FEBS openbio

Volume 15 Supplement 2 July 2025

POSTERS

Table of Contents

POSTERS – RESEARCH

69 Bioenergetics
72 Biophysics
79 Cell biology
97 Cell fate determination
98 Development
101 Epigenetics
107 Evolution
107 Genome organization and stability
108 Genomics and transcriptomics
113 Marine biology
114 Membrane trafficking, vesicles, organelles
117 Plant biology
124 Redox biochemistry
125 Regulation of gene expression
130 RNA biology
134 Signal transduction
139 Structural biology
157 Chemical biology
162 Glycobiology
163 Protein chemistry
165 Protein homeostasis
167 Proteomics
174 AI in molecular life sciences
176 Computational biology
183 Synthetic biology
184 Systems biology
185 Biotechnology
210 Enzymology
217 Cancer – basic research
274 Cancer diagnostics
283 Cancer therapy
326 Advanced laboratory techniques (in medical biochemistry)
336 Biosensors
341 Drug development
364 Endocrinology
369 Immunology
375 Inflammation
388 Metabolism and metabolomics
402 Microbiology
419 Molecular basis of disease (excluding cancer)
451 Neuroscience
474 Nutrition
480 Oxidative stress, oxidants and antioxidants
499 Toxicology
507 Virology
513 Miscellaneous

POSTERS – EDUCATION

518 Undergraduate teaching/learning
523 Postgraduate teaching/learning
524 Faculty and career development

POSTERS – ADDENDUM

526 Additional Research Posters

Abstracts submitted to the 49th FEBS Congress from 5th to 9th July 2025 and accepted by the Congress Organizing Committee are published in this Supplement of *FEBS Open Bio*. The abstracts are available as two PDF files: Talks (Plenary Lectures, Symposia and Special Sessions) and Posters.

About these abstracts

Abstracts submitted to the Congress are **not peer-reviewed**. In addition, abstracts are generally published as submitted and **are not fully copyedited** prior to publication. We are unable to make **corrections of any kind** to the abstracts once they are published.

Indexing

The *FEBS Open Bio* supplement for the 49th FEBS Congress will be indexed in the Web of Science Core Collection.

How to cite these abstracts

AuthorOne, A., AuthorTwo, B. (2025). Abstract title. *FEBS Open Bio*, 15: Abstract number*. doi:10.1002/2211-5463.70071

* Each poster has been given a unique number beginning with the letter P (or LB for 'late-breaking abstracts'); the next numerical part relates to the topic grouping as listed below.

FEBS Open Bio **15** (Suppl.2) (2025) 67–527 DOI: 10.1002/2211-5463.70071

significant threats to human health. A major challenge in these scenarios is mitigating radiation-induced damage to healthy tissues, driving the need to develop effective radioprotective agents. This study focuses on the design, synthesis, and evaluation of novel piperazine derivatives as potential radioprotective agents to overcome the limitations of current countermeasures. A series of 1-(2-hydroxyethyl)piperazine derivatives was synthesized and their cytotoxicity tested for in various human cell lines. Their radioprotective properties were further assessed in the MOLT-4 lymphoblastic leukemia cell line and peripheral blood mononuclear cells (PBMCs) exposed to gamma radiation. To quantify their ability to reduce DNA damage, the dicentric chromosome assay (DCA) and apoptosis detection were employed. Among the synthesized compounds, compound 6 demonstrated the most potent radioprotective effects *in vitro*, with minimal cytotoxicity. Compound 3 also exhibited notable efficacy, particularly in reducing dicentric chromosomes, suggesting its potential in mitigating DNA damage. Both compounds showed enhanced safety and effectiveness compared to amifostine, highlighting their promise as improved radioprotective agents. Based on the obtained findings, the mentioned compounds served as molecular templates for the synthesis of a new series of substances which show enhanced radioprotective properties. Acknowledgements: This work was supported by grant project NU23-08-00256 of the Ministry of Health.

