SBFTE

54 Anos

1966 - 2020

PROGRAM

52nd

Brazilian Congress of Pharmacology and Experimental Therapeutics

Online

October: 8th, 15th, 22nd, 29th

November: 5th, 12th, 17th, 19th, 24th, 26th

Welcome Letter

Dear Friends and Colleagues,

On behalf of the board of directors of SBFTE and the members of the Organizing Committee, it is my great honour to welcome you to our 52nd Brazilian Congress of Pharmacology and Experimental Therapeutics!

The Covid-19 pandemic imposed on them, organizers, speakers and attendees, the challenge of holding our 52nd Congress digitally online. However, we must remember that although this challenge is significant, it is far less than the burden faced by those who were directly or indirectly affected by the disease; we take this opportunity to convey our solidarity and condolences.

The Scientific Committee of the 52nd Congress has carefully prepared a rich Scientific Program to provide a comprehensive overview of the latest research developments in various areas of pharmacology. A distinguished group of speakers from Brazil and abroad will participate in the 52nd Congress and share with us the most significant advances in our conferences, lectures, symposia and round tables.

We would like to express our thanks to CNPq for providing the financial support for our 52nd Congress, despite the major budget cuts that have been recently imposed to them.

SBFTE would also like to thank its entire community, which massively registered for the 52nd Congress, totalling an impressive number of almost 1,200 attendees. This is a strong demonstration of the solidarity and maturity of Brazilian Pharmacology and points out that those who preceded us paved a safe route whose final inescapable destination is excellence in our scientific area.

Our Annual meetings have traditionally combined great scientific content with a fantastic opportunity to see old friends, make new ones and boost our network of collaborations. Although we are prevented from maintaining this tradition in our 52nd Congress, we are certain that we will make the most of all scheduled activities.

We truly appreciate all SBFTE members, Colleagues, Invited Speakers and Collaborators and hope we all have a great time during our 52nd Congress, and look forward to welcoming all of you next year to Florianópolis!

André S. Pupo
Congress President

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SBFTE Board of Directors (2018-2020)

Executive Secretariat SBFTE
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SBFTE Board of Directors (2021-2023)

The SBFTE has a long history of outstanding contributions to scientific training and the development of research and teaching in Pharmacology in Brazil. Since its foundation 54 years ago, these contributions have been built with the effort, dedication and creativity of many presidents, directors and council members. In these troubled times we are living in, more than ever, and more than anything, we will need to be united and strong in facing the challenges that are on the horizon.

The elected Directory Board has been discussing a list of priorities and how to get them implemented. There are several ideas of scientific society that we share and that we will seek to put into practice, in partnership with the Deliberative Council, Postgraduate Forum, SBFTE Youth and, of course, with the involvement of all SBFTE members.

The primary motivation is to keep the Brazilian Pharmacological Society active, integrated and sensitive to the legitimate wishes of the Brazilians, in addition to being inclusive and representative of all areas of interest in teaching and research in Pharmacology.

We believe that investing in science and education is a necessary attitude within modern political and social thought. Our commitment and willingness to make a vigorous defense of the ideals of reason, science, humanism, and progress will be permanent.

In these 54 years of existence and consolidation of SBFTE, the National Science and Technology System has also been strengthened. Today a dense and diversified network of public and private organizations that includes universities, public foundations, research centers and development agencies at the three levels of government are in place. Thanks to the articulated action of these agencies, it was feasible for the country to modernize its agricultural sector, discover and make the pre-salt layer oil reserves viable, develop medicines, structure the Unified Health System (Brazilian acronym, SUS), as well as to organize the combat of the Zika virus epidemic and the COVID-19 pandemic, among many other initiatives. However, the economic crisis of the last years, the budget cuts and quota restrictions let to the risk of dismantling fundamental conquests that took more than half a century to happen. Thus, alongside with scientific entities such as the Brazilian Society for the Science Progress, the Brazilian Academy of Science and the Federation Societies of Experimental Biology (Brazilian acronyms: SBPC, ABC and FESBE, respectively) our engagement in defense of the National Science and Technology System will also be permanent. Stay safe,

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Thiago Cunha

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Teresa Dalla Costa

Administrative Director
Flavia Almeida Santos

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Richardt Gama Landgraf

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General Secretary: Rosely O. Godinho
First-Secretary: Teresa C. T. Dalla Costa
Treasurer: Ronaldo de A. Ribeiro

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General Secretary: François G. Noël
Secretary: Isac A. Medeiros
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Aron Jurkiewicz (Unifesp-EPM)
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Maria Christina W. Avellar (Unifesp-EPM)
Rita C. A. Tostes (USP
Yara Cury (Instituto Butantan)

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Vice-President: Manassés C. Fonteles
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Treasurer: Mauro M. Teixeira

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Edson Antunes (Unicamp)
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João Batista Calixto (UFSC)
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Regina P. Markus (USP-SP)
Rita C. A. Tostes (USP-SP)

2000-2001

President: Antonio José Lapa
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Edson Antunes (Unicamp)
Gloria E. P. de Souza (USP-RP)
Jamil Assreuy (UFSC)
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Zuleica Bruno Fortes (USP-SP)

1998-1999

President: Maria Cristina O. Salgado
Vice-President: Regina P. Markus
General Secretary: Gustavo Ballejo
Secretary: José Geraldo Mill

Treasurer: Jamil Assreuy

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Fernando de Q. Cunha (USP-RP)
Gilberto de Nucci (Unicamp)
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Zuleica B. Fortes (USP-SP)

