NEWSLETTER

SOCIEDADE PORTUGUESA DE BIOQUÍMICA

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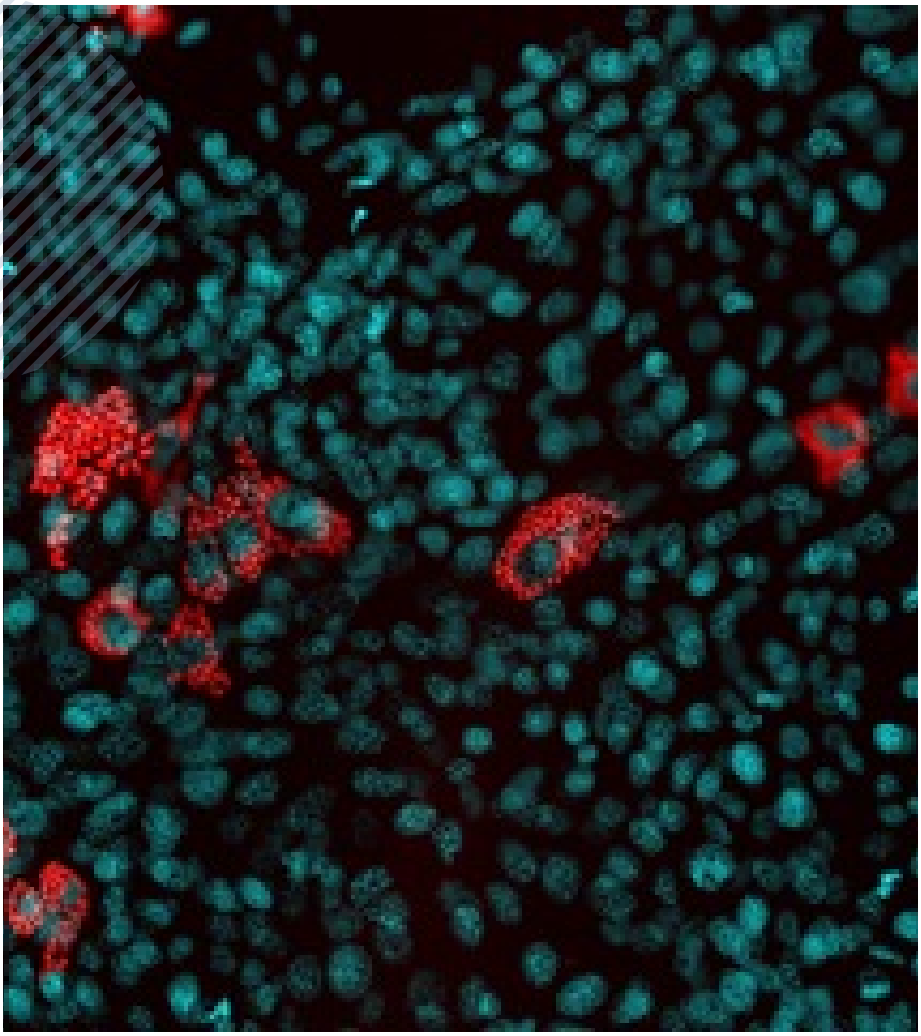


Human cells infected with Trypanosoma cruzi (red). Nuclei are stained with DAPI (cyan).

Carolina Vieira, Católica Biomedical Research Centre, Católica Medical School, Universidade Católica Portuguesa

IN THIS EDITION

Editorial

12th Edition

In this Edition:

In this issue of the SPB Newsletter, we feature Alexandre Quintanilha of the University of Porto, who reflects on his academic career, highlights his scientific activities, and discusses his contributions to the SPB.

Carolina Vieira and Sara Silva Pereira, from the Católica Medical School, share their insights into how T. *cruzi* survives exposure to reactive oxygen species.

This newsletter also highlights the representation of Portugal in the FEBS Education and Training Conference and in the 50th FEBS Congress, as well as upcoming events, including the XXIII National Congress of Biochemistry.

