatoid arthritis, asthma and cancer. Studies with focus on PI3Ks are providing new insights into the mechanisms and the extent of their involvement in innate immunity and chronic inflammatory diseases, highlighting new potential targets for therapeutic intervention using PI3K inhibitors, opening up potentially opportunities for these drugs.

NFkB and IRF3/7 signaling pathway in the innate immunity.
Daniel Santos Mansur (UFMG)
Production of pro-inflammatory mediators such as cytokines and chemokines is intimately depen-

dant on certain transcription factors that are activated after the recognition of pathogens and danger signals. Here we will focus on the kinases involved in signal transduction and activation of the main transcription factors involved in initiating an immune response in an intracellular level: NF-κB and IRFs 3/7.

Recognition of micro-organisms and danger signals by pathogen recognition receptors (PRRs) leading to transcription of pro-inflammatory cytokines and chemokines and other genes is arguably one of the most important features of innate immunity and is responsible for immediate response to infection and orchestration of an efficient adaptative immune response. Playing a central role in this process is NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells). First related to B cells development, NF-κB is a heterodimer consisted of p50 and p65 that shuttles from cytoplasm to nucleus in its inactive form, bound to the repressor IκBα.

Let's use the example of one of the most studied PRRs, TLR4, to detail the activation process of NF-κB. LPS is a main component of gram-negative bacterial cell wall and it is the best described PAMP (pathogen associated molecular pattern) that activates TLR4. After TLR4 activation by LPS, mediated by CD14 and MD2, a complex of adaptor molecules is recruited to the plasma membrane, amongst them MyD88 that binds TLR4 through its TIR domain. This recruits IRAKs and the scaffold protein TRAF6 that ultimately activates the master kinase TAK1. TAK1 phosphorylates another kinase, IKKγ (NEMO) that on its turn phosphorylates IKKβ, in the same complex called IκB kinase (IKK) complex.

Activation of IKKβ through NEMO is considered the canonical pathway of NF-κB activation. IKKβ phosphorylates IκBα that is ubiquitinated, and hence degraded by 26S proteasome. The degradation of the repressor IκBα leads to the exposition of a NLS (nuclear localization signal) and leave NF-kB able to bind DNA and play its role in gene transcription.

Another important transcription factor involved in starting innate immunity is IRF3 (interferon regulatory factor 3). IRF3 is activated after the ligation of LPS into the CD14/MD-2/TLR4 as well, or after cytosolic recognition of nucleic acids. The differences from the NF-κB to IRF3 activation start on the adaptor that is recruited to the TIR domain after ligation of LPS to the receptor. TRIF and TRAM are adaptors that are engaged to TLR4 and recruit TRAF3 that complexes two main kinases, IKKepsilon and TBK-1, that are required for IRF3 phosphorylation, dimerization and translocation to the nucleus, where it will regulated transcription of many genes, amongst them type 1 interferons.

During cytosolic nucleic acid recognition different kinds of receptors are recruit and the pathways involved in signal transduction are being studied, but most of them, like RIG-I, that recognizes 5'3pRNA use IKKε or TBK-1 to activate gene transcription mediated through IRF3.

Nucleic acid recognition can engage other transcription factors depending on the cell that is being activated. Importantly, when dendritic cells recognize nucleic acid through TLR7/8 or 9, they are able to induce the dimerization, mediated by MyD88 and IRAKs, of IRF7, that is a potent transcriptional factor for type 1 interferon production.

Coordinated those two families of transcription factors are of great importance to immediate response to viruses and bacteria and to orchestration of an efficient adaptative immune response.

Symposia

Roles of GRKs and beta-arrestins activities in the regulation of β2 adrenergic signaling.

Jamil Assreuy (UFSC)

Introduction: Sepsis is a systemic inflammatory response resulting from the inability of the host to restrict local infection. From the cardiovascular point of view, septic shock is characterized by cardiac collapse and decreased peripheral resistance due to dilatation of systemic resistance vessels induced by nitric oxide (NO) production from inducible NO synthase (NOS-2). G protein–coupled receptor (GPCR) kinases (GRKs) are specific kinases interacting with GPCR proteins, inducing receptor phosphorylation and thereby desensitization of GPCR desensitization even in the agonist presence. Therefore, it is conceivable that increased expression of GRKs could increased adrenergic receptor desensitization and in turn reduces cardiovascular responses. Therefore, we hypothesized that the hyporesponsiveness observed in sepsis could result from signal receptor desensitization mediated by a NO-induced GRK increased activity. Methods: Female C57Bl/6 mice were submitted to cecal ligation and puncture (CLP). Vascular responsiveness was evaluated in endothelium-bearing aorta rings contracted with phenylephrine (1 μM). Cardiac responsiveness was evaluated in isolated contracted with isoproterenol (1 μM). Aortic and ventricle responsiveness was evaluated 6, 12 and 24 h after CLP surgery in the presence or absence of a selective NOS-2 inhibitor 1400W (100 μM). GRK2 expression was analyzed on heart and aorta harvested from sham and CLP treated or not with 1400W (1 mg/kg) 6, 12 and 24 h after CLP by immunofluorescence analysis. All procedures have been approved by the institutional Animal Ethics Committee (Proto-

col PP003/CEUA). Results: The vascular response to phenylephrine was significantly reduced in aorta rings from septic mice evaluated 6 (55% reduction compared to the response of sham-operated animals), 12 (57%) and 24 (78%) h after CLP. Incubation with 1400W reverted vascular hyporesponsiveness 6 and 12 h after CLP. The cardiac responsiveness to isoproterenol was significantly reduced in ventricles from septic mice evaluated 12 (73%) and 24 (88%) h after CLP. Conversely, the 1400W treatment prevented the cardiac hyporesponsiveness 12 (70% reduction compared to the response of sham-operated animals) and 24 (80%) h after CLP. Moreover, high expression of GRK2 was detected in aorta 6 (65%), 12 (70%) and 24 (88%) h, and heart of septic mice 12 (52%) and 24 (63%) h after CLP. The treatment of septic mice with 1400W reduced the GRK2 high expression on aorta (75%) and heart (79%). Finally, the pre and post-treatment with 1400W enhanced significantly the survival rate of the septic mice (55%). Discussion: Our findings show that NO, produced mainly by NOS-2 during sepsis seems to activate GRK2, which induces adrenergic receptor desensitization. Increased in the GRK expression is associated with impairment vascular response, contributing to severe cardiovascular hyporesponsiveness observed during septic shock. Moreover, NO synthesis inhibition improves output cardiovascular, and as consequence, enhances the survival of septic mice. Therefore, the results suggest that GRK2 could be a new potential target to sepsis pharmacotherapy. Financial support: CNPq, CAPES, FAPESC.

Therapeutic potential of blocking PI3Kg in inflammation.

Danielle G. Souza, Erica L. Martin, Fernando Q. Cunha, Emilio Hirsch, V. Marco Ranieri, Mauro M. Teixeira

Rationale: Sepsis is a leading cause of ICU-death, characterized by a systemic inflammatory response (SIRS) and bacterial infection, which can often induce multi-organ damage and failure. Leukocyte recruitment, required to limit bacterial spread, depends on PI3Kg signaling *in vitro*; however the role of this enzyme in polymicrobial sepsis has remained unclear. Objective: This study aimed to determine the specific role of the kinase activity of PI3Kg in the pathogenesis of sepsis and multi-organ damage. Methods: PI3Kg wild-type, knockout and kinase-dead mice were exposed to cecal ligation and perforation-induced sepsis and assessed for survival, pulmonary, hepatic and cardiovascular damage, coagulation derangements, systemic inflammation, bacterial spread and neutrophil recruitment. Additionally, wild-type mice were treated either before or after the onset of sepsis with a PI3Kg inhibitor and assessed for survival, neutrophil recruitment and bacterial spread. Measurements and Main Results: Both genetic and pharmaceutical PI3K γ kinase inhibition significantly improved survival, reduced multiorgan damage and limited bacterial decompartmentalization, while modestly effecting systemic inflammation (SIRS). Protection resulted from both neutrophil-independent mechanisms, involving improved cardiovascular function, and neutrophil-dependent mechanisms, through reduced susceptibility to neutrophil migration failure during severe sepsis by maintaining neutrophil surface expression of the chemokine receptor, CXCR2. Furthermore, PI3Kg pharmacological inhibition significantly decreased mortality, improved neutrophil migration and bacterial control, even when administered during established septic shock. Conclusions: This study establishes PI3Kg as a key molecule in the pathogenesis of septic infection and the transition from SIRS to organ damage, and identifies it as a novel possible therapeutic target.

Antioxidants as anti-inflammatory agents.

Juliano Ferreira (UFSM)

A vast amount of circumstantial evidence implicates reactive oxygen species (ROS) as mediators of inflammation. ROS activate cellular redox-sensitive proteins in target cells involved in mediating inflammatory responses, such as the transcription factor NF-kB that regulates the expression of numerous genes that encode pro-inflammatory molecules, such as some cytokines, adhesion molecules and cyclooxygenase-2. Antioxidants can scavenger ROS and have long been advocated for the treatment of inflammatory diseases. However, the value of antioxidants in the prevention and treatment of such diseases has been questioned. We will review the anti-inflammatory effect of antioxidants in pre-clinical models of inflammation and in clinical setting, focusing their potential use in arthritis treatment.

Can antioxidants prevent dyskinesia?

