



AICC NEWSLETTER

ITALIAN ASSOCIATION OF CELL CULTURES



Latest AICC News

☀️ Upcoming Events



More on the **35° AICC International Meeting** that will be held in **L'Aquila** on **4-6 December 2023!!!**

REGISTRATION IS NOW OPEN and **Preliminary Programme** is available online.

Registration and Abstract Submission Deadline: 15/10/2023

Confirmed invited **SPEAKERS** are:

- ☀️ **Antonio IAVARONE**, University of Miami
- ☀️ **Giovanni BLANDINO**, Regina Elena National Institute for Cancer Treatment and Research, Rome
- ☀️ **Ugo CAVALLARO**, European Institute of Oncology, Milan
- ☀️ **Gerry MELINO**, "Tor Vergata" University of Rome
- ☀️ **Lucia ALTUCCI**, University of Campania "L. Vanvitelli", Naples
- ☀️ **Giorgio STASSI**, University of Palermo
- ☀️ **Lucia DI MARCOTULLIO**, La Sapienza University of Rome
- ☀️ **Annamaria TETI**, University of L'Aquila
- ☀️ **Anguraj SADANANDAM**, The Institute of Cancer Research, London
- ☀️ **Giuseppe SCIUMÈ**, La Sapienza University of Rome
- ☀️ **Giulia BIFFI**, University of Cambridge
- ☀️