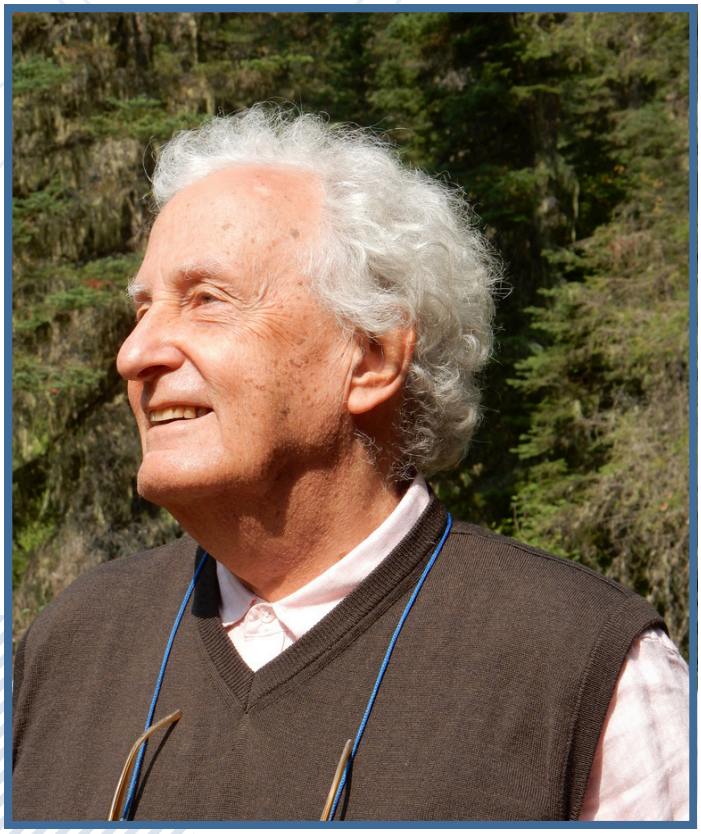
We look forward to seeing you in Covilhã!

The SPB Directive Committee

Interview

Alexandre Quintanilha

For this edition's Newsletter, we talked with Alexandre Quintanilha, Full Professor (retired) at ICBAS, University of Porto.



We'll begin by asking for a brief summary of your career and what motivated/attracted you at each stage.

At the end of high school in Lourenço Marques (now Maputo), my favourite discipline was descriptive geometry. The beauty and the elegance of the solutions to the problems that we were given was very rewarding. In the final exam I obtained the grade of 20 (out of 20).

At that time, in 1962, this was almost unheard of! But I also liked mathematics, physics and the life sciences. At the University of the Witwatersrand in Johannesburg, I pursued a degree in Physics and Mathematics.

Halfway through my degree, I decided to take one year off to study philosophy, French and German literature. A very wise decision. My Ph.D. supervisor, Frank Nabarro, was one of the leading figures in the solid-state theory of crystal dislocations, and my thesis was on the pinning of superconducting flux lines by crystal dislocations. I realised then that my interests were always at the interface of different domains. Superconductivity and the elasticity in solids were an obvious example.

The inspirational meeting with Sydney Brenner towards the end of my Ph.D. was what made me move toward the life sciences. I joined the Membrane Bioenergetics Group in Berkeley to start working on mitochondria and other energy converting bio-systems.

I studied the generation of electric gradients and production of oxygen free radicals. The Berkeley Campus and the adjoining Lawrence Berkeley National Laboratory (LBNL) allowed me to pursue many collaborations. At the end of my post-doc, I was asked to put together a multidisciplinary Centre to study the Atmospheric and Biospheric Effects of Technology and become the Assistant Head of the Applied Science Division at LBNL.

Not wanting to give up teaching, I continued to serve as an adjunct professor of physiology and biophysics on the Campus. All the projects that I became involved with were in interdisciplinary domains. I studied oxidative stress under many physiological and pathological conditions but was also involved in the development of a variety of novel spectroscopic tools. We also launched a Graduate Program in Applied Science between the Campus and LBNL.