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Vice-President: Maria Cristina O. Salgado
General Secretary: Jamil Assreuy
Secretary: Giles A. Rae
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Gilberto de Nucci (UNICAMP)

1994-1995

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Vice-President: William A. do Prado
General Secretary: Giles A. Rae
Secretary: Manoel Odorico de M Filho
Treasurer: Jamil Assreuy Filho

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Catarina S. Porto(Unifesp-EPM)
Fernando M. A. Correa (USP-RP) (presidente do Conselho)
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1992-1993

President: Renato S. B. Cordeiro
Vice-President: João B. Calixto
General Secretary: Giles A. Rae
Secretary: Manoel Odorico de M. Filho
Treasurer: Patrícia M. R. e Silva

Council Members (1992-1993)

Caden Souccar (Unifesp-EPM) (1990-1992)
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Gilberto de Nucci (Unicamp)
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William A. do Prado (USP-RP)
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1990-1991

President: Renato S. B. Cordeiro
Vice-President: João B. Calixto
General Secretary: Regina P. Markus
First Secretary: Krishnamurti M. Carvalho
Treasurer: Patrícia M. R. e Silva

Council Members (1990-1991)

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Mario Tannhauser (UFRGS)
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Zuleica Bruno Fortes (USP-SP)
Paulina Sannomya (USP)
Sergio H. Ferreira

1988-1989

President: Sergio H. Ferreira
Vice-President: Guilherme Suarez-Kurtz
General Secretary: João Garcia Leme
First Secretary: Fernando Morgan de A. Correa
Treasurer: William A. do Prado

Council Members (1988-1989)

Antonio J. Lapa (Unifesp-EPM)
Aron Jurkiewicz (ex-Presidente)
Frederico Graeff (USP-RP)
João Batista Calixto (UFSC)
Mario Tannhauser (UFRGS)
Regina P. Markus (USP-SP)
Renato Balão Cordeiro (Fiocruz)
Therezinha B. Paiva (Unifesp-EPM)
Zuleica Bruno Fortes (USP-SP)

1986-1987

President: Sergio H. Ferreira
Vice-President: Guilherme Suarez-Kurtz
General Secretary: João Garcia Leme
First Secretary: Fernando Morgan de A. Correa
Treasurer: William A. do Prado

1984-1985

President: Aron Jurkiewicz
Vice-President: Roberto Soares de Moura
General Secretary: Sergio H. Ferreira
First Secretary: João Palermo Neto
Treasurer: Therezinha Bandieira Paiva

Council Members (1984-1985)

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E. A. Carlini (Unifesp-EPM)
Frederico G. Graeff (USP-RP)
Guilherme Suarez-Kurtz (INCa)

1982-1983

President: Alexandre P. Corrado
Vice-President: Aron Jurkiewicz
General Secretary: Sergio H. Ferreira
First Secretary: Roberto Soares de Moura
Treasurer: Adolfo M. Rothschild

1966-1981

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Vice-President: José Ribeiro do Valle
General Secretary: Alexandre P. Corrado
First Secretary: Lauro Sollero
Treasurer: Hanna A. Rothschild

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Geanne Arantes Freitas (USP-SP)
João Agostinho Machado Neto (USP-SP)
Sanseray Cruz Machado (USP-SP)

Speakers

INTERNATIONAL SPEAKERS

Adriano Rossi
University of Edinburgh
United Kingdom

Christopher J. Madden
Oregon Health & Science University
USA

Christopher Sobey
La Trobe University
Australia

Fernando Lopes
McGill University
Canada

Frederic Checler
Institut Pharmacologie Molecular Cellulaire (CNRS)
France

Graziano Pinna
University of Illinois at Chicago
USA

Humphey Hung-Chang Yao
NIEHS-NIH
USA

Jason McDougall
Dalhousie University
Canada

Marzia Malcangio
King's College London
United Kingdom

NATIONAL SPEAKERS

Aleksander R. Zampronio
Federal University of Paraná (UFPR)

Ana Paula Aquistapase Dagnino
Pontifical Catholic University of Rio Grande do Sul (PUCRS)

Anicet Okinga
National Institute of Industrial Property (INPI)

Benedito Guimarães Aguiar Neto
President Coordination for the Improvement of Higher Education Personnel (Capes)

Carlos Henrique de Carvalho
President Pro-Rector of Post-Graduate Studies Forum and Research (FOPROP)

Eliezer Barreiro
Federal University of Rio de Janeiro (UFRJ)

Evaldo Ferreira Vilela
President National Council for Scientific and Technological Development (CNPq).

Helena Tannhauser Barros
Federal University of Health Sciences of Porto Alegre (UFCSPA)

Leandro Ladislau Alves
MSL - Daiichi Sankyo

Luiz Guilherme de S. Branco
Ribeirão Preto Dentistry School (FORP)

Maurício Schuler Nin
Centro Universitário Metodista (IPA)

Pedro Antônio Castelo Teixeira
Federal University of Rio de Janeiro (UFRJ)

Thiago M. Cunha
Ribeirão Preto Medical School (FMRP)

Patricia Moriel
State University of Campinas (Unicamp)

About SBFTE Jovem

SBFTE Jovem, founded in 2013, is a Committee of the Brazilian Society of Pharmacology and Experimental Therapeutics (SBFTE). The Committee is composed of young Pharmacologists who are members of SBFTE, working in association with the SBFTE Board of Directors. Our mission is to create a permanent political-scientific forum dedicated to undergraduate, graduate students, Post-Docs, as well as young investigators and Junior faculty members of SBFTE to discuss scientific topics related to Pharmacology in order to promote the development of early-career investigators, stimulating the participation, insertion and collaboration of our members into the activities of the Society.