Roberto Frussa-Filho (UNIFESP)

The clinical symptoms, the epidemiology and the risk factors of tardive dyskinesia will be briefly characterized. Afterwards, the first pathophysiological hypothesis of the disease – the dopaminergic supersensitivity hypothesis - will be discussed. The pitfalls of the supersensitivity hypothesis and the proposal of the oxidative stress pathophysiological hypothesis will be detailed and followed by both preclinical and clinical evidence supporting the later hypothesis. Finally the potential use of antioxidant agents to prevent tardive dyskinesia and the antioxidant properties of atypical neuroleptics will be discussed.

Quimiocinas e inflamação no sistema nervoso central.

Antonio Lúcio Teixeira (UFMG)

As quimiocinas constituem uma grande família de citocinas responsáveis pelo recrutamento de leucócitos, incluindo a migração dos mesmos para locais de inflamação tecidual a partir da

circulação sanguínea. As quimiocinas são polipeptídios de 8 a 12 kDa, sendo classificadas nas subfamílias XC, CC, CXC e CX3C conforme o número e a localização dos resíduos de cisteína N-terminais. Nosso grupo de pesquisa vem investigando ativamente os níveis circulantes de quimiocinas e outras moléculas relacionadas com a resposta imune/inflamatória em várias doenças cerebrais humanas, procurando correlacioná-las com parâmetros clínicos. Essa estratégia, além de contribuir para o estudo do envolvimento de moléculas inflamatórias nessas doenças, tem o potencial de identificar candidatos a biomarcadores diagnósticos e/ou prognósticos.

Estudamos os níveis séricos e no líquor de quimiocinas em crianças portadoras de coréia de Sydenham, a manifestação neurológica da febre reumática. Observamos que as quimiocinas MIG/CXCL9 e IP-10/CXCL10, envolvidas no recrutamento de linfócitos de perfil predominantemente Th1, encontravam-se elevadas no soro de pacientes com coréia aguda em relação a controles assintomáticos e pacientes com a forma persistente da coréia (Teixeira et al., 2004). Esses resultados corroboraram a natureza imune-mediada da coréia reumática, questionando se processos autoimunes estariam relacionados à persistência dos sintomas além da fase aguda. Investigamos o envolvimento das quimiocinas na esclerose múltipla, doença inflamatória desmielinizante do sistema nervoso central. De acordo com dados da literatura internacional, observamos que os pacientes em surto (ou seja, com sintomas agudos indicativos da presença de processo inflamatório em atividade) apresentavam simultaneamente níveis elevados de IP-10/CXCL10 e níveis diminuídos de MCP-1/CCL2 no líquor. Demonstramos ainda que, após o tratamento dos pacientes em surto com pulsoterapia com metilprednisolona, ocorria uma queda dos níveis de IP-10/CXCL10 e uma elevação de MCP-1/CCL2 no líquor (Moreira et al., 2006). Sugerimos que essas duas quimiocinas poderiam ser utilizadas como marcadores da atividade ou surto da doença. Posteriormente, passamos a explorar o potencial das quimiocinas como biomarcadores em doenças infecto-parasitárias. Demonstramos, por exemplo, que pacientes com esquistossomose apresentavam níveis séricos elevados das quimiocinas MCP-1/CCL2, MIP-1α/CCL3, eotaxina/CCL11 e eotaxina2/CCL24, todas envolvidas no recrutamento de células comprometidas com resposta de perfil predominantemente Th2. Quando o líquor de pacientes com esquistossomose medular foi analisado, não se observou aumento dos níveis de quimiocinas, contrariamente ao encontrado, por exemplo, na mielopatia associada ao HTLV-1 (HAM/TSP) (Sousa-Pereira et al., 2006). Interessantemente, observamos que os pacientes portadores de HAM/TSP apresentavam níveis séricos muito elevados das quimiocinas MIG/CXCL9 e IP-10/CXCL10 (envolvidas na resposta Th1) em relação às pacientes infectados com o vírus HTLV-1 sem a mielopatia (Guerreiro et al., 2006). Em contraste, os pacientes com HAM/TSP exibiam níveis circulantes menores de quimiocinas relacionadas ao recrutamento de células de perfil predominantemente Th2, como o MCP-1/CCL2. Em conjunto, esses dados sugerem que as quimiocinas poderiam auxiliar o diagnóstico diferencial das mielopatias e, eventualmente, predizer a conversão de estado de infecção pelo HTLV para HAM/TSP.

Em conclusão, fenômenos inflamatórios estão direta ou indiretamente relacionados com uma série de doenças neurológicas. Assim, determinadas moléculas inflamatórias têm o potencial de serem relevantes biomarcadores dessas doenças. Referências bibliográficas: Guerreiro JB, Santos SB, Morgan DJ, Porto AF, Muniz AL, Ho JL, Teixeira AL, Teixeira MM, Carvalho EM. Levels of serum chemokines discriminate clinical myelopathy associated with human T lymphotropic virus type 1 (HTLV-1)/tropical spastic paraparesis (HAM/TSP) disease from HTLV-1 carrier state. Clin Exp Immunol. 2006; 145: 296-301. Moreira MA, Tilbery CP, Monteiro LP, Teixeira MM, Teixeira AL. Effect of the treatment with methylprednisolone on the cerebrospinal fluid and serum levels of CCL2 and CXCL10 chemokines in patients with active multiple sclerosis. Acta *Neurol Scand.* 2006;114:109-113.

Sousa-Pereira SR, Teixeira AL, Silva LC, Souza AL, Antunes CM, Teixeira MM, Lambertucci JR. Serum and cerebral spinal fluid levels of chemokines and Th2 cytokines in *Schistosoma mansoni* myeloradiculopathy. Parasite Immunol. 2006; 28: 473-478. Teixeira AL, Cardoso F, Souza AL, Teixeira MM. Increased serum concentrations of monokine induced by interferon-gamma/CXCL9 and interferon-gamma-inducible protein 10/CXCL-10 in Sydenham's chorea patients. J Neuroimmunol. 2004;150:157-162.

Neurochemical and behavioral changes after systemic inflammation and sepsis.

Felipe dal Pizzol (UNESC)

It has been demonstrated that septic present both acute and long-term central nervous system dysfunction, including alterations in memory, attention, concentration, and global loss of the cognitive function. However, the mechanisms associated to these alterations are still unclear. Using an animal model of cecal ligation and perforation (CLP) we demonstrated several behavioral changes, mainly memory impairment and depression-like symptoms, in rats. In addition, it was observed that animals submitted to CLP presented decreased mitochondrial respiratory chain activity associated with short-term oxidative

damage. In addition, during sepsis brain-produced cytokines and chemokines is an early event and seemed to participate both on CNS dysfunction and blood brain barrier permeability alterations, leading to neuronal death. All these alterations seemed to be implicated in the long-term behavioral changes and neurotransmitters alterations observed in sepsis animal models and septic humans. Supported by CNPq, INCT-Translacional Medicina, FAPESC.

New vistas for an old friend: Lipoxin A4 as an allosteric endocannabinoid in the brain.

Fabrício Pamplona (UFSC)

Lipoxins and endocannabinoids are endogenous eicosanoids that are released on demand following neuronal stimulation or injury. Lipoxin A_4 (LXA_4) activates ALX receptors and has an important role in the resolution of inflammation, but information about its effects on the central nervous system is scarce. The available evidence suggests that LXA_4 may influence brain function in a cannabinoid-like fashion, which would imply participation of CB_1 cannabinoid receptors. Although endocannabinoids and lipoxins share structural and functional similarities, the pharmacological relationship between them has not yet been described. Hence, this study aimed to investigate the participation of the endocannabinoid system in the central effects of LXA_4.

As an initial behavioral characterization, mice were injected i.c.v. with LXA_4 or control evaluated in the cannabinoid tetrad test (catalepsy, locomotion, analgesia and rectal temperature), considered predictive for cannabimimetic activity. These responses were further investigated by injecting the CB_1 receptors antagonist SR141716A or the ALX receptor antagonist BOC-2 before i.c.v. injection of LXA_4. LXA_4 induced catalepsy, hypolocomotion, analgesia and hypothermia in mice. These effects were antagonized by the CB_1 receptor antagonist SR141716A, but not by the ALX receptor antagonist BOC-2. The role of CB_1 receptors on LXA_4 effects was further confirmed in CB1 knockout mice.

Binding of LXA_4 to CB_1 receptors was tested in a competitive binding assay against [^{3}H]SR141716A in mouse brain membranes. The putative pharmacological interaction between LXA_4 and the endocannabinoids was addressed by co-injecting sub-effective doses of anandamide (AEA) or 2-araquidonilglicerol (2-AG) and LXA_4. Possible effects of LXA_4 on degradation of endocannabinoids were assessed by *in vitro* assays of the metabolic enzymes of AEA e 2-AG. LXA_4 virtually did not inhibit the binding of [^{3}H]SR141716A, but enhanced the inhibition of [^{3}H]SR141716A binding by AEA. LXA_4 potentiated the cataleptic effects of AEA, but not of 2-AG. There was no effect of LXA_4 on the endocannabinoid-degrading enzymes FAAH and MAGL.

The present results suggest that LXA_4 exerts central effects via CB_1 cannabinoid receptors and interacts positively with the endocannabinoid AEA. While we did not find any effect of LXA_4 on endocannabinoid metabolism, this study brings evidence that LXA_4 enhances the affinity of AEA for CB_1 receptors through a mechanism of positive allosteric modulation. This is a pioneer finding on the endocannabinoid pharmacology.