After almost two decades in Berkeley, we moved to Porto. As a professor of biophysics at ICBAS, the major challenge was presiding over the launch of the IBMC, followed by the Associated Laboratory IBMC-INEB and later the i3S consortium. I also played a role as co-founder of the undergraduate degrees of Veterinary Medicine and Bioengineering as well as the first Gulbenkian Doctoral Programme in Medicine and Biology. Always expanding the opportunities for interdisciplinary research and innovation.

Forced to retire at age 70, I accepted the challenge of becoming a Member of Parliament and to chair the Commission on Education and Science, were I played major roles in the Climate Basic Law and the Euthanasia Law. These were and continue to be key societal concerns.

What motivated you to start working on bioenergetics? Was it driven by an interest in applying physical principles to explain this biological process?

I went to Berkeley after becoming aware of the groundbreaking studies being conducted on mitochondria and chloroplasts. I was fascinated by the idea that a flow of electrons could drive proton pumping and that an extraordinary protein could use the resulting proton gradient and convert it into chemical energy stored in the form of ATP. Suddenly, I became aware of exciting links between physics and biology.

My move to the Bioenergetics group, however, was also strongly influenced by my conversations with Sydney Brenner, who himself had a profound appreciation for Physics. His intellectual curiosity and interdisciplinary perspective reinforced my conviction that the fundamental principles of physics could provide powerful insights into biological systems.

What inspired you to work on oxidative stress?

I was intrigued by the paradox that oxygen is both vital to our lives and potentially toxic. The oxidation of biological molecules can cause damage, and we need a vast array of defense and repair mechanisms to counteract oxidative damage.

At the same time, reactive oxygen species, at low levels, act as signaling molecules, inducing protective responses to increase cellular resistance. Plus, the oxidation of drugs can facilitate its detoxification and clearance.

I like to use these ideas to illustrate the price that we must pay to have much more efficient metabolic pathways. Understanding how life manages this delicate balance remains one of the most fascinating aspects of biology.

Did your interest in the risks posed by free radicals influence your broader interest in risk perception?

Yes, it is related to the observation that free radicals of oxygen can become detrimental during strenuous physical exercise. There is evidence suggesting that athletes engaged in prolonged, intense training live fewer years than individuals who perform regular, moderate exercise. In other words, the risks may be linked to the price we pay for certain physiological advantages.

In general, the risk is associated with the unknown. During strenuous exercise, our studies have shown that the liver becomes depleted of glutathione, one of the most important antioxidants involved in the mechanisms of drug detoxification that the liver is responsible for.

This occurs because glutathione is released from the liver to help protect muscles against oxidative stress. This finding was quite surprising. Later, Helmut Sies demonstrated that glutathione depletion in the liver is hormone (e.g., vasopressin) regulated. However, very little is still known about these effects on the integrity of the liver and the ageing of our organs.

What other discoveries have surprised you the most?

I found it particularly interesting that visible light, especially blue light, can inactivate the respiratory chain of mitochondria. We were the first to show that this inactivation was because flavins absorb blue light, leading to their degradation. This observation has broader implications, especially considering the growing awareness of the biological effects of blue light emitted by electronic screens.

The ability to follow, in real time, the dynamics of intracellular polymerization of haemoglobin S in red blood cells of sickle cell anaemia patients, using novel spectroscopic tools was also very exciting.

Would you like to mention other contributions? What about your contributions to scientific societies, namely to SPB?

As president of SPB, in addition to strengthening the link with our Spanish colleagues, I also encouraged Claudina Rodrigues Pousada to get more involved.

She was responsible for developing a much stronger FEBS connection as well as providing greater visibility to the role of women in science.

She was deeply committed to SPB and succeeded me as president of the Society. I also invited Mina Bissell to give one of the most memorable key lectures at one of our yearly meetings. She was a pioneer in demonstrating that mammary cells in vitro can be made to form three-dimensional gland-like structures resembling normal breast tissue. This groundbreaking work laid the foundation for the development of mammary organoid research.

