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Abstracts submitted to the 48th FEBS Congress from 29th June to 3rd July 2024 and accepted by the Congress Scientific Committee are published in this Supplement of *FEBS Open Bio*. Late-breaking abstracts are not included in this supplement. The abstracts are available as three PDF files: Talks (Plenary Lectures, Symposia and Speed Talks), Posters and Posters Annex.

About these abstracts

Abstracts submitted to the Congress are **not peer-reviewed**.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

Indexing

Abstracts published in the *FEBS Open Bio* Supplement for the 48th FEBS Congress will be included individually in the Conference Proceedings Citation Index published by Web of Science.

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* Each poster has been given a unique number beginning with the letter P; the next part relates to the session in which the poster will be presented.

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outstanding tendency to metastatic dissemination (1). In this scenario, the elucidation of cellular mechanisms featuring OS tumorigenesis, and the identification of new molecular therapeutic targets, become crucial. In this study, we focused on paraoxonase-2 (PON2), a ubiquitously expressed intracellular protein, mainly localized in the endoplasmic reticulum (ER) and associated with the inner mitochondrial membrane. PON2 functions as an antioxidant enzyme, by counteracting reactive oxygen species release, thus displaying an antiapoptotic effect and preventing the formation of atherosclerotic lesions. Enzyme overexpression was reported in a wide variety of solid tumors, promoting tumor cell aggressiveness (2). Preliminary immunohistochemical analyses were carried out to explore PON2 expression in tumor and normal bone tissue specimens, obtained from OS patients and healthy subjects, respectively. In order to investigate enzyme contribution to cancer cell phenotype, shRNA-mediated gene silencing was used to achieve PON2 knockdown in OS cell lines and the impact of cell proliferation, migration and chemosensitivity was further assessed. Obtained results showed a significant PON2 overexpression in OS with respect to control samples. Data collected from cell-based assays demonstrated that enzyme downregulation was associated with a significant decrease of proliferative capacity and migration ability, as well as with an enhancement of sensitivity to chemotherapeutic treatment. The sum of this evidence seems to suggest a promising role for PON2 as molecular biomarker and therapeutic target for OS. References 1. Kansara M et al. (2014) Nat Rev Cancer 14, 722–35. 2. Campagna R et al. (2024) Biomolecules 14, 208.

P-27-039
Discovery of benzimidazole-indazole based inhibitors targeting mutant FLT3 kinases for the treatment of acute myeloid leukemia

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The FLT3 gene encodes a receptor tyrosine kinase expressed in hematopoietic stem cells. Mutations in FLT3, which are observed in 30% of acute myeloid leukemia (AML) cases, result in aberrant activation of the receptor's kinase, leading to the proliferation of immature myeloblast cells. Although small molecule inhibitors targeting FLT3 kinase have been approved, new inhibitors are still needed due to side effects and drug resistance caused by kinase domain mutations such as D835Y and F691L. This study presents novel benzimidazole-indazole based inhibitors that are designed to target mutant FLT3 kinases by optimizing various chemical moieties around the core structure. Compound 22f showed potent inhibition of FLT3 and FLT3/D835Y, with IC_{50} values of 0.941 and 0.199 nM, respectively. It also demonstrated significant antiproliferative activity against an AML cell line (MV4-11 cells) with a GI_{50} of 0.26 nM. Notably, 22f exhibited single-digit nanomolar GI_{50} values against mutant FLT kinase-expressing Ba/F3 cell lines, including FLT-D835Y (GI_{50} = 0.29 nM) and FLT3-F691L (GI_{50} = 2.87 nM). Molecular docking studies showed that the compound is a type 1 inhibitor in the homology model of the active conformation of FLT3 kinase.

P-27-040
Investigating intra- and extra-cellular functions of Transglutaminase 2 by use of inhibitors with different localization in breast cancer cells

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Transglutaminase 2 is involved in breast cancer with several processes, such as epithelial-mesenchymal transition, aggressiveness, and metastatization. This protein displays intracellular and extracellular roles, which we have investigated using membrane-permeable and impermeable inhibitors. The alteration of transcriptome following the treatment of two triple-negative breast cancer cell lines allows us to determine the modulated genes, pathways, and networks. Integrin signaling and p53 were commonly affected by each inhibitor, while other pathways were specific. AA9, entering the cell, induced apoptosis in MDA-MB-436, affecting cadherin, Wnt, gastrin, and cholecystokinin receptors (CCKR) signaling, with RHOB and GNG2 as relevant players, while it decreased glycolytic enzymes by impact on the Warburg effect. In MDA-MB-231 cells, AA9 significantly modulated genes belonging to HIF-mediated hypoxia, AKT, and mTOR pathways. These effects suggest an anti-tumor efficacy exhibited by inhibiting intracellular TG2 functions. Generally, these effects suggest an anti-tumor efficacy exhibited by inhibiting intracellular TG2 functions. In contrast, NCEG2 increased the expression of ATP synthase and DNA replication-related proteins, indicating that inhibition of extracellular functions could encourage cell division as pro-cell replication action. This study underlines opposite effects following the treatment with inhibitors having different cell localization that will need to be considered for the applications of anti-tumor strategies.

P-27-041
FTIRM analyses as a tool to explore cisplatin sensitivity of oral squamous cell carcinoma cell lines after PON2 knockdown

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Oral squamous cell carcinoma (OSCC) is the most frequent and aggressive variant of head and neck cancer. The management of this malignancy is complicated by the chemoresistance, which can be primary or can quickly arise during the chemotherapeutic treatment, leading to a 5-year survival rate lower than 50%. For these reasons, the identification of novel biomarkers that could

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