Stress and inflammation.

Moises Evandro Bauer (PUCRS)

Cytokines have been implicated in the pathophysiology of a series of neuropsychiatric disorders, including major depression, bipolar disorder, schizophrenia, dementia and post-traumatic stress disorder (PTSD). There is growing evidence supporting the link between traumatic stress or depression to a pro-inflammatory profile, with increasing serum concentrations of C-reactive protein, TNF-α, IFN-γ, IL-1β and IL-6. Recently, we observed that depressed patients with PTSD symptoms had poor memory performance, negatively related to lower brain-derived neurotrophic factor (BDNF) plasma levels. Patients had also significant lower salivary cortisol and DHEAS across the day in parallel with blunted T-cell proliferation and lower IL-2 levels than controls. Patients had also lower RANTES but higher sTNF-R2 levels when compared to controls. The involvement of cytokines in the pathophysiology of depression may involve changes of synaptic transmission, especially in hippocampal-amygdala structures, influencing various aspects of memory related to traumatic memories. The phenomenon of low-grade inflammation could be also involved with increased morbidity in depression. Peripheral administration of pro-inflammatory cytokines or increased levels observed during infections has been associated with changes in patient's behavior known as "sickness behavior". The patient becomes irritable and exhibits increased sleep, depression, fatigue, decreased appetite and sexual drive. Pro-inflammatory cytokines have been implicated in this phenomenon. For instance, depression has been reported in up to 60% of patients with hepatitis C under treatment with IFN-α. The activity of the HPA axis is also enhanced by pro-inflammatory cytokines and may further contribute to pathophysiology of depression.

The impact of stress on body weight gain.

Ruth B.S. Harris, Department of Physiology, Medical College of Georgia.

Stress induces a complex array of physiological, neurological and behavioral responses that allow an animal to respond to a change in its environment. These responses are initiated by corticotrophin releasing factor (CRF) and the urocortins. There are two major subtypes of CRF receptors (CRFR1 and CRFR2) that each mediate specific aspects of

the stress response. Stress may cause weight gain or weight loss, dependent upon the type and severity of stress and an individual's perception of the degree of stress. Humans may gain weight in response to chronic daily stress, but will lose weight in response to a traumatic event. Hamsters gain body fat in following exposure to social stress, whereas food intake and growth are decreased in rats, mice and monkeys living in a stressful environment. We have investigated a rat model in which 3 hours of restraint stress on each of 3 consecutive days results in a chronic down-regulation of body weight. The rats lose weight on the days of restraint and then gain weight at the same rate as their non-stressed controls, but do not compensate for the stress-induced weight loss. Five days after the end of restraint the stressed rats weigh approximately 10-15% less than controls and this weight difference is maintained for at least 90 days. Third ventricle infusions of a CRFR1 antagonist, antalarmin, immediately before restraint do not prevent weight loss on the days of restraint or stress-induced activation of the HPA axis, but do prevent the long-term down-regulation of body weight. There is no evidence of chronic activation of the CRF system in the post-stress period, but rats that have been restrained are hyper-responsive to subsequent mild stressors. We are investigating whether this is associated with a change in threshold or an exaggerated response to stress and whether the increased sensitivity to daily events contributes to the sustained reduction in body weight. (Supported by NIMH grant MH068281)

High- or low-salt diet from weaning to adulthood: effect on body weight, **food intake and energy balance in rats**.

Michella S. Coelho[1], Mariana D. Passadore[2], Alessandra L. Gasparetti[3], Theo Bibancos[4], Patrícia O. Prada[3], Luciene L. Furukawa[1], Luzia N.S. Furukawa[1], Rosa T. Fukui[1], Dulce E. Casarini[e], Mario J.A. Saad[3], Jacqueline Luz[2], Silvana Chiavegatto[4], Miriam S. Dolnikoff[1], Joel C. Heimann[1]. [1]FM-USP – Nephrology, [2]UNIFESP – Physiology [3]FCM-UNICAMP [4]IPq-USP/InCor-HC-FMUSP, [5]UNIFESP – Internal Medicine

Objective: To get some additional insight on the mechanisms of the effect of salt intake on body weight. Design and methods: Rats were fed a low (LSD), normal (NSD), or high (HSD) salt diet. In a first set, body weight, tail-cuff blood pressure, fasting plasma thyroid-stimulating hormone, triiodothyronine, L-thyroxine, glucose, insulin, and angiotensin II were measured. Angiotensin II content was determined in white and brown adipose tissues. Uncoupling protein 1 expression was measured in brown adipose tissue. In a second set, body weight, food intake, energy balance, and plasma leptin were determined. In a third set of rats, motor activity and body weight were evaluated. Results: Blood pressure increased on HSD. Body weight was similar among groups at weaning, but during adulthood it was lower on HSD and higher on LSD. Food intake, L-thyroxine concentration, uncoupling protein 1 expression and energy expenditure were higher in HSD rats, while non-fasting leptin concentration was lower in these groups compared to NSD and LSD animals. Plasma thyroid-stimulating hormone decreased on both HSD and LSD while plasma glucose and insulin were elevated only on LSD. A decrease in plasma angiotensin II was observed in HSD rats. On LSD, an increase in brown adipose tissue angiotensin II content was associated to decreased uncoupling protein 1 expression and energy expenditure. In this group, a low angiotensin II content in white adipose tissue was also found. Motor activity was not influenced by the dietary salt content. Conclusions: Chronic alteration in salt intake is associated with changes in body weight, food intake, hormonal profile, and energy expenditure and tissue angiotensin II content.

Hypertension, **kidney disease**, **and cardiovascular outcomes in childhood: is there a role for birth weight**?

Franco MCP and Sesso R. (UNIFESP)

Objectives: Low birth weight due to intrauterine growth retardation may be a risk factor for hypertension, renal impairment and cardiovascular disease in the adult life. Our purpose was to investigate whether alterations in C-reactive protein, homocysteine, leptin and NO are present in small for gestational age children and to determine if the levels of these plasma biomarkers are associated with birth weight, vascular function and blood pressure. In addition, we investigated levels of both cystatin C and creatinine, and to evaluate whether there was an association between reduced GFR estimated by these markers and low birth weight. Methods: In this cross-sectional study the concentrations of leptin, homocysteine, C-reactive protein, cystatin, creatinine and NO were measured in 71 children between 8 and 13 years of age. Results: Leptin and homocysteine levels were significantly elevated in children born small for gestational age compared to those with appropriate birth weight. Nevertheless, NO concentration was significantly reduced in small birth weight children and the levels of C-reactive protein remained unchanged. There was a significant association between the circulating levels of both NO and homocysteine with vascular function as well as with blood pressure levels in our population. In addition, no differences were found for serum creatinine or GFRcr levels in the birth weight quartiles. There was a significant linear trend of increasing cystatin C (decreasing GFRcys) in the

lower birth weight quartile groups. Systolic blood pressure correlated with plasma levels of cystatin C (r = 0.311, p=0.008) and GFRcys (r = - 0.261, p=0.028). Conclusion: As both homocysteine and NO are associated with a risk of cardiovascular disease, it is possible that part of the association of low birth weight with elevated risk for vascular and metabolic disease in later life is mediated by perturbation in pathways for these biomarkers. Moreover, lower birth weight is associated with reduced renal function in 10 yr. old children with adequate gestational age.

Vascular reactivity of femoral arteries from diabetic trained rats.

Angelina Zanesco (UNESP-Rio Claro)

The number of diabetic individuals is increasing in the world due to a number of factors such as population growth, increase of lifespan and elderly population as well as increased prevalence of obesity and sedentary lifestyle. Evidences have pointed out that a massive production of ROS is the basis to macro- and micro-vascular disease in diabetic patients that contributes to the loss of endothelial function which is the critical phase of all the vascular diabetes mellitus (DM) complications. Therefore, overproduction of ROS by oxidative stress in response to hyperglycemia in DM leads to severe endothelium dysfunction. A healthy lifestyle has been strongly associated with the practice of regular physical activity. Evidences have shown that physically active subjects have more longevity with reduction of morbidity and mortality. Physical exercise prevents or reduces the deleterious effects of pathological conditions such as arterial hypertension, coronary artery disease, atherosclerosis, and diabetes mellitus. This work was to evaluate the effect of exercise training and aminoguanidine treatment (AG) on the femoral vascular responsiveness from diabetic rats. Methods: Male Wistar rats (180-200 g) were divided into: sedentary (C/SD); diabetic (DB/SD), diabetic trained (DB/TR), diabetic treated with AG (DB/SD-AG) and diabetic trained treated with AG (DB/TR-AG). Diabetes mellitus was induced by streptozotocin (60mg/Kg, i.p.) and run training (RT) consisted of 5 days/week, 60 minutes, 0.9 km/h and 0% grade during 8 weeks. The AG (1g/L) treatment was administrated in the drinking water. Concentration-response curves for acetylcholine (ACh), sodium nitroprusside (SNP) cromakalin (CRO), phenylephrine (PHE) and thromboxane A_2 analogue (U46619) were obtained in femoral artery. Blood glucose, glycated hemoglobin and β-ketone levels were measured. Results: Glucose was reduced in RT groups (8%) whereas glycated hemoglobin and β-ketone levels were not affected compared to DB/SD group. Both potency and Emax for ACh were decreased in DB/SD (6.0±0.1; 62±2%) compared to C/SD (6.8±0.09; 76±2%) while RT or AG treatment partly restored this reduction in DB/TR (6.4±0.06; 68±3%) and DB/SD-AG (6.4±0.07; 69±4%). Combination of RT and AG treatment completely reversed this reduction (6.7±0.02; 74±3%). Neither potency nor Emax were modified for SNP in all groups. Potency for CRO was decreased in all DB groups that it was not restored by RT or AG treatment. No changes were observed for PHE and U46619 in all groups. Conclusion: Endothelium-dependent relaxing responses in femoral artery were restored by combination of RT and AG treatment.