As a long-standing member of Academia Europæa, the World Academy of Arts and Science and more recently the Academia das Ciências de Lisboa, I have strived to defend the role of fundamental science in our society.

What was your greatest professional challenge?

Probably switching from solid state physics to animal physiology. I still remember the whole lab in Berkeley watching how a theoretical physicist kills a rat to prepare liver mitochondria. After the slaughter, I froze. A few weeks later I was preparing mitochondria every day.

Moving to Portugal in 1990, with Richard, was also a major challenge. I had spent almost 30 years of my initial professional life in the "Anglo" world and adapting to the "Luso" world was not easy. I am still trying.

Having the opportunity initially to collaborate with several research groups working on oxidative stress led by Pedro Moradas Ferreira and Francisco Carvalho Guerra made it easier to feel integrated with the scientific community of Porto.

What do you see as the main challenges facing bioscience research and education in the coming years?

What worries me most is the recurrent idea that priority should be given to knowledge that has immediate applications and is economically profitable. In fact, many of the most successful private companies have realized that basic research is key to innovation.

Innovation should not be seen merely as something that delivers immediate applications and generates profit. There must be a balance between fundamental and applied science, as the two are mutually reinforcing.

Clear examples include the vaccines developed during the COVID-19 pandemic, which were built upon research conducted 20 years earlier, and the Maxwell's equations, which were fundamental to the development of electronics.

Many applications emerge from fundamental discoveries and, in turn, raise new questions that can only be answered through basic research.

The most inspiring figures in scientific development have always understood this. Mariano Gago was one such person, arguing that all fields of knowledge are important and should receive equal support, and promoting scientific literacy through the creation of Ciência Viva.

What opportunities within SPB would you recommend to scientists at yours or earlier career stages?

Try to find questions at the interface of different domains within, but also outside, biochemistry. It is never easy, always risky, but very worthwhile.

I have done so all my life and am very grateful that my parents and some of my most memorable teachers have encouraged me to do so.

News & Views

Too much ROS will kill you, except if you're a parasite.

Carolina Silva Dias Vieira and **Sara Silva Pereira,** Católica Biomedical Research Centre, Católica Medical School, Universidade Católica Portuguesa, Portugal.

Carolina Silva Dias Vieira

Sara Silva Pereira

Reactive oxygen species (ROS) are usually presented as "toxic byproducts" of aerobic metabolism that damage cells, drive aging, and contribute to disease. Yet, in many parasite-host systems, ROS play a double role: they are not just weapons used by host immune cells to kill parasites, but also exploited as signals to drive important processes for their life cycles.

Understanding this dual nature of ROS in parasite biology is essential for both the advance of basic biochemistry and the design of new antiparasitic strategies.

What are ROS and where do they come from?

Aerobic respiration allows organisms to efficiently extract energy from organic molecules. During this process, electrons from these molecules are transferred in the electron transport chain, promoting the complete reduction of the molecular oxygen (O_2) to water. However, a fraction of the electrons leaks from this system and reacts with O_2, generating ROS.

ROS are highly reactive molecules derived from the partially reduced O_2 and include both radical species (such as superoxide, $O_2^{\bullet -}$; hydroxyl radical, $^{\bullet}OH$; alkoxyl, $RO^{\bullet}$; and peroxyl, $ROO^{\bullet}$) and non-radical species (such as hydrogen peroxide, H_2O_2; hypochlorous acid, HOCl; and singlet oxygen, $^1O_2$). Free radicals are molecules missing an electron, which makes them unstable. To fix this, they steal electrons from nearby compounds.

As consequence, those compounds turn into new free radicals, setting a domino effect of reactions that ultimately leads to cellular damage (1).

At constantly high concentrations, ROS react with lipids, proteins, and nucleic acids, resulting in cell membrane damage, enzyme inactivation, mutations, etc., which forms the basis of the classical "oxidative stress" scenario that often leads to cell death. Interestingly, phagocytes use the same principle to kill pathogens, launching a "respiratory burst" that fills pathogens with ROS (2).