P-35-034
Unlocking PD-1/PD-L1 axis in non-small cell lung cancer: *in silico* design of tetrahydroquinoline-based small-molecule immune checkpoint inhibitors

B. Işın*[I], M. H. Şehitoğlu*[II], N. Kılınç*[III], M. Z. Kazancıoğlu*[IV], E. A. Kazancıoğlu*[V]
[I]*Çanakkale Onsekiz Mart University, Institute of Health Sciences, Bioinformatics and Systems Biology, Çanakkale, Türkiye,* [II]*Canakkale Onsekiz Mart University, Faculty of Medicine, Department of Medical Biochemistry, Çanakkale, Türkiye,* [III]*Igdir University, Vocational School of Health Services, Igdir, Türkiye,* [IV]*Kilis 7 Aralik University, Faculty of Health Sciences, Kilis, Türkiye,* [V]*Kilis 7 Aralik University, Vocational School of Health Services, Kilis, Türkiye*

The programmed cell death protein-1 (PD-1)/PD-L1 immune checkpoint axis is a critical therapeutic target in non-small cell lung cancer (NSCLC); however, treatment resistance and high costs continue to limit the effectiveness and accessibility of current therapies. Development of small-molecule inhibitors targeting the PD-1/PD-L1 interaction offers a promising approach to enhancing efficacy and accessibility. Tetrahydroquinoline (THQ) derivatives, known for their immunomodulatory and apoptosis-inducing effects, have emerged as compelling candidates for dual checkpoint inhibition due to their broad biological activity and role in modulating key cellular processes, making them an attractive scaffold for anticancer drug development. This study employs a structure-based drug design approach to develop novel THQ-based PD-1/PD-L1 inhibitors. Molecular docking (Schrödinger 2020-3, Glide XP) and MM-GBSA calculations identified five leads with superior ΔGbind values ranging from (PD-L1: -28.04 to -49.82 kcal/mol; PD-1: -3.08 to -46.11 kcal/mol), significantly outperforming BMS-200 (ΔGbind = -2.42 for PD-1; -7.54 for PD-L1). Key interactions involve hydrogen bonding with PD-L1's Lys124 and hydrophobic contacts near Tyr56, alongside analogous interactions with PD-1, reinforcing the dual affinity of THQ derivatives. Guided by these computational insights, chemical synthesis of selected THQ derivatives is currently in progress using optimized synthetic methodologies. To further assess their therapeutic potential, future studies will focus on ADME profiling, molecular dynamics simulations, kinetic and thermodynamic evaluations of PD-1/PD-L1 inhibition, and *in vitro* validation of lead compounds in NSCLC cells (A549). This work aims to advance the development of next-generation small-molecule immune checkpoint inhibitors, bridging the gap between computational drug discovery and translational oncology. *The authors marked with an asterisk equally contributed to the work.

P-35-035
Identification of potent inhibitors of Zika and Dengue viruses proteases from small-molecule compound libraries

I. Dolci[I], L. Cipriano[II], G. Dias Noske[III], A. Schutzer Godoy[II], M. Fairhead[IV], R. Sachetto Fernandes[II], **G. Oliva**[II]
[I]*Institute of Physics of São Carlos, University of São Paulo (IFSC-USP), São Carlos, Brazil,* [II]*Institute of Physics of São Carlos, University of São Paulo, São Carlos, Brazil,* [III]*National Nanotechnology Laboratory (LNNano), National Center for Research in Energy and Materials (CNPEM), Campinas, Brazil,* [IV]*Centre for Medicines Discovery, University of Oxford, Oxford, United Kingdom*

Zika virus (ZIKV) is a flavivirus transmitted by *Aedes* mosquitoes, responsible for outbreaks of zika fever, a disease that can result in severe neurological complications such as congenital Zika syndrome and Guillain-Barré. Despite its re-emerging potential, there are no approved antivirals or vaccines against the virus. The NS2B-NS3 protease (NS2B-NS3^{pro}) plays a critical role in processing the viral polyprotein and is a promising drug target. Here, we conducted a high-throughput screening (HTS) of 12 000 compounds from three small-molecule libraries (ReFRAME, HGL1, and HGL2) against the recombinant ZIKV NS2B-NS3^{pro}. Eleven compounds inhibited the enzyme by > 80% at 10 μM and six exhibited a concentration-dependent effect with IC_{50} values in the micro to submicromolar range. Those compounds were further evaluated against the viral replicon and three showed antiviral activities resulting in selectivity indices up to 5.2. Additionally, differential scanning fluorimetry (DSF) revealed varied thermal stability effects, supporting direct protein-ligand interactions. To assess cross-inhibition potential, the most potent ZIKV protease inhibitors were tested against the NS2B-NS3^{pro} from Dengue virus (DENV) serotypes 1 to 4. Four compounds inhibited DENV1 protease, while Compound 1 was active against all tested serotypes. Our findings provide valuable insights for antiviral drug discovery targeting flavivirus proteases, with Compound 1 emerging as a promising lead for further optimization.