Financial Support: FAPESP

Potential application of flavonoids in the therapeutics of diabetes mellitus

Gabriel Forato Anhê (UNICAMP)

Aim/hypothesis: A low grade inflammatory response probably accounts for insulin resistance in Type 2 Diabetes Mellitus. Quercetin, a potent anti-inflammatory flavonoid, was described to increase glucose tolerance in streptozotocin-induced diabetic rodents but its benefits to insulin resistance have yielded contradictory results. The present study aimed to determine whether intraperitoneal quercetin treatment increases insulin sensitivity in ob/ob mice and reduces inflammatory response in the skeletal muscle. Methods: L6 myotubes were treated with palmitate or TNFα plus quercetin. Obese ob/ob mice were treated with quercetin for 10 weeks. Cells and muscles were processed for mRNA quantification of GLUT4, TNFα and iNOS and phosphorylation of JNK and IκK. Myotubes were assayed for glucose uptake and nuclear NF-κB translocation. Chromatin immunoprecipitation assessed NF-κB p50 binding to Slc2a4 promoter. Results: Quercetin increased whole body insulin sensitivity and increased GLUT4 expression and decreased JNK phosphorylation, and TNFα and iNOS expression in skeletal muscle. L6 myotubes exposure to quercetin suppressed palmitate-induced upregulation of TNFα and iNOS and restored normal levels of GLUT4. In parallel, quercetin suppressed both palmitate- and TNFα-induced reduction of glucose uptake in myotubes. Nuclear accumulation of NF-κB in myotubes and binding of NF-κB p50 to Slc2a4 promoter in muscle of ob/ob mice were also reduced by quercetin. Conclusions/interpretation: We demonstrate that intraperitoneal quercetin decreases the inflammatory status in skeletal muscle of obese mice, which was mimicked in L6 myotubes. These effects were followed by an improvement in insulin action, suggesting that quercetin is a putative strategy to manage insulin resistance in obesity.

Novel endogenous peptide agonists of cannabinoid receptors.

Lakshmi A. Devi, Prahlad T. Ram[Δ], Avi Ma'ayan, and Ivone Gomes Department of Pharmacology & Systems Therapeutics, Mount Sinai School of Medicine, New York, USA, [Δ]M. D, Anderson Cancer Center, Texas, U.S.A.

Hemopressin, a 9-residue α-hemoglobin-derived peptide, was previously reported to function as a CB_1 cannabinoid receptor-selective ligand (Heimann et al., PNAS, 194:20588, 2007). Recent peptidomics analyses of mouse brain extracts using mass spectrometry identified N-terminally extended forms of hemopressin containing either three (Arg-Val-Asp) or two (Val-Asp) additional amino acids. Characterization of these 'longer hemopressins' revealed that they function as CB_1 cannabinoid receptor agonists, in contrast to the 9-residue hemopressin that functions as an antagonist (Gomes et al., FASEB J. 23:3020, 2009). We have recently found that longer hemopressins activate a signal transduction pathway that is distinct from that of classical CB_1R agonists. For example, the longer hemopressins activate G_{alphai} independent signaling leading to a robust mobilization of intracellular Ca^{+2} levels whereas the classic CB_1R agonists activate G_{alphai}-mediated signaling leading to G-protein activation. To further explore this ligand-directed signaling, we used a combination of reverse phase protein array and graph theory- inspired network analysis. Data from these analyses confirm the notion that longer hemopressins activate a signaling network distinct from that of classic ligands. Further characterization of the longer hemopressin-activated signaling pathway revealed that a number of proteins involved in neurogenesis and neuronal survival are selectively activated indicating that this ligand-directed signaling expands the functional repertoire of CB_1R. Taken together, these results suggest that CB_1 receptors are involved in the integration of signals from both lipid- and peptide-derived endocannabinoids. Furthermore, the fact that longer hemopressins possess unique agonistic activity at CB_1 receptors provides additional tools to understand how the endocannabinoid system is modulated as well as novel candidates to be developed as therapeutics in the treatment of pathologies involving cannabinoid receptors. Supported by NIH grants DA01952; DA08863 (to LAD) and GM071558 to (to AM and LAD).

Therapeutic potential and mechanisms of non-psychotropic phytocannabinoids.

Francisco Silveira Guimarães (USP)

Δ^9-tetrahydrocannabinol (Δ^9-THC) is usually recognized as the major component of *Cannabis sativa*, responsible for its behavioral and physiological effects. However, the Cannabis plant contains several other cannabinoids with potential therapeutic effects, including Cannabidiol (CBD). Unlike Δ^9-THC, CBD is devoid of the typical psychotomimetic effects of the plant. Systemic administration of CBD can produce several neuropharmacological effects such as anticonvulsant, neuroprotective, antipsychotic and anxiolytic. Antipsychotic effects have been shown in animal models and in pilot studies with schizophrenic or Parkinson patients with L-Dopa-induced psychosis. Regarding the anxiolytic properties of CBD, they have been demonstrated by several preclinical studies which employed animal models such as the Vogel conflict test, the elevated plus- and T-maze, aversive electrical stimulation and conditioned emotional response. Similar to the data obtained in animal models, results from studies in healthy volunteers strongly suggested that CBD has an anxiolytic action. Moreover, CBD is able to attenuate the anxiogenic effects induced by high doses of Δ^9-THC in humans. We have recently shown that CBD also possess antidepressant and anticompulsive behavior properties. The mechanisms of these effects remain poorly understood due to the multiple pharmacological effects induced by CBD. Although the mechanisms of the antipsychotic effects are unknown, CBD produces a behavioral, clinical and cFos expression profile akin to atypical rather than typical antipsychotics. Recent results obtained in our laboratory suggest that the anxiolytic and antidepressant effects of CBD are mediated by activation of 5HT1A receptors in brain areas such as the dorsolateral periaqueductal gray, bed nucleus of the stria terminalis, hippocampus and the medial prefrontal cortex. However, the anticompulsive effects of CBD seem to be mediated by facilitation of endocannabinoid-mediated neurotransmission. Financial support: FAPESP, CNPq, CAPES

Hemopressin: a new target for drug development.

Camila Squarzoni Dale (IEP-HSL)

Studies on cannabinoid receptors (CB) have raised a new perspective about the therapeutical potential of those receptors on different pathologies such as those with painful origin. In this aspect it has been demonstrated that agonists and antagonists of CB inhibit experimental pain in different models of nociception evaluation. Hemopressin (PVNFKFLSH) is a nonapeptide derived from the α_1 chain of hemoglobin (95-103 fragments) which acts as an inverse agonist of CB1 receptors modulating the signaling mediated by this receptor. Data obtained by our group demonstrates that hemopressin induces antinociception in acute and chronic experimental models. Hemopressin acts by inhibiting nociceptive activation at spinal level, directly on sensory neurons and involves peripheral CB1 receptors on acute models of nociception evaluation. More

interestingly, hemopressin is effective in inhibiting pain when administered by either intrathecal, intraplantar, or oral routes underscoring its therapeutic potential without affecting locomotors activity or sleeping time of animals indicating and absence of motor abnormalities or sedative effect. More recently we observed that hemopressin does not interfere with swimming time of animals demonstrating that hemopressin does not despair any depressant effects on Central Nervous System. These results represent a demonstration of a peptide ligand for CB1 cannabinoid receptors that also exhibits analgesic properties. These findings are likely to have a profound impact on the development of novel therapeutics targeting CB1 receptors.

Inverse agonism in G-protein coupled receptors (GPCRs) seen in light of classical mechanisms of receptor activation.

Laerte Oliveira (UNIFESP)

GPCRs may be activated constitutionally or by agonist binding thus becoming apt for signal transduction across their seven-trans-membrane (7TM) structure. Initiated at the extracellular side of the membrane, the signal can attain the receptor cytosolic domains thereby activating the G-protein system, the phosphorylation and the internalization of receptors. In both constitutional and agonist-mediated mechanisms, activation seems to result from an expansion of the receptor 7TM structure, so extensively that its cytosolic ends become accessible for coupling G-alpha chains and protein kinases. This theory has now been compellingly challenged by recent studies on mechanisms of action for inverse agonism. Identified by their action reverting the constitutive activation of receptors, inverse agonists have recently been shown to bind at the receptor agonist site at the same residue side-chains involved in agonist binding or even in agonist-mediated activation. In fact, inverse agonists should primarily be competitive antagonists of receptors but special features were added to these molecules transforming them into inverse agonists. Consistent interpretation of these findings may be hardly attained considering that the mechanism of GPCR activation is assumed to be generalized all over the receptor structure. Would the inverse agonism be also a reversion of this widespread effect or a parallel event simply annulling the activation? Thus, other hypotheses stressing the importance of interactions localized in the agonist site surroundings to trigger receptor activation, seem useful for discussion of these problems. These are derived from finding such as: (1) only the retinal-free opsin, with an extracellularly-located Lys-to-Glu mutation can activate transducin; (2) special features of agonists as aromatic rings may control the activation of GPCRs by binding of these molecules at the specific site.