The Redox Paradox

In healthy conditions, cells use its antioxidant defense system to prevent oxidative stress. This includes a network of enzymatic and non-enzymatic antioxidants that work to neutralize excessive ROS. At lower levels, ROS reversibly oxidize cysteine residues in some target proteins, inducing conformational changes and modulating their activity, thereby propagating redox signaling cascades that can stimulate or inhibit cellular processes. Thus, ROS can mediate either oxidative stress or redox signaling, depending mostly on their concentration, type, and subcellular localization (2) (Figure 1).

Aerobic respiration in mitochondria consumes 90% of the body's oxygen. About 1-2% of this oxygen is naturally converted into superoxide, which makes this organelle the main site of ROS production in aerobic cells (2,3). Besides, other cellular compartments (like peroxisomes and the endoplasmic reticulum) can produce ROS, as can membrane-bound NADPH oxidases (NOX enzymes).

However, contrary to the classic "ROS equal death" picture, several important pathogens not only survive in this hostile environment, but replicate in the presence of abundant ROS, using specialized antioxidant systems to control their levels and, in some cases, exploiting ROS as signals for their own processes.

Trypanosoma cruzi, a vector-borne pathogen that causes Chagas disease, provides one of the clearest examples. When transmitted to mammals, macrophages are among the first cells to be infected. In response, the macrophage oxidative burst generates ROS and nitric oxide, which were long assumed to eliminate the intracellular parasite.

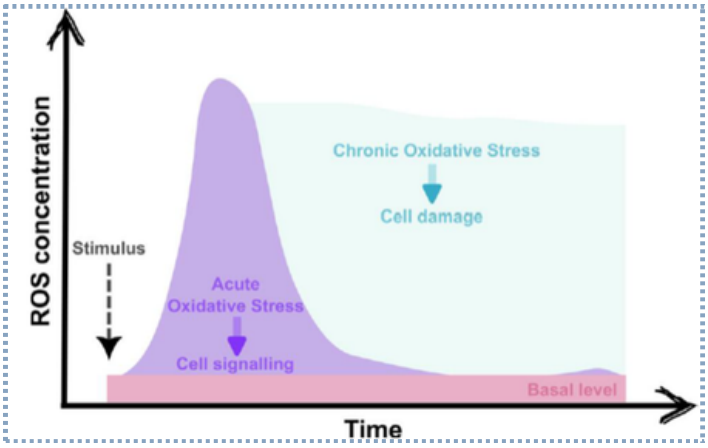


Figure 1. ROS act as concentration- and time-dependent mediators of cellular signaling. At low levels, ROS maintain basal redox homeostasis. A controlled and transient increase of specific ROS induces a temporary imbalance that drives reversible redox signaling, modulating signal transduction and gene expression. In contrast, constant or excessive ROS production disrupts this balance, leading to persistent alterations that can promote pathological results.

However, this hostile oxidative environment fuels the parasite's growth, increasing parasitemia in rodent models, while the presence of antioxidants that scavenge host-derived ROS reduces parasite burden and infection (4,5). The same trend is observed in the insect vector: the replicative stage proliferates more rapidly under oxidative than reductive conditions.

These parasites proliferate in high levels of H_2O_2 and heme, whereas antioxidant molecules block growth *in vitro* and *in vivo*. While these examples involve exogenous ROS, endogenous signaling plays a major role as well.

Heme induces parasite replication by compromising its mitochondrial respiratory system. This metabolic perturbation drives an increase in H_2O_2 levels, which directly stimulates proliferation, while removing this oxidative signal reverses elevated growth (6,7,8).

In the mammalian host, H_2O_2 induces parasite differentiation into intracellular stages, contributing to infection persistence (5,9) (Figure 2).