This year, SBFTE Young will promote one activity during the 52nd Brazilian Congress of Pharmacology and Experimental Therapeutics. The activity, *Além da Academia* (Beyond the Academy) is a roundtable to open a discussion about opportunities to Brazilian early-career scientists regarding innovation, new challenges in science, how to engage into carriers in the industry or any other relevant opportunities that are beyond scholar-driven carriers. This section is scheduled for November 17th, 2020 from 12:00 pm to 14:00 pm.

SBFTE Young Committee

João Alfredo de Moraes (UFRJ, Coordinator)
Jamylle Nunes de Sousa Ferro (UFAL, Vice-Coordinator)
Geanne Arantes Freitas (USP-SP)
João Agostinho Machado Neto (USP-SP)
Sanseray Cruz Machado (USP-SP)

Program at a Glance

OCTOBER				
	12h00	13h00	18h00	19h00
08/10/20 (Thursday	Meeting of the Board of SBFTE Directors and Deliberative Counci			Opening Lecture
15/10/20 (Thursday)	Roundtable		Meeting of SBFTE Permanent Forum of Postgraduation Programs in Pharmacology	
22/10/20 (Thursday)	SBFTE Assembly			Lecture
29/10/2019 (Thursday			SBFTE Jovem Assembly	

NOVEMBER			
	12h00	13h00	19h00
05/11/20 (Thursday)		Lecture	Lecture
12/11/20 (Thursday)		Lecture	
17/11/20 (Tuesday)	Roundtable		
19/11/20 (Thursday)	Symposium		
24/11/20 (Tuesday)	Symposium		
26/11/20 (Thursday)	Symposium		Closing Lecture

Scientific program

OCTOBER

08/10/2020 (Thursday)

12h00-14h00

Meeting of the Board of SBFTE Directors and Deliberative Council (Council and Directory Board Members only)

19h00-20h00 – **Opening Lecture**

Targeting brain inflammation after stroke - from the laboratory to the clinic

Christopher Sobey (La Trobe University, Australia)

Presented by Jamil Assreuy (UFSC)

15/10/2020 (Thursday)

12h00-14h00 – **Roundtable**

Políticas de Ciência e Tecnologia e de Pós-Graduação em Período de Pandemia

Chair: Rui Prediger (UFSC, SBFTE Permanent Forum of Postgraduation Programs in Pharmacology)

- Benedito Guimarães Aguiar Neto (Presidente Capes)
- Evaldo Ferreira Vilela (Presidente CNPq)
- Carlos Henrique de Carvalho (Presidente FOPROP)

18h00-20h00

Meeting of SBFTE Permanent Forum of Postgraduation Programs in Pharmacology (only for Heads of Postgraduation Courses in Pharmacology, Deliberative Council and Society Board)

22/10/2020 (Thursday)

12h00-14h00

SBFTE Assembly

19h00-20h00 – **Lecture**

Harnessing the chemistry behind host-parasite interactions to treat colitis

Fernando Lopes (McGill University, Canada)

Presented by Vanessa Pinho (UFMG)

29/10/2020 (Thursday)

18h00-20h00

SBFTE Jovem Assembly

NOVEMBER

05/11/2020 (Thursday)

13h00-14h00 – **Lecture**

Neuroimmune interactions and novel targets for chronic pain

Marzia Malcangio (King's College London, UK)

Presented by Soraia K P Costa (USP-SP)

19h00-20h00 – **Lecture**

From Glowing Penis to Oocyte-Producing Testis: Power of Mouse Genetics for the Understanding of Organogenesis and Human Diseases

Humphrey Hung-Chang Yao (NIEHS-NIH, USA)

Presented by Maria Christina W de Avellar (Unifesp-EPM)

12/11/2020 (Thursday)

13h00-14h00 – **Lecture**

Regulation of apoptotic cell clearance during resolution of inflammation

Adriano Rossi (University of Edinburgh, UK)

Presented by John Wallace (University of Calgary)

17/11/2020 (Tuesday)

12h00-14h00 – **Roundtable**

Além da academia (Beyond the Academy)

Chair: João Alfredo de Moraes (UFRJ, SBFTE Jovem)

- *Ciência no controle de dopagem* (Science allied to Sport: Doping control)
  Pedro Antônio Castelo Teixeira (UFRJ)
- *Msl: Assignments and perspectives*
  Leandro Ladislau Alves (MSL - Daiichi Sankyo)
- *Patent: The intellectual property right (IP) that transforms the world (Patente: O direito de propriedade intelectual (PI) que transforma o mundo)*
  Anicet Okinga (INPI)

19/11/2020 (Thursday)

12h00-14h00 – **Symposium**

Energy balance, thermoregulation and fever: advances in neural circuitry, pharmacological interventions and sex differences

Chair: Aleksander Roberto Zampronio (UFPR)

- *Advances in neural circuitry for thermoregulation and fever.*
  Christopher Madden (Oregon Health & Science University, USA)
- *Thermoregulatory and neuroimmunomodulatory effects of serotonin*
  Luiz Guilherme Branco (USP-RP)
- *Experimental febrile response in females: influences of estrous cycle and ovariectomy*
  Aleksander Roberto Zampronio (UFPR)

24/11/2020 (Tuesday)

12h00-14h00 – **Symposium**

Brain neurosteroids in stress and mood disorders

Chair: Helena Maria Tannhauser Barros (UFCSPA)

- *Brain neurosteroids and mood disorders*
  Helena Maria Tannhauser Barros (UFCSPA)
- *Brain neurosteroids in stress disorders*
  Maurício Schuler Nin (IPA)
- *Neurosteroids levels in stress and mood disorders*
  Graziano Pinna (University of Illinois at Chicago, USA)