Computational chemistry underpinning carbohydrate drug discovery.

Ivone Carvalho (USP)

Computational approaches have been progressively incorporated into the drug discovery process since several in silico methods are available, such as docking, pharmacophore modeling, QSAR (Quantitative Structure-Activity Relationship) and virtual screening. Computational chemistry and informatics can be used to integrate workflow and dataflow for optimum effectiveness in achieving project goals. This integration makes possible fast iterative virtual screening, to effectively prioritize targeted synthesis and screening efforts. In this context, the importance of selecting hit compounds with an appropriate scaffold is crucial for the successful in lead-optimization phase during drug discovery. The ideal scaffold should be both chemically and biologically stable and contain rigidity to enable the molecule to maintain a controlled three-dimensional presentation of pharmacophores. The advantage of carbohydrates is that they provide a series of scaffolds, as well as mediate many biological processes (cell adhesion, differentiation and growth, signal transduction, protozoa, bacterial and virus infections, and immune response). Amongst the large array of enzymes as therapeutic targets, our group has focused the exploration of TcTS (*Trypanosoma cruzi Trans*-Sialidase) and α-glucosidase enzymes. Concerning TcTS studies, short glycopeptide fragments based on *T. cruzi* mucin sequences (building blocks), potential drugs based on 1,4-disubstituted 1,2,3-triazole derivatives of galactose and cyclic pseudo-galactooligosaccharides were synthesized and tested, giving promising results. On the other hand, for studies involving α-glucosidase, a 3D model was built and validated, and further used to perform docking simulations. In addition, pharmacophore modeling and molecular interaction fields were carried out in order to establish the most relevant structural features of both enzyme and inhibitors to drive the synthesis of more active compounds.

Structure-based discovery of novel anti-inflammatory protein kinase inhibitors.

Carlos Alberto Manssour Fraga (UFRJ)

Abstract: NF-κB is a member of a family of cellular transcription factors that are implicated in the inducible expression of various genes involved in immune responses, inflammation, cell survival and cancer. In unstimulated cells, NF-κB is kept in the cytoplasm through interaction with IκB inhibitory proteins. In response to specific external stimuli, including proinflammatory cytokines, viral infection, oxidants, phorbol esters and ultraviolet irradiation, the IκB component of the complex is phosphorylated and degraded, resulting in translocation of NF-κB into the nucleus and the in-

duction of target gene transcription. Two protein kinases, IKK-α and IKK-β, phosphorylate IκB proteins and represent a convergence point for most signal transduction pathways leading to NF-κB activation. IKK-β is a key regulator of keratinocyte and epidermal differentiation and suppresses skin cancer, being implicated in some of the anti-inflammatory properties of commercial drugs, such as aspirin and salicylates. These pharmacological effects indicate that an inhibitor of IKK-β could effectively treat autoimmune and inflammatory disorders such as rheumatoid arthritis, lupus, Crohn's disease and multiple sclerosis.

The discovery and development of small molecule IKK-β modulators is a significant area of research, and several classes of inhibitors have been previously reported. The majority of these inhibitors share a core heterocycle that mimics the adenosine ring of ATP, promoting additional interactions with the target protein.

In this study, we describe the rational design, molecular modeling and pharmacological profile of (*E*)-*N*-(4-nitrobenzylidene)-2-naphthohydrazide (LASSBio-1524), a novel small molecule inhibitor of IκB Kinase-β. The design based on the IKK-β active site, and a *privileged structure* template yielded a novel IKK-β inhibitor scaffold with significant selectivity over IKK-α and CHK2, as assessed by a robust kinase assay. For a better understanding of the structural requirements of IKK-β inhibition, molecular dynamics simulations of staurosporine and LASSBio-1524 were performed. The NAH derivative LASSBio-1524 was able to suppress arachidonic acid-induced edema formation in a dose-dependent manner, demonstrating an in vivo anti-inflammatory effect. The molecular architecture of this novel, low-molecular weight IKK-β inhibitor is encouraging for further lead optimization toward the development of innovative anti-inflammatory drug candidates.

Financial support: This work was supported by grants and fellowships from INCT-INOFAR (573.564/2008-6), CNPq, FAPERJ and CAPES.

Atrofia muscular na Insuficiência cardíaca: efeito do treinamento físico aeróbico

Patricia Chakur Brum EEFE-USP – Biodinâmica do Movimento do Corpo Humano

Apesar das alterações no tecido cardíaco serem causais e principais no desenvolvimento da insuficiência cardíaca (IC), vários estudos tem demonstrado que a limitação da capacidade funcional perante o agravamento da IC não está somente relacionada ao grau de disfunção ventricular. A intolerância ao esforço físico, que é amplamente observada em portadores de IC correlaciona-se principalmente às alterações morfo-funcionais da musculatura esquelética, que contribuem para a antecipação da fadiga nesses indivíduos. Dentre essas alterações musculares observa-se: a) redução da capacidade oxidativa, b) mudanças na composição dos tipos de fibras musculares em direção as fibras glicolíticas de contração rápida e c) atrofia muscular. Além disso, a perda excessiva de massa muscular (caquexia) conjuntamente com o consumo de oxigênio de pico em pacientes com IC grave eram preditores independentes de mortalidade.

Apesar dos avanços terapêuticos e progresso da farmacoterapia da IC, o prognóstico da síndrome ainda merece atenção, pois pacientes apresentam expectativa de vida média de seis anos após diagnóstico. Nesse sentido, terapias adjuvantes que melhorem a qualidade de vida do portador de IC são hoje imprescindíveis.

O treinamento físico aeróbico tem sido preconizado como adjuvante à terapia farmacológica da IC. Dentre seus benefícios destaca-se: a) redução da atividade nervosa simpática, b) melhora da tolerância aos esforços físicos e c) redução da atrofia muscular e melhora da capacidade oxidativa muscular.

Na presente palestra daremos destaque aos benefícios do treinamento físico aeróbico sobre a função e o trofismo da musculatura esquelética em modelo experimental de IC e em pacientes com IC. Daremos destaque aos estudos em andamento do nosso grupo visando o efeito do treinamento físico aeróbico sobre a sinalização de vias proteolíticas como a das calpaínas e o sistema ubiquitina-proteassoma.

Apoio financeiro dos estudos: FAPESP (06/61523-7) e CNPq (301519-2008-0)

Sympathetic actions on the skeletal muscle protein metabolism.

Luiz Carlos Navegantes (USP)

Skeletal muscle protein mass depends on the balance between synthesis and degradation. The main intracellular proteolytic systems in the skeletal muscles are the lysosomal, the Ca^{2+}-dependent and the ubiquitin-proteasome system (UPS). The majority of intracellular proteins are degraded by the UPS. In all types of atrophying muscle, the UPS is activated, and catalyzes the degradation of the bulk of muscle proteins. In addition, there is a dramatic induction of two muscle-specific ubiquitin-ligases, Atrogin-1 and MuRF-1, whose induction occurs before the onset of muscle weight loss and which is necessary for rapid atrophy. The key mediators of this catabolic response during atrophy are the FoxO (Forkhead box O) family of transcription factors, whose activity is suppressed during growth by phosphorylation by AKT. We have been studying the mechanisms through which the rates of these different proteolytic components are regulated by hormonal, nutritional and neural factors. Among the factors that regulate proteolysis, the Sympathetic Nervous System (SNS) has an important physiological role. We have previously shown that SNS, through the activation of beta-2 and beta-3 adrenoceptors and

cAMP signaling cascade, exerts an anabolic effect on muscle protein metabolism by inhibiting the activity of the Ca^{2+}-dependent proteolytic system. In the present study, we present evidences that the beta-2 adrenergic agonist clenbuterol (CB) in vivo induces hypertrophy and reduces UPS activity in skeletal muscles from normal rats. In addition, CB suppresses the transcriptional upregulation of the ubiquitin ligases Atrogin-1 and MuRF-1 in muscles from mice under atrophic conditions. This effect is independent of PGC-1α (Proliferator-Activated Receptor-γ Coactivator-1α) since it has been also observed in muscles from PGC-1α muscle-specific knock-out animals. The CB-induced reduction of atrophy-related genes occurs through the activation of AKT, which leads to phosphorylation of FOXO3a. The anti-proteolytic effect of CB on UPS is probably mediated through the cAMP pathway since elevated cAMP levels induced by cAMP-phosphodiesterase inhibitors in vitro suppresses the UPS activity, the levels of ubiquitin-conjugated proteins and the mRNA and protein levels of Atrogin-1 transcripts in muscles from normal rats and in C2C12 myotubes treated with dexametasone. These data suggest that stimulation of beta-2 adrenoceptors, through the activation of cAMP and AKT signaling pathways, inhibit ubiquitin-proteasome proteolysis by increasing FOXO3a phosphorylation and suppressing Atrogin-1 mRNA expression in skeletal muscle from rodents. The understanding of the precise mechanisms by which endogenous catecholamines promote muscle anabolic effects may bring new perspectives for efficient treatment of muscle-wasting conditions.

New function of the kallikrein-kinin system in the muscular atrophy.

Parreiras-e-Silva, LT[1], Pires-Oliveira M[2], Lira EC[3], Reis RI[1], dos Santos GA[1], Pesquero JB[4], Kettelhut IC[1], Navegantes LCC[3], Gomes MD[1], Godinho RO[2], Costa-Neto CM[1] [1]FMRP-USP – Biochemistry and Immunology, [2]UNIFESP – Pharmacology, [3]FMRP-USP – Physiology, [4]UNIFESP – Biophysics

Introduction: Skeletal muscle fibers loose mass due to catabolic signals such as those driven by pro-inflammatory molecules and malnutrition. Ultimately those effects are mainly mediated by the ubiquitin-proteasome system (UPS), but also by other proteolytic systems. Muscle proteolysis is increased during several pathologies such as cancer, diabetes and sepsis, in some cases leading to severe muscular atrophy. Objective: Since the B_1 and B_2 kinin receptors are involved in inflammatory responses, we decided to analyze the participation of the kallikrein-kinin system (KKS) in three different *in vivo* models of muscle atrophy, and also *in vitro* using primary culture and C2C12 cells. Methods: Wistar rats, Balb-C mice and C57BL/6 wild-type and kinin B_1 receptor *knockout* mice were gonadectomized for 2, 7, 15 and 30 days to induce levator ani (LA) muscle atrophy. In another atrophy model, C57BL/6 mice were fasted for 2 days and gastrocnemius muscles were collected. In the third model, sepsis was induced in young Wistar rats by the cecal ligation and puncture (CLP) method, which afterwards also causes muscle atrophy. After process-sing, samples from different muscles were analyzed concerning the expression of transcripts for kinin B_1 and B_2 receptors, atrogin-1 and MuRF-1 (key enzymes of UPS), TNF-α and IL-6 (pro-inflammatory cytokines with known catabolic roles in muscular tissue), IGF-1 (an anabolic peptide), and LC3 and cathepsin L (involved in lisosomal proteolysis). All experiments were conducted in accordance with the local Animal Care and Use Committee (FMRP-USP 046/2006). Concerning the *in vitro* approach, to analyze the molecular effects of activation of the KKS we treated cultured myotubes obtained from all the hindlimb muscles from young Wistar rats and developed from the murine C2C12 cell line, with the agonists of kinin receptors, bradykinin (BK) and des-Arg[9]-BK (DABK). After sample processing, the expression levels of targeted transcripts were analyzed by conventional or quantitative PCR, as well as western blotting analyses. RESULTS: We show that mRNA expression levels of atrogin-1 and MuRF-1 were increased in the muscles collected from animals submitted to the three *in vivo* models. The kinin B_1 receptor mRNA was also increased in the same muscles and the B_2 receptor mRNA was increased in the murine fasting model. Moreover, LA muscles from B_1 receptor *knocktout* mice did not induce MuRF-1 transcript expression and exhibited a slight increase in relative mass. Expression of LC3 mRNA was induced to a lower extent when compared to the WT animals. Western blotting analyses showed an increase in ERK 1/2 phosphorylation in LA muscles from Balb-C mice treated with the kinin B_2 receptor antagonist HOE-140, suggesting a possible mechanism for KKS role in regulating muscle mass in physiological situations. *In vitro*, we showed that the treatment of C2C12 myotubes with the B_1 receptor agonist, DABK, induced a decrease in myotubes diameter after 12 to 48 hours (5 and 15%, respectively). Transcript expression analysis showed an increase of the inflammatory cytokines, TNF-α and IL-6, and the lisosomal protease cathepsin L. It was also shown a decrease in IGF-1 mRNA expression. In myotubes from primary culture of rat hindlimbs, activation of the B_2 receptor induced an increase in MuRF-1 mRNA and in atrogin-1 protein levels. Activation of both receptors induced an increase in reactive oxygen species (ROS) production until 1 hour following stimulus. DIS-

CUSSION: Our data evidence the participation of KKS in muscle mass control, and therefore its involvement in muscular atrophy, mainly via UPS and lisosomal proteolytic systems. Finally, our results also suggest possible mechanisms by which kinins regulate such process, as follows: *i)* activation of NF-kB, since B_1 receptor stimulation in cultures of myotubes induced an increase in inflammatory cytokines and ROS production, and B_1-*knockout* mice did not induce MuRF-1 mRNA expression in LA of castrated animals; *ii)* regulation of MAPK pathway, since the antagonism of the B_2 receptor in mice induced an increase of ERK 1/2 phosphorylation levels in LA. Also, the increase of MuRF-1 and atrogin-1 levels in myotubes concomitantly with the increase of ROS production induced by BK, suggest a possible activation of p38 MAPK; *iii)* regulation of PI3K/Akt signaling pathway, since B_1-*knockout* mice showed a lower increase in LC3 mRNA expression in LA muscle, and DABK decreased IGF-1 expression (which activates PI3K/AKt) in cultured myotubes, and finally because BK induced atrogin-1 protein expression in rat myotubes, which expression is known to be regulated by Akt/Foxo. Financial support: FAPESP, CAPES, CNPq, FAEPA.

Excitation-secretion coupling in Leydig cells.

Wamberto A. Varanda (USP)

Leydig cells are responsible for the synthesis and secretion of testosterone, processes controlled by the hypophysis via the Luteinizing Hormone (LH). Binding of LH to a G Protein Coupled Receptor in the plasma membrane of Leydig cells, results in a primary increase in cAMP and in the intracellular calcium concentration ($[Ca^{2+}]_i$). Both processes are known to be essential for testosterone production. The question we ask here is: how events occurring in the plasma membrane are linked to and to what extend determine changes in the intracellular calcium concentration? Using the whole cell variation of the patch clamp technique we show the presence of T-type calcium currents which can be enhanced by treatment of the cells with LH. This effect is also evident when the cells are exposed to dibutiryl-cAMP. The electrohysiological properties of the currents and immunofluorescence experiments support the conclusion that the calcium currents are carried by $Ca_v3.2$ (α_{1H}) channels. Measurements of intracellular calcium activity with the fluorescent dye Fluo3 and confocal microscopy, show that the changes in $[Ca^{2+}]_i$ induced by LH require the presence of extracellular calcium and do not occur when $Ca_v3.2$ are blocked by nickel. Using specific antibodies we show that Leydig cells express the tree isoforms of both ryanodine receptors (RYRs) and inositol 1,4,5-trisphosphate receptors (IP3Rs). The RYRs and IP3Rs are functional, as judged both from their activation by caffeine and IP3 and block by ryanodine and 2-APB, respectively. Both types of receptors are involved in a calcium-induced calcium-release mechanism (CICR), which amplifies the initial Ca^{2+} influx through plasma membrane T-type calcium channels. Nevertheless, our results show that RYRs are the principal players involved in the release of Ca^{2+} from the endoplasmic reticulum. This assumption is supported mainly by the fact that the global Ca^{2+} changes evoked by LH are readily blocked by 100 µM ryanodine but not by 2-APB or xestospongin C. Both calcium currents and transients are predominantly modulated by PKA but PKC also participates in the process. Finally, we will show that blockage of the ryanodine receptors, but not IP3 receptors, inhibits both the hormone-induced $[Ca^{2+}]_i$ transients and the subsequent testosterone production by the Leydig cells. These results not only broaden our understanding of the role played by calcium in Leydig cells but also show, for the first time, that ryanodine receptors play a crucial role in determining testosterone secretion by the testis. Financial Support: FAPESP, CNPq, FAEPA.

Nucleoplasmic calcium regulates cell through legumain

Andrade VA[‡], Guerra MT[§], Jardim, CA[†], Melo FM[†], Silva Jr WA[¶], Ortega MJ[‡] Robert ME[♣], Nathanson MH[§], Leite MF[†,δ]
[‡]UFMG − Biochemistry and Immunology, [†]UFMG − Physiology and Biophysics, [¶]FMRP-USP − Genetics and Center for Cell Based Therapy, [♣]Yale University − Pathology, [§]School of Medicine New Haven − Internal Medicine/ Cell Biology, [δ]Howard Hughes Medical Institute.

Nucleoplasmic Ca^{2+} regulates cell growth, but the proteins through which this occurs are unknown. To investigate this, we used Rapid Subtraction Hybridization to subtract genes in SKHep1 liver cells expressing the Ca^{2+} buffer protein parvalbumin (PV) targeted to the nucleus, from genes in cells expressing a mutated form of nuclear-targeted PV which has one of two Ca^{2+}-binding site inactivated. The subtraction permitted selection of genes whose expression was affected by a small alteration in nuclear Ca^{2+} concentration. The asparaginyl endopeptidase legumain (LGMN) was identified by this assay and differential expression of this gene was validated by Real Time-PCR. When Ca^{2+} was buffered in the nucleus of SKHep1 cells, LGMN mRNA was decreased by 97%, and decreased expression at the protein level was observed by immunoblot and immunofluorescence. Knockdown of LGMN by siRNA decreased proliferation of SKHep1 cells by ~50% as measured both by BrdU uptake and mitotic index. A significant reduction in the fraction of cells in G2/M phase was seen as well. This was associated with increases in expression of cyclins A and E. Furthermore, LGMN expression was increased in hepatocellular carcinoma cells rel-

ative to normal hepatocytes in the same tissue specimens. These findings identify a new role for LGMN and provide evidence that nuclear Ca^{2+} signals regulate cell proliferation in part through modulation of LGMN expression, and suggest that increased expression of LGMN may be involved in carcinogenesis in the liver.

Physiological and pathological aspects of brain mitochondrial Ca^{2+} transport.

Roger Frigério Castilho (UNICAMP)

Changes in mitochondrial integrity, reactive oxygen species release and Ca^{2+} handling are proposed to be involved in the pathogenesis of many neurological disorders. In mitochondria, Ca^{2+} influx occurs electrophoretically in response to the internally negative membrane potential generated by the respiratory chain or ATP hydrolysis by the reverse activity of the mitochondrial ATP synthase. The channel that promotes mitochondrial Ca^{2+} uptake is inhibited by ruthenium red and has low affinity for Ca^{2+}, presenting a K_m of 10-30 μM. Ca^{2+} efflux from mitochondria involves two pathways. The Na^+-dependent pathway, presented mainly in excitable tissue, which exchanges one Ca^{2+} ion for two Na^+ ions; while the ubiquitous Na^+-independent pathway exchanges Ca^{2+} for $2H^+$. Under steady-state, mitochondrial Ca^{2+} uptake typically maintains extramitochondrial Ca^{2+} ions in the 0.5 to 1.0 μM range. The main function of mitochondrial Ca^{2+} uptake appears to be the regulation of matrix Ca^{2+} concentrations, which stimulate the activity of regulatory enzymes of the Krebs cycle.

Energy metabolism defects in neurons cause increases in intracellular Ca^{2+} levels, either by directly impairing Ca^{2+} removal systems or due to *N*-methyl-D-aspartate (NMDA) receptor activation. Under these conditions, the mitochondrion is the main organelle responsible for Ca^{2+} sequestration, a required step in NMDA-induced neurotoxicity. Excessive mitochondrial Ca^{2+} uptake and oxidative stress can cause non-selective inner mitochondrial membrane permeabilization, known as the permeability transition (MPT). MPT results in mitochondrial Ca^{2+} release, organellar swelling, release of mitochondrial apoptogenic factors such as cytochrome *c* and loss of inner membrane potential and ATP synthesis. MPT can result both in necrosis and apoptosis.

We have recently shown that the addition of the succinate dehydrogenase (SDH) inhibitor 3-nitropropionic (3NP) to Ca^{2+}-loaded brain mitochondria leads to MPT (Maciel et al., 2004). Brain and heart mitochondria were generally more sensitive to 3NP and Ca^{2+}-induced MPT than mitochondria from liver and kidney. In addition, 3NP resulted in more pronounced MPT in striatal mitochondria than in cortical or cerebellar organelles (Mirandola et al., 2010). We propose that the increased susceptibility of the striatum to 3NP-induced neurodegeneration in rats systemically treated with this toxin may be partially explained by its susceptibility to MPT.

The adenine nucleotides ADP and ATP are probably the most important endogenous inhibitors of MPT. We studied the inhibitory effects of adenine nucleotides on brain MPT (Saito and Castilho, 2010). We observed that ATP lost most of its inhibitory effects on MPT when the experiments were carried out in the presence of ATP-regenerating systems. These results indicate that MPT inhibition observed in the presence of added ATP could be mainly due to hydrolysis of ATP to ADP. From mitochondrial swelling measurements, half-maximal inhibitory values (K_i) of 4.5 μM and 98 μM were obtained for ADP and ATP, respectively. In addition, a delayed mitochondrial swelling sensitive to higher ADP concentrations was observed. Mitochondrial anoxia / reoxygenation did not interfere with the inhibitory effect of ADP on Ca^{2+}-induced MPT, but oxidative phosphorylation markedly decreased this effect. We conclude that ADP is a potent inhibitor of brain MPT. Our results suggest that ADP can have an important protective role against Ca^{2+}-induced MPT and tissue damage under conditions of brain ischemia and hypoglycemia.

Maciel EN, Kowaltowski AJ, Schwalm FD, Rodrigues JM, Souza DO, Vercesi AE, Wajner M, Castilho RF (2004) Mitochondrial permeability transition in neuronal damage promoted by Ca^{2+} and respiratory chain complex II inhibition. *J Neurochem* 90: 1025-1035.

Mirandola SR, Melo DR, Saito A, Castilho RF (2010) 3-nitropropionic acid-induced mitochondrial permeability transition: comparative study of mitochondria from different tissues and brain regions. *J Neurosci Res* 88: 630-639. Saito A, Castilho RF (2010) Inhibitory effects of adenine nucleotides on brain mitochondrial permeability transition. *Neurochem Res* (in press).

Supported by FAPESP and CNPq.

Dopamine turnover following administration of 7-nitroindazole to rats with L-DOPA-induced dyskinesia. E.A. Del Bel[1,2], R.S. Szawka[1], F.E. Padovan-Neto[1,2], C.A. da-Silva[1], J.A. Anselmo-Franci[1]. [1]FORP-MEF - Physiology and [2]FMRP- Neurology

Purpose of the study: Administration of L-3,4-dihydroxyphenylalanine (L-DOPA) enhances dopamine synthesis and release in dopamine-deafferented striatum leading to improvements in parkinsonian symptoms. Long-term use of L-DOPA leads to side-effects such as unwanted movements or dyskinesias. We have recently described that nitric oxide synthase inhibition is able to reduce L-DOPA-induced dyskinesias in experimental Parkinson (3). This result suggests that controlling nitric oxide production may be useful for the prevention of dyskinesias. Be-

cause the mechanism of this effect is poorly understood, it is of interest to determine whether nitric oxide synthase inhibitor 7-nitroindazole would affect neurochemical responses. Therefore, we determined the effects of 7-nitroindazole in striatal levels of catecholamines and indoleamines in 6-hydroxydopamine (6-OHDA) lesioned rats with L-DOPA induced dyskinesias. Methods. Male Wistar rats with unilateral 6-OHDA lesions of the medial forebrain bundle or sham animals (2, n=5-7/group) were treated chronically (21 days) with L-DOPA (30mg/kg) to induce abnormal involuntary movements (AIMs) (1). Comparisons between 6-OHDA lesioned (dyskinetic) and sham (non-dyskinetic) L-DOPA-treated rats, receiving either saline or 7-nitroindazole, were then carried out with regard to striatal levels of dopamine (DA), DOPAC (DA metabolite), serotonin (5-HT) and 5-hydroxyindoleacetic acid (5-HIAA, a 5-HT metabolite), measured by HPLC analyses. Since it has been shown that the nigrostriatal DA system has functional and neurochemical asymmetry, the measurements were made separately in each side. Results: L-DOPA induced AIMs in all 6-OHDA lesioned rats, which was attenuated by 7-nitroindazole. 6-OHDA lesion induced a decrease in DA and DOPAC levels in the striatum ipsilateral to lesion (85.2 and 90%, respectively, $F_{1,19}=87.3$ $p<0.001$); 7-nitroindazole treatment decreased DOPAC in both striatum (28-30%). 7-nitroindazole treatment per se increased DA level in sham-L-DOPA treated rats (42-60%). The content of 5-HT decreased in the ipsilateral striatum (28.2% of control); however 7-nitroindazole increased it in the contralateral one (24.5%). 5-HIAA decreased in the striatum ipsilateral to lesion (35.6%) and 7-nitroindazole treatment did not change lesion effect. DOPAC/DA ratio regarded as a measure of DA turnover. was significantly higher (391%, $F_{1,19}=4.38$, $p=0.05$) in the ipsilateral striatum of dyskinetic rats. This effects was prevented by 7-nitroindazole. 5HIAA/5HT ratio increased in the striatum ipsilateral (119%) but did not change after 7-nitroindazole treatment. Conclusion: Confirming previous results (3), treatment with 7-nitroindazole attenuated L-DOPA-induced dyskinesias in animals with unilateral striatal 6-OHDA lesions. Dyskinetic animals show an increase in dopamine metabolism as expressed by increased DOPAC/DA levels. This increase in dopamine turnover could serve to maintain dopamine levels in the dopamine-depleted striatum and may account for the therapeutic benefit of L-DOPA. However, it may also be related to the dyskinesias induced by this drug. Interestingly, 7-nitroindazole, a preferential neuronal nitric oxide synthase, was able to prevent both this turnover increase and attenuate the drug-induced dyskinesias. Together these results suggest that nitric oxide production could be important for the occurrence of L-DOPA-induced dyskinesias. Financial support: FAPESP, FAPESP/INSERM, CNPq, CAPES/COFECUB. Reference(s): (1) Cenci MA, Lee CS Björklund A (1998) L-DOPA-induced dyskinesia in the rat is associated with striatal overexpression of prodynorphin- and glutamic acid decarboxylase mRNA Eur J Neurosci 10: 2694-2706. (2) Gomes MZ, Raisman-Vozari R, Del Bel EA (2008) A nitric oxide synthase inhibitor decreases 6-hydroxydopamine effects on tyrosine hydroxylase and neuronal nitric oxide synthase in the rat nigrostriatal pathway Brain Res 1203:160-169. (3) Padovan-Neto FE, Echeverry MB, Tumas V, Del-Bel EA. (2009) Nitric oxide synthase inhibition attenuates l-DOPA-induced dyskinesias in a rodent model of Parkinson's disease Neuroscience 159(3):927-935.

Brain in movement: the role of physical exercise in Parkinson's disease.

Aderbal S. Aguiar-Jr. (UFSC)

Parkinson's disease (PD) is considered a motor neurodegenerative disease, and its diagnosis is based on cardinal motor signs. In addition, subtle cognitive impairments are present even during the PD earlier phases that includes attentional and working memory deficits. Dopamine-replacement therapy has dominated the treatment of PD since the early 1960s and although the currently approved antiparkinsonian agents offer effective relief of the motor deficits, they have not been found to alleviate the underlying dopaminergic neuron degeneration, and drug efficacy is gradually lost. In addition, this approach did not shows modifying-disease effects to cognitive dysfunction. Thus, the management of non-motor symptoms of PD remains a challenge. A putative strategy is the physical exercise, because there are several evidences showing benefits to aged and diseased brain. Clinical studies have been inconsistent to show exercise-induced reverse PD symptoms, which can be largely attributed to methodological issues. On the other hand, recent findings have shown that exercise programs can present beneficial effects in neurointoxicant rodent models of PD such as 6-hydroxydopamine (6-OHDA) and 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). However, no consistent evidence for the potential of exercise to prevent cognitive impairments in animal models of PD has to date been documented. Here, we evaluated the neuroprotective properties of exercise in ameliorate animal models of basal ganglia dysfunction, such as 6-OHDA-induced motor dysfunction and MPTP-induced olfactory and cognitive dysfunction. Five to 6-month-old male C57BL/6 mice were maintained under controlled environment (UFSC ethical board number

23080.019001/2009-27). Initially, mice were assigned to two groups: untrained and runners. Animals were exercised with a treadmill ergometer during 6 weeks, where blood lactate levels were weekly monitored to control exercise intensity. Five animals per groups were sacrificed 48h after the exercise program ending to analyze skeletal muscle mitochondrial function. Moreover, we separated animal in two additional groups: 6-OHDA and MPTP treated mice. The animals were treated with the neurotoxins 6-OHDA (4 μg), injected into the right midstriatum (anterior 0.4, lateral 1.8, ventral 3.5), or MPTP (65 mg/kg) that was administered intranasally (Prediger et al, Neurotox Res, 17, 114, 2010) or their respective vehicle 48 h after the end of physical program. Blood lactate levels of trained mice remained within the moderate levels of intensity effort. Moreover, the activity of citrate synthase and complex I of skeletal muscle isolated mitochondria were increased in relation to sedentary controls. In the striatal 6-OHDA model, apomorphine treatment (0.6 mg/kg, s.c.) induced a progressive rotation in sedentary animals, which was not observed in the 6-OHDA trained mice, suggesting a reduced dopamine receptors sensitization in trained mice. We assessed rota rod performance of mice and verified a per se effect in the exercised mice, while 6-OHDA sedentary mice presented poor latency to fall than saline treated animals. Moreover, exercise was also able to ameliorate the rota rod performance of exercised mice impaired by the 6-OHDA treatment. We also evaluated the role of physical exercise on basal ganglia-dependent stimulus-response learning and memory. The intranasally MPTP treatment induces poor freezing response retrieval in the tone fear conditioning tasks and the exercise presented not per se effect. However, we observed that 6 weeks of moderate running exercise ameliorate this impairment in the exercise MPTP-treated group. Moreover, MPTP-treated groups also showed dopamine receptor sensitization as indicated by a marked increase in climbing behavior induced by a low dose of apomorphine (0.2 mg/kg, s.c.) that was prevented by exercise. Indeed, exercise prevented the catalepsy induced by haloperidol (0.32 mg/kg, i.p.) in MPTP-treated mice. These effects seem not be related to neuroprotective actions of exercise since it did not prevent the MPTP-induced reduction in the levels of dopamine and tyrosine hydroxylase enzyme in the striatum. However, exercised MPTP-treated mice presented decreased striatal DA turnover in comparison to sedentary MPTP-treated mice. Our evidences are coherent with this statement due to modifying-disease effects induced by exercise. Taken together, the present findings suggest that physical exercise can reduce behavioral alterations associated to dopamine receptors imbalance in neurointoxicant models of PD and that this response is an association between neuroprotection and post-synaptic changes. Financial support: FAPESC, CAPES and CNPq.

Behavioral and neurochemical alterations induced by intranasal administration of MPTP, an experimental model of Parkinson´s disease, in mice with genetic deletion of the heparin binding growth factors Pleiotrophin and Midkine.

Rita Raisman-Vozari[1], Argelia Esperanza Rojas-Mayorquin[1], Elaine Del Bel[2], Raymond Mongeau[3], Laurence Lanfumey[3] Rui D. S. Prediger[4]. [1]INSERM-UMR, [2]FORP-USP, [3]INSERM – Neuropsychopharmacologie, [4]UFSC - Farmacologia

The “olfactory vector hypothesis” postulates that Parkinson’s disease (PD) may be caused or catalyzed by agents that enter the brain via the olfactory mucosa. We have recently demonstrated that rats treated with intranasal (i.n.) infusion of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), a neurotoxin largely used to modeling PD in primates and rodents, suffer from progressive signs of PD that are correlated with time-dependent degeneration in dopaminergic neurons. On the other hand, previous studies have suggested that heparin binding growth factors, such as Midkine (Mdk), might play an important role in nigrostriatal system development and in the compensatory mechanisms that take place in PD. Here, we employed Mdk knockout mice (Mdk-Ko) to evaluate the relevance of such heparin binding growth factor in the olfactory, emotional, learning and memory, and motor deficits induced by a single i.n. administration of MPTP (1 mg/nostril). Mimicking the clinical condition, mice exhibited an early disruption in olfactory discrimination ability and social recognition memory during the first two weeks after MPTP treatment. These responses were not due to locomotor impairments, since no behavioral alterations in the activity chambers were observed in the first post-treatment week. Interestingly, Mdk-Ko mice have a poorer performance in the olfactory discrimination and social recognition tests, but not in the elevated plus-maze, than wild-type controls. Of high importance, selective locomotor impairments evaluated in activity chambers were observed in Mdk-Ko mice at 3 weeks after i.n. MPTP infusion.

The present results suggest, for the first time, the role of Mdk in olfactory and short-term social memory processes in rodents. Moreover, our findings reinforce the involvement of Mdk in compensatory mechanisms in PD, indicating that the genetic deletion of Mdk confers increased susceptibility to behavioral deficits induced by MPTP in mice.

Molecular approaches to the study of natural products modulating nociception.
Marilia Zaluar P. Guimarães (UFRJ)
Natural products have been fundamental tools in the understanding of how pain occurs and also served as medicines to treat pain for a long time. For instance, acetylsalicylic acid extracted from the willow bark was important to confirm the role of prostaglandins in sensitizing nociceptors. More recently, capsaicin, the pungent ingredient in chili peppers, was used to clone TRPV1, a channel responsible for the transduction of several painful stimuli. Upon activation by vanilloids, protons, endogenous lipids or high temperatures, TRPV1 causes nociceptor excitation via cation influx. In addition, TRPV1 has been linked to some toxins and venoms produced by animals and plants that act as repellents to predators, such as resiniferatoxin, spider toxins and jellyfish venom. Other toxins are likely to produce burning pain via activation of TRPV1. One good candidate is the bee venom, responsible for thousands envenomations a year in Brazil. Previous works have suggested that some of the bee venom-caused inflammatory hypersensitivity is of neurogenic origin. In particular, authors have suggested the involvement of TRPV1 channels in some of the bee venom effects. However, this was not clearly demonstrated on the molecular level. We decided to investigate this matter using *Xenopus* oocytes expressing TRPV1 and performing electrophysiology experiments. Preliminary data suggest that crude bee venom is able to activate TRPV1 channels. These initial experiments indicate that crude venom might contain a toxin or toxins capable of activating TRPV1. Further experiments will help clarify which constituents are responsible for modulating TRPV1 and cause burning pain sensation.

Cell and tissue responses to melittin and its antagonist.
Camila El-Kik (UFRJ)
Apis mellifera bee venom is composed by a mixture of many components some as proteins with different molecular weight but the toxic effect of the venom is attributed mainly the presence of melittin. Venom components present pharmacologic and allergic effects producing located pain, edema and erythema caused by the increase of the vascular permeability. Melittin is a cytotoxic protein, that induces hemolysis, cardiotoxic and myotoxic effects because the property of decreased superficial tension of plasma membrane, acting like a natural detergent. This component has a potent destructive action on biological membranes mainly when acts synergic with the venom phospolipase A2 in the membrane phospholipids acting as diffusion factor. Recently studies have reported that suramin, a polysulfonated naphtylurea derivative, is an enzymatic inhibitor that prevents the effects produced by polications present in the snake venom and some isolated myotoxins that act like *A. mellifera* components. We evaluated the ability of suramin to antagonize the vascular permeability, edema and citotoxicity in endothelial cells induced by *Apis mellifera* crude venom and by melittin.
The plasma extravasation was assessed by using a i.v. injection of a visual marker, Evans blue and measured the absorbance which was express in arbitrary units. Intradermical injection of bee venom or melittin induced intense plasma extravasation in the local injection and was compared with control animals that received only PSS injection. The effect of crude venom and mellitin was reduced when was preincubated, pre and post treated with suramin. Paw edema induced by injection of bee venom or mellitin was inhibited by suramin treatments. Primary cultures of microvascular endothelial cells were obtained from rat cremaster muscle microcirculation according to a method described previously. Cells culture were incubated per 1 hour with venom or mellitin alone or with suramin. After incubation sobrenadant was collected and LDH measured. Data show that low concentrations of suramin inhibited completely the cytotoxic activity of bee venom and mellitin effects. These data suggest that the toxic effect of bee venom is due the melittin action and that suramin has a protective effect against damage caused by bee venom components.