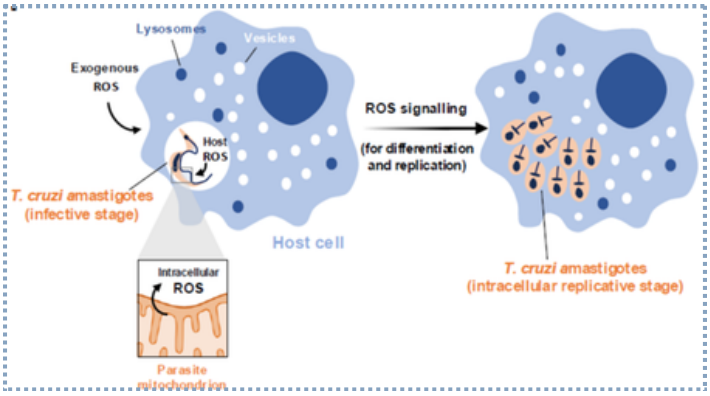


Figure 2. Multiple ROS sources fuel T. *cruzi* infection in host cells. Exogenous ROS, host-derived ROS (*e.g.*, from macrophage respiratory bursts, cardiomyocytes, and other host cells) (5), and parasite-generated ROS from mitochondrial and other metabolic pathways all contribute to redox signals that stimulate differentiation toward intracellular stages during mammalian infection. This ROS-driven differentiation increases parasite burden and promotes disease progression.

But how can *T. cruzi* survive ROS?

This resistance to oxidative environments has potentially been shaped by continuous redox pressures throughout the parasite's life cycle. To cope, persist, and proliferate within these hostile conditions, *T. cruzi* relies on a unique trypanothione-based redox system.

Analogous to the antioxidant glutathione (GSH) in mammals, this pathway centers on trypanothione itself, alongside with key enzymes, such as trypanothione synthetase and trypanothione reductase (10,11,12).

T. cruzi has mastered the art of living on the edge, using host-derived ROS to fuel its own life cycle. However, this act relies entirely on its specialized trypanothione shield.

By developing drugs that inhibit this unique network, we can strip away the parasite's armor and let the host's immune system do what it was meant to do. Eventually, and with a little help, too much ROS really will kill you, even if you are a parasite.

References

1) Phaniendra A, Jestadi DB, Periyasamy L. Free radicals: properties, sources, targets, and their implication in various diseases. Indian J Clin Biochem. 2015;30(1):11-26. doi: 10.1007/s12291-014-0446-0.

2) Checa J, Aran JM. Reactive Oxygen Species: Drivers of Physiological and Pathological Processes. J Inflamm Res. 2020;13:1057-1073. doi: 10.2147/JIR.S275595.

3) Murphy MP. How mitochondria produce reactive oxygen species. Biochem J. 2009;417(1):1-13. doi: 10.1042/BJ20081386.

4) Paiva CN, Feijó DF, Dutra FF, Carneiro VC, Freitas GB, Alves LS, Mesquita J, Fortes GB, Figueiredo RT, Souza HS, Fantappié MR, Lannes-Vieira J, Bozza MT. Oxidative stress fuels *Trypanosoma cruzi* infection in mice. J Clin Invest. 2012;122(7):2531-42. doi: 10.1172/JCI58525.

5) Paiva CN, Medei E, Bozza MT. ROS and *Trypanosoma cruzi*: Fuel to infection, poison to the heart. PLoS Pathog. 2018;14(4):e1006928. doi: 10.1371/journal.ppat.1006928.

6) Nogueira NP, Saraiva FM, Sultano PE, Cunha PR, Laranja GA, Justo GA, Sabino KC, Coelho MG, Rossini A, Atella GC, Paes MC. Proliferation and differentiation of *Trypanosoma cruzi* inside its vector have a new trigger: redox status. PLoS One. 2015;10(2):e0116712. doi: 10.1371/journal.pone.0116712.

7) Nogueira NP, de Souza CF, Saraiva FM, Sultano PE, Dalmau SR, Bruno RE, Gonçalves Rde L, Laranja GA, Leal LH, Coelho MG, Masuda CA, Oliveira MF, Paes MC. Heme-induced ROS in *Trypanosoma cruzi* activates CaMKII-like that triggers epimastigote proliferation. One helpful effect of ROS. PLoS One. 2011;6(10):e25935. doi: 10.1371/journal.pone.0025935.