26/11/2020 (Thursday)

12h00-14h00 – **Symposium**

Managing Pain Beyond 2020: Role of Gender and Other Patient Variables

Chair: John L. Wallace (University of Calgary, Canada) & Nathalie Vergnolle (INSERM, France)

- *Novel mechanisms of Analgesia: What is on the horizon?*
  Thiago M. Cunha (USP-RP)

- *Age as a Factor in the Safe Management of Chronic Pain*
  Jason McDougall (Dalhousie University, Canada)
- *Pre-clinical and clinical evidence on the effectiveness of NOP ligands on chronic pain states*
  Ana Paula Aquistapase Dagnino (PUCRS)

19h00-20h00 – **Closing Lecture**

Repositioning of drugs, a useful strategy in drug discovery and development

Eliezer Barreiro (UFRJ)

Presented by Marco Aurélio Martins (Fiocruz)

Lecture abstracts

Lectures

Targeting brain inflammation after stroke – from the laboratory to the clinic. Christopher G. Sobey[1], Thanh G. Phan[2], Henry Ma[2], Euan Wallace[2,3] and Rebecca Lim[2,3]. La Trobe University[1], Bundoora, Victoria, Australia; Monash University[2], Clayton, Victoria, Australia; Hudson Institute of Medical Research[3], Clayton, Victoria, Australia.

Stroke accounts for more than 10% of deaths worldwide, and over a third of survivors are left with major neurological impairment. The need for new and effective therapies for stroke is therefore clear and urgent. There is increasing interest in cell therapy as treatment modality in stroke, particularly for patients who are unable to receive endovascular clot retrieval or thrombolysis therapies, or for whom standard treatment has failed. Human amnion epithelial cells (hAECs) are non-immunogenic, non-tumorigenic, anti-inflammatory cells normally discarded with placental tissue following childbirth. We have shown that hAECs provide neuroprotection in models of stroke in mice and non-human primates. hAEC therapy attenuated infarct growth and/or promoted functional recovery, even when administered 1-3 days after the onset of stroke. The mechanisms of action involve modulation of the immune response to minimise further injury in the peri-infarct region, the so called 'inflammatory penumbra'. Unlike neuroprotection with unimodal action, multipotent hAECs have the ability to adapt their actions on the peri-infarct region over time, appropriate to the pathophysiological state of the target tissue. To translate these preclinical findings, we have commenced a Phase 1 dose escalation trial to assess the safety of hAECs in stroke patients to provide an evidence platform for future Phase 2 efficacy. Our protocol involves a modified 3+3 dose escalation study design with additional components for measuring magnetic resonance signal of efficacy and the effect of hAECs on immunosuppression after stroke. Patients are eligible if they have ischemic stroke in the territory of the middle cerebral artery, present within 24 h of stroke onset and are not eligible for thrombolysis or clot retrieval, aged 18-85 years, and have NIHSS stroke severity score of 6-15. Currently 6 patients have received 2-4 million cells/kg, with no adverse events for up to 14 months.

Funding: Australian National Health and Medical Research Council; Beluga Foundation.

Harnessing the chemistry behind host-parasite interactions to treat colitis. Toshio Arai, Elizabeth Siciliani, Armando Jardim and Fernando Lopes. Institute of Parasitology & Department of Microbiology and Immunology, McGill University, Montreal, Canada

The inflammatory bowel diseases (IBD), Crohn's disease and ulcerative colitis are characterized by relapsing symptoms of watery or bloody diarrhea, abdominal pain, fatigue, and weight loss, which often is devastating for the patients and their families. Developing nations have a lower incidence of IBD compared to developed nations, whereas Canada ranks as one of the nations with the highest incidences worldwide. Different hypothesis to account for this observation have been proposed. Among these is the suggestion that the absence of gastrointestinal parasites in Canadians may be a contributing factor for the higher prevalence. High levels of parasitic helminth infections in developing nations, potentially provides an evolutionary advantage for protection against chronic inflammatory diseases. My research seeks to elucidate signaling cascades or pathways that are controlled by molecules released by helminths in modulating inflammation. In contrast to previous studies on helminth molecules, my research focus on small helminth metabolites with immunomodulatory or tissue healing activity. Ultimately, this will provide novel pharmaceutical solutions to mimic the advantages that parasitic infections provide. Financial support: Fonds de Recherche du Québec – Nature et technologies; Natural Sciences and Engineering Research Council – Canada

Parkin: Novel insights on its transcriptional function. Checler Frédéric, Duplan Eric, Rouland Lila, El Manaa Wejdene and Alves da Costa Cristine. IPMC, UMR7275 CNRS-UCA, Sophia-Antipolis, 660 route de lucioles, 06560 Valbonne , France

Parkin is an E3-ubiquitin-ligase. This function conditions the cellular load of a subset of cytosolic proteins prone to proteasomal degradation and a loss of this function that yields an accumulation of potentially toxic substrates, at least partly, likely accounts for the neuronal loss observed in Parkinson's disease-affected substantia nigra. We previously showed that parkin also behaves as a transcription factor (*Da Costa et al. Nat. Cell Biol. 2009*). Thus, parkin was shown to repress the transcription of the tumor suppressor, p53. Further, parkin modulates the transcription of presenilins 1 and 2 that are the catalytic core of the g-secretase complex responsible for Ab production and subsequent senile plaques accumulation in Alzheimer's disease. Overall, this supports the bedrock hypothesis of our laboratory postulating common denominators involved in distinct pathologies and underpins the key role of parkin in neurodegenerative diseases. We will present recent structural and functional data further supporting the transcription factor function of parkin and will show that it could well explain various physiological functions that are altered in neurodegenerative diseases and cerebral cancer. This laboratory belongs to the laboratory of Excellence DistALZ (Development of Innovative Strategy for a Transdisciplinary Approach of Alzheimer's Disease). ED and EMW were supported by DistALZ and ANR, respectively. References: da Costa CA, Sunyach C, Giaime E, West A, Corti O, Brice A, Safe S, Abou-Sleiman PM, Wood NW, Takahashi H, Goldberg MS, Shen J, Checler F. (2009) Transcriptional repression of p53 by parkin and impairment by mutations associated with autosomal recessive juvenile Parkinson's disease. *Nat Cell Biol*. Nov;11(11):1370-1375.

Neuro-immune interactions and novel targets for chronic pain. Marzia Malcangio. Wolfson CARD, IoPPN, King's College London

Whilst acute pain is a warning mechanism that prevents tissue damage, chronic pain persists for months and reduces quality of life. In chronic pain states plastic changes occur in peripheral and central pain pathways, which lead to exaggerated responses to noxious stimuli (hyperalgesia) and non-noxious stimuli (allodynia). Immune cells contribute to chronic pain mechanisms by releasing mediators which sensitise neurons and enable positive feedback. The focus of this talk is on the plastic changes that occur under neuropathic pain conditions at nerve injury site, dorsal root ganglia (DRG) and dorsal horn of the spinal cord where endothelial damage and increased neuronal activity result in recruitment of monocytes/macrophages in the periphery and activation of microglia centrally. Our group has delineated a signalling pathway that mediates neuron-microglia communication in the dorsal horn of the spinal cord, namely the P_2X_7/Cathepsin S/Fractalkine/CX_3CR_1 pathway. In models of chemotherapy-induced pain, endothelial activation at the site of peripheral nerve injury results in recruitment of CX_3CR_1-expressing monocytes/macrophages, which sensitise nociceptive neurons through the release of reactive oxygen species that activate $TRPA_1$ channels and evoke a pain response. Very recently we have observed that following peripheral nerve injury, a neuro-immune cross talk takes place in the DRG and this is performed through the release of extracellular vesicles from neurons, which are engulfed by nearby macrophages. These vesicles deliver microRNAs (miRs) with the potential to afford long-term alterations in macrophages that impact pain mechanisms. Indeed, i) neuron-derived miR-21 engulfed by macrophages polarises these cells towards a pro-inflammatory/pro-nociceptive phenotype and ii) silencing miR-21 expression in sensory neurons prevents development of neuropathic allodynia and recruitment of macrophages in the DRG.

Definition of the modalities by which neuron and immune cells communicate under neuropathic pain states may enable the identification of novel targets for chronic pain. Supported by MRC, Versus Arthritis and EU ITN grant 764860.

From Glowing Penis to Oocyte-Producing Testis: The Power of Mouse Genetics on the Understanding of Organogenesis and Human Diseases. Humphrey Hung-Chang Yao, Ph.D. Senior Principal Investigator. Reproductive Developmental Biology Group, National Institute of Environmental Health Sciences, North Carolina, USA

Disorders of sex development, or birth defects of reproductive organs, affect about one in 4,000 babies. Defects in male reproductive system, such as hypospadias and cryptorchidism, are even more prevalent (~1 in 100-200). Familial studies and the advancement of genomic sequencing have helped scientists and clinicians identify potential genetic components of these disorders; however, pinpointing the exact underlying causes of the defects remains a challenge. My laboratory uses mouse embryos as the model to understand the basic mechanisms of sexual differentiation of reproductive organs. Combining tissue-specific genetic models (knockout and lineage tracing) and state-of-the-art single cell sequencing and light sheet imaging technique, we uncover a novel cell population responsible for proper penis morphogenesis and a regulatory pathway that control testis differentiation, respectively. The novel cell population migrates from outside of the penis and contributes to the proper closure of the urethra. Without this cell population, mouse embryos develop various degrees of hypospadias. In the testis, when the regulatory pathway is inactivated, a part of the testis turns into the ovary with the capacity to produce oocytes. These findings demonstrate the power of mouse genetics that enable us to not only understand the basic biology of male reproduction, but also provide potential candidates for human disorders of sex development. (Supported by NIEHS/NIH Intramural Research Fund in the USA)

Regulation of apoptotic cell clearance during resolution of inflammation. Adriano G Rossi, BSc, PhD, DSc, FRSB, FBPhS Deputy Director and Postgraduate Director of the University of Edinburgh Centre for Inflammation Research Professor of Respiratory and Inflammation Pharmacology

Host defence and beneficial inflammatory responses directed against invading organisms or trauma-induced tissue damage is orchestrated by leukocytes such as granulocytes (especially neutrophils and eosinophils) and macrophages. If the recruitment, activation and/or removal of such leukocytes from inflammatory sites is dysregulated, these cells have the potential to elicit and contribute to tissue damage found in patients with chronic inflammatory diseases (e.g., asthma, rheumatoid arthritis, atherosclerosis, multiple sclerosis, etc.). Resolution of inflammation is an active and regulated physiological process that terminates inflammation and limits tissue damage. Apoptosis and non-inflammatory phagocytosis of apoptotic cells by macrophages and other phagocytic cells (termed efferocytosis) are key cellular processes that lead to efficient inflammation resolution. Importantly, we contend that inflammation resolution processes dictate successful tissue repair and regeneration. Using a combination of primary human leukocytes, mouse and zebrafish models, together with state-of-the-art equipment and technology we show that resolution of inflammation can be enhanced pharmacologically and genetically to promote tissue repair and regeneration. Such approaches that elucidate underlying mechanisms and processes involved in inflammation resolution, we believe, will lead to the development of novel therapeutic strategies for the treatment of inflammatory diseases.

Pharmacogenomic related a drug adverse reaction. Prof. Dra. Patricia Moriel. Faculdade de Ciências Farmacêuticas da UNICAMP

Adverse drug reactions (ADRs) are a significant health concern worldwide. ADRs remain a challenge in modern healthcare, particularly given the increasing complexity of therapeutics, an aging population, and rising multimorbidity. There are multiple causes of ADRs, some of which are preventable. Genetic susceptibility is an essential feature of severe adverse drug reactions. There is considerable interest in the possibility that the development of genetic tests to identify all those at risk of adverse events prior to prescription might lead to valuable drugs being retained. The highest genetic variability in response to drugs is multifactorial, influenced by numerous genes with compensation or overlapping of functions. Pharmacogenomics uses the complete genome as an approach, including evaluating gene expression and all paths of variability in response to drugs. Pharmacogenomics can also be defined as the combination of pharmacology and genomics to advance research and development of drugs and manage their selection and dosage. The genetic variation in genes involved in drug metabolism can decrease the functional activity or expression of the metabolizing enzymes, thus giving rise to distinct individual phenotypes ranging from slow metabolizer, intermediate metabolizer, fast metabolizer, and ultra-fast metabolizers. Differences in individual phenotypes have an essential impact on the pharmacokinetics of the drug and, therefore, its effectiveness and safety. Pharmacogenetic/pharmacogenomic studies have shown that the detection of polymorphisms in enzymes, transporters and/or receptors that participate in the metabolization of different drugs significantly contribute to a more favorable response to the drug, thus increasing the effectiveness and safety of the treatment and reducing associated costs. Pharmacogenomics accounts for ≈80% variability in drug efficacy and safety. Over 400 genes are clinically relevant in drug metabolism, and ≈200genes are associated with ADRs. In this presentation, we will present the main genes involved in ADRs and exemplify with drugs that are known to use pharmacogenomics to improve safety in their use. Financial Support: FAPESP/CAPES/CNPq

Repositioning of drugs, a useful strategy in drug discovery and development. Eliezer J. Barreiro. LASSBio & INCT-INOFAR Federal University of Rio de Janeiro, Institute of Biomedical Sciences, Cidade Universitária, Rio de Janeiro, RJ. Brazil

Drug repositioning (D-Re), also referred to as drug repurposing, has become an increasingly important part of the drug discovery and development (*DDD*) process in recent years, special nowadays due SARS-CoV-2 pandemia. The strategy of identifying new therapeutic indications for existing drugs, following the Sir Black dictum "The most fruitful basis for the discovery of a new drug, is to start from with an old drug", represents part of incremental innovation strategy in *DDD* process.

In this talk it will be treated from general aspects through classical examples to more recent and specific examples of D-Re to demonstrate their usefulness of *DDD* process including very recent in-house research results by applying repurposing of chemical library of bioactive compounds in a medicinal chemistry project that has a main goal to identify new hits for one SARS-CoV-19 target.

Symposia and Roundtables

Science allied to sport: Doping control. Pedro Antônio Castelo Teixeira (UFRJ)Federal University of Rio de Janeiro - UFRJ, Brazilian Doping Control Laboratory - LBCD, LADETEC, Av. Horácio Macedo, 1281 - Polo de Química - Bloco C - Cidade Universitária - Ilha do Fundão - Rio de Janeiro, Brazil.

Doping and its control are presented in a historical perspective. Prohibited substances and methods used by some athletes to increase sport performance are discussed. Information regarding sophisticated doping means and the evolution of the analytical techniques to control them are briefly introduced. The doping control system, according to the World Anti-Doping Agency (WADA), as performed in the Brazilian Doping Control Laboratory (LBCD), is shown, as well as its complexity. Famous and interesting athletes doping cases are exhibited and discussed. The science allied to sport, in the form of doping control, is intended to preserve the physical and mental integrity of the athletes.

MSL: Assignments and Perspectives. Leandro Ladislau Alves Daiichi Sankyo Brazil

The development of innovative treatment to improve quality of life and increase patient survival has raised unmet need for qualified professional in pharmaceutical and biotechnology companies around the world. This professional, called Medical Science Liaison (MSL) has the main assignment disseminate the scientific information regarding biotechnology and new drugs that are necessary for medical doctors choose the best option for the patient between all available drug arsenal. The MSL position emerged in 1960 at United States with the main role make the bridge between the pharma industry and medical community – main key opinion leaders (KOL) – providing scientific information. Nowadays, the MSL´s are from healthcare professional, must have a solid scientific formation usual with PhD (sometimes medical doctors degree are mandatory) and should have good soft skill in relationship. The role of MSL has specifics assignments work that differs from sales customer. First, there is no sales responsibility, second providing scientific information and third building relationships with KOL´s. Other assignments are typical for MSL as routine of travel, a lot of meeting (congress, group and personal), presentations (group and personal) and study update. As the MSL profile and assignments are compatible with academic research many PhD have awake for the role. Nevertheless, the shift from academic to business mind in search process to find the first MSL position could be painful and prolonged. The person must craft an attractive

curriculum vitae, create an important networking (use social media as LinkedIn), search the match job announcement (match with the scientific formation) and create knowledge of the hiring process (start to think the business world). Finally, healthcare PhD professionals are scientific ready to be MSL but should change the mindset before starting in the path to search the first position. It is really important during the hiring process know (at least) about the MSL role, the day-by-day of the MSL, study about human resources interview (create business mindset), have a business speech (avoid the academic speech) and learn about the company (mission and values).

Patent: The intellectual property right (IP) that transforms the world Anicet Okinga (INPI) Instituto Nacional da Propriedade Industrial_INPI, Rua Mayrink Veiga 9, Centro, Rio de Janeiro, Brasil.
Industrial Property Researcher (Patent Examiner) in the Division of Pharmacy. Intellectual property is the field that comprises all exclusive rights granted to intellectual creations. In addition to encouraging innovations and creating favorable conditions to encourage creativity, the main objective of protecting the intellectual property is the guarantee of authors' property rights and the right to take legal action against anyone who violates the protection and use it without prior permission. Industrial Property - one of the branches of Intellectual Property - is related to patents, trademarks, industrial designs, geographical indications, industrial secrets and to the rights to repression of unfair competition. The patent is a property right that, since the Industrial Revolution, has transformed our planet into practically all sectors of life.

Advances in neural circuitry for thermoregulation and fever. Christopher J. Madden
Department of Neurological Surgery, Oregon Health & Science University, Portland, Oregon, USA
The production of heat in brown adipose tissue (BAT) is a significant component of the homeostatic regulation of body temperature during low environmental temperature and plays a key role in elevating body temperature during the febrile response to infection. The sympathetic neural outflow determining BAT thermogenesis is primarily regulated by the central nervous system in response to cutaneous and deep body thermoreceptor inputs. Since energy consumption during BAT thermogenesis involves oxidation of lipid and glucose fuel molecules, the regulation of BAT thermogenesis in response to metabolic signals can contribute to energy balance, regulation of body adipose stores and glucose utilization. This symposium will summarize our understanding of the functional organization and neurochemical influences within the central nervous system networks that modulate the level of BAT sympathetic nerve activity to produce alterations in BAT thermogenesis. Based on novel aspects of the neurochemical regulation of BAT thermogenesis this symposium will also highlight several pharmacological agents whose use may be effectively repurposed for therapeutic hypothermia, antipyresis, or obesity. This work was supported by a grant from the National Institutes of Health R01-DK112198.

Thermoregulatory and neuroimmunomodulatory effects of serotonin. Luiz G. S. Branco. Department of Basic and Oral Biology, Dental School of Ribeirão Preto, University of São Paulo, Ribeirão Preto, SP, Brazil.
Serotonin or 5-hydroxytryptamin (5-HT) arises from tryptophan metabolism and plays several roles in the brain (mediating behavior and sleep, for instance) and peripheral organs (as controlling gastrointestinal function). Needless to say, serotonin reuptake inhibitors have been successfully used to treat depressive disorders. More recently studies have suggested that immune system activation may also alter brain 5-HT signaling. Here we report data consistent with the notion that central 5-HT modulates systemic inflammation (SI), as observed during sepsis. An exceptionally high mortality rate is observed in sepsis and septic shock. Systemic administration of lipopolysaccharide (LPS) has been used as an experimental model for sepsis resulting in an exacerbated immune response, brain neurochemistry adjustments, hypotension, and hypothermia followed by fever. We sought to determine if central 5-HT plays a role in SI induced by intravenous administration of LPS (1.5 mg/kg) in male Wistar rats by assessing hypothalamic 5-HT levels, mean arterial pressure, heart rate, body temperature, plasma and spleen cytokine levels, nitric oxide (NO) plasma levels, and prostaglandin (PG) E_2 levels in the hypothalamus. We observed reduced hypothalamic 5-HT levels, hypotension, tachycardia, hypothermia followed by fever, as well as increased plasma NO, plasma and spleen cytokines and hypothalamus PGE_2 levels in SI. Intracerebroventricular (icv) administration of 5-HT 30 min before LPS administration prevented hypotension and hypothermia, which were accompanied by reduced plasma NO, as well as plasma TNF-α, IL-1β, IL-6, and IL-10 and spleen TNF-α and IL-10 surges. We suggest that SI causes a reduction in 5-HT levels in the hypothalamus favoring an increased pro-inflammatory status both centrally and peripherally that converge to hypotension and hypothermia. Interestingly, exogenous 5-HT given icv prevents hypotension and hypothermia probably activating the splenic anti-inflammatory pathway.
Support: FAPESP, CNPq.

Experimental febrile response in females: influences of estrous cycle and ovariectomy. Aleksander Zampronio. Department of Pharmacology, Biological Sciences Center, Federal University of Paraná, PR, Brazil
The febrile is a well-known response and an important defense response against infection. However, most of the present knowledge on how this response occurs comes from studies in male rats and mice. Several studies have shown that important differences arise in inflammatory/immune response between males and females. This presentation will summarize our current knowledge about the differences observed in the febrile response between males in female rats. This sex differences will be explored in terms of some of the mediators involved in fever in males and females, particularly involving prostaglandin E_2, endothelin-1 and the ligand for the receptor

activator of nuclear factor κB (RANK), its ligand (RANKL), and its decoy receptor osteoprotegerin (OPG). In this context, differences related to the estrous cycle phases and the characteristics of the febrile response inducer (bacterial, viral and fungal origin) will be highlighted and discussed. The importance of sexual female hormones, particularly estrogen, will also be explored showing the febrile response in ovariectomized animals as a model of menopause. Support: Fundação Araucária and CNPq

Brain neurosteroids and mood disorders- Helena Maria Tannhauser Barros (UFCSPA) There is influence of neurosteroids in mood disorders, from a behavioral and neurochemical point of view, considering the main diseases involved with changes in the level of neurosteroids, in male and female individuals and the effect promoted by the change in the levels of neurosteroids in these pathologies. The clinical and preclinical aspects related to the influence of neurosteroids in the main mood disorders will be addressed, outlining a comprehensive map of the participation of these endogenous substances in brain activity.

Brain neurosteroids in stress disorders- Maurício Schuler Nin (IPA); In this segment, the main clinical and preclinical results involved with the anxiolytic and antidepressant effect of the main neurosteroids will be presented, with the main focus of allopregnanolone, associated with these disorders, as well as the fluctuation of the level of these substances related to these disorders. The direct effect of the fluctuation of neurosteroids on PTSD will be addressed, as well as the prospects for treatment with substances that directly or indirectly alter the endogenous level of neurosteroids.

Neurosteroids levels in stress and mood disorders -Graziano Pinna (University of Illinois at Chicago, USA). As a sequence, the neurogenic effect of substances associated with mood disorders and the neurosteroids associated with these fluctuations will be addressed. Biomarkers discovery in blood represents a fundamental tool to predict, diagnose, and monitor treatment efficacy in depression and PTSD. Dr. Pinna will discuss the advances in novel neurobiological methods that may improve the assessment and diagnosis of anxiety, depression, and PTSD, which offers a new means of appropriately treating patients. The future of blood-based tests for PTSD and depression is not only on the horizon but, possibly, already around the corner.

Age as a Factor in the Safe Management of Chronic Pain. Jason J. McDougall, Dalhousie University, Halifax NS. Canada

Chronic pain can strike in any age group but tends to become more prevalent as we get older. The most common class of drugs taken by the elderly are the non-steroidal anti-inflammatory drugs (NSAIDs) followed by acetaminophen and then opioids. As we age our bodies lose lean muscle mass and total body water while our bones and organs shrink in size. Basal metabolic rate decreases, fat content increases, and renal and hepatic function decline. Taken together, these normal changes with age affect drug metabolism, pharmacokinetics, and efficacy. Renal clearance decreases by approximately 10% per decade after the age of 30 years. By age 70 years, renal function could have reduced by up to 50% which will significantly affect the elimination of drug metabolites. Hepatic function also declines with age while the effectiveness of gastrointestinal absorption is also compromised. For example, NSAIDs are almost exclusively metabolized by cytochrome P450 in the liver whose activity changes with age. Analgesic efficacy is reduced in older populations and this may be due to changes in pain processing and nerve fibre density. The algogenic neuropeptide substance P typically sensitizes nociceptors leading to the generation of heightened pain. However, in preclinical studies it was found that the sensitizing effect of substance P was attenuated in aged animals compared to young ones. This may explain some of the failure of neurokinin-1 receptor antagonists which have yet to show efficacy in clinical trials. Older adults are more inclined to be on multiple medications and drug-drug interactions become a greater concern. Qunidine, for example, ameliorates the analgesic capacity of codeine by inhibiting its metabolism into active agents. This, when developing and testing new drug entities for the management of chronic pain conditions, age is an important consideration.

Pre-clinical and clinical evidence on the effectiveness of peptide ligands in chronic pain states. Ana Paula Aquistapase Dagnino. Programa de Pós-graduação em Medicina e Ciências da Saúde, Escola de Medicina, PUCRS. Fibromyalgia is a nociplastic pain type, accompanied by functional and affective disorders, such as fatigue, depression and anxiety. The pathophysiology of fibromyalgia remains unraveled, and some clinically used drugs are ineffective or even increase of fatigue symptoms and cognitive deficits. Therefore, further studies are necessary to identify the mechanisms underlying fibromyalgia pathogenesis and potential targets for novel therapies. The repeated administration of reserpine lead to monoamine depletion, triggering fibromyalgia-like symptoms in rodents. Recent studies investigated the relevance of peptide receptors in this pre-clinical experimental model. It has been suggested that both B_1R (bradykinin B_1 receptor) and B_2R (bradykinin B_2 receptor) display a relevant role in fibromyalgia painful symptoms in mice. The authors reported that mechanical allodynia induced by reserpine was decreased in $B1R^{-/-}$ and $B2R^{-/-}$ mice, or after treatment of wild-type animals with B_1R (DALBK) and B_2R (Icatibant) antagonists. The expression of both receptors was increased in the spinal cord and cerebral cortex of reserpinized mice. In addition, the administration of reserpine increased bradykinin levels in the sciatic nerve and cerebral cortex. Recently, our group provided novel evidence on the mechanisms underlying the fibromyalgia pathogenesis, supporting a role for nociceptin receptor (NOPr) in this syndrome. The intraperitoneal treatment with selective NOPr antagonist UFP101 reduced the mechanical allodynia, thermal hyperalgesia and improved the motor coordination. This antagonist was also effective in reducing fatigue. Reserpine-induced fibromyalgia was associated with an increase in nociceptin mRNA expression in the lumbar

spinal cord and masseter, whereas NOPr mRNA expression was increased in the masseter muscle. Alternatively, NOPr mRNA expression was reduced in the thalamus/hypothalamus. Immunohistochemistry revealed an increased expression of NOPr in the dorsal root ganglion. Additionally, UFP101 was able to recover the brain hypermetabolism and the structural changes of skeletal muscle in this experimental paradigm. It is tempting to propose that antagonists of peptide receptors might represent useful alternatives for management of fibromyalgia. Financial support and acknowledgments: FINEP, CAPES, CNPq, PUCRS

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