8) Nogueira NP, Saraiva FMS, Oliveira MP, Mendonça APM, Inacio JDF, Almeida-Amaral EE, Menna-Barreto RF, Laranja GAT, Torres EJL, Oliveira MF, Paes MC. Heme modulates *Trypanosoma cruzi* bioenergetics inducing mitochondrial ROS production. Free Radic Biol Med. 2017;108:183-191. doi: 10.1016/j.freeradbiomed.2017.03.027

9) Paula JIO, Pinto JDS, Rossini A, Nogueira NP, Paes MC. New perspectives for hydrogen peroxide in the amastigogenesis of *Trypanosoma cruzi* in vitro. Biochim Biophys Acta Mol Basis Dis. 2020;1866(12):165951. doi: 10.1016/j.bbadis.2020.165951.

10) Maldonado E, Rojas DA, Morales S, Miralles V, Solari A. Dual and Opposite Roles of Reactive Oxygen Species (ROS) in Chagas Disease: Beneficial on the Pathogen and Harmful on the Host. Oxid Med Cell Longev. 2020;2020:8867701. doi: 10.1155/2020/8867701

11) Mesías AC, Sasoni N, Arias DG, Pérez Brandán C, Orban OCF, Kunick C, Robello C, Comini MA, Garg NJ, Zago MP. Trypanothione synthetase confers growth, survival advantage and resistance to anti-protozoal drugs in *Trypanosoma cruzi*. Free Radic Biol Med. 2019;130:23-34. doi: 10.1016/j.freeradbiomed.2018.10.436.

12) González-Montero MC, Andrés-Rodríguez J, García-Fernández N, Pérez-Pertejo Y, Reguera RM, Balaña-Fouce R, García-Estrada C. Targeting Trypanothione Metabolism in Trypanosomatids. Molecules. 2024;29(10):2214. doi: 10.3390/molecules29102214.

Events

FEBS Education and Training Conference

Margarida Fardilha, University of Aveiro, and **Manuel João Costa**, University of Minho

Portugal was well represented at the second FEBS Education and Training Conference, held in Kuşadası, Türkiye, from 25th to 29th March 2026, with a delegation of five participants who contributed through a number of notable lectures and workshops.

Organized by FEBS through its Education and Training Committee, the conference has established itself as the leading European platform for innovation in education and training in the Molecular Life Sciences, bringing together around 200 participants from across the world.

The conference also hosted the FEBS Academy, a training initiative based on small-team learning that fostered the exchange of experiences, best practices, and skills development among educators and scientists.

The programme featured plenary lectures, thematic sessions on classroom innovation, artificial intelligence in education, and career development, as well as short presentations and interactive formats such as the "Debate of the Day".

The event provided valuable networking opportunities, and the lectures and workshops were of exceptional quality.

More information about the conference can be found at: https://febsetc.org/

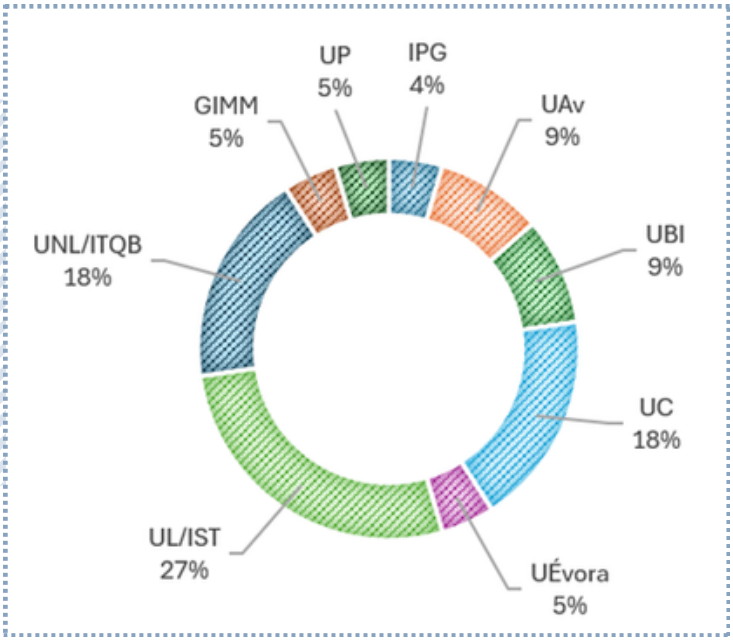
FEBS Education and Training Conference
Kuşadası, Türkiye, 2026.

SPB strengthens Portugal's presence at the 50th FEBS congress

Célia Antunes, University of Évora

The Portuguese Biochemical Society (SPB) was represented at the 50th Congress of the *Federation of European Biochemical Societies* (FEBS), a particularly significant event marking five decades of FEBS Congresses and bringing together the international molecular life sciences community.

Portugal was represented by 29 participants, reflecting the active engagement of the Portuguese scientific community (Figure 1A) in this important international forum for knowledge exchange, discussion of the latest scientific advances, and the establishment of new collaborative networks.



Distribution of SPB researchers participating in the 50th FEBS Congress 2026

Among the highlights of the Portuguese contribution was an invited oral presentation by Carlos Farinha entitled "The CFTR Interactome in Health and Disease: Towards Mechanisms of Cystic Fibrosis", which showcased research carried out in Portugal and highlighted its relevance within the international scientific community.

SPB poster presented at the congress

SPB's presence at FEBS was further strengthened by the increased Portuguese representation within the Federation's scientific structures, with the election of two new members to FEBS committees: Margarida Fardilha (University of Aveiro) and Cecília Arraiano (NOVA University Lisbon), appointed to the Education & Training Committee and the Advanced Courses Committee, respectively.

These appointments recognize the active participation and valuable contributions of the Portuguese biochemical community to the FEBS mission, while also enhancing opportunities for SPB involvement in European initiatives in the fields of education, advanced training, and scientific development.

As part of the celebrations marking the 50th FEBS Congress, SPB also presented a poster highlighting its long-standing collaboration with FEBS through the organization of the 27th and 46th FEBS Congresses, held in Lisbon in 2001 and 2022, respectively (Figure 1B).

This contribution formed part of the commemoration of FEBS's history and showcased the role and continued presence of the Portuguese Biochemistry and Molecular Life Sciences community throughout this journey.

SPB strengthens Portugal's presence at the 50th FEBS congress

Participation in this congress further reinforces SPB's commitment to the internationalization of Portuguese science, the promotion of Biochemistry and the Molecular Life Sciences, support for the training and development of the next generation of researchers, and the strengthening of collaboration with the European scientific community.

SPB congratulates all Portuguese participants and, in particular, the colleagues who are now taking on new responsibilities within FEBS committees, and wishes them every success in this new stage of their service to the scientific community.

Calendar & Events

DATE	EVENT	PLACE	LINK
Monthly Online Seminars	**PAGE 2026: Web Seminars of the Portuguese Ageing Research Group**	Monthly Online Seminars	LINK
04 - 06/10	**19th Congress of the International Union of Microbiological Societies (IUMS 2026)**	Lisboa, Portugal	LINK
07 - 09/10	**4th International Conference on Nitric Oxide Therapeutics in Cancer and Other Diseases**	Lisboa, Portugal	LINK
10 - 16/10	**Champalimaud Research Symposium 2026** *Neural and Immune Codes in Cancer*	Lisboa, Portugal	LINK
14 - 16/10	**FEBS-IUBMB-ENABLE Conference**	Kraków, Poland	LINK
22 - 23/10	**XXIII National Congress of Biochemistry**	University of Beira Interior, Covilhã, Portugal	LINK
28 - 30/10	**European Rosetta Con**	Lisboa, Portugal	LINK
09 - 13/11	**EMBO Workshop: Proteostasis shaping health span: from protein folding to failure**	Ericeira, Portugal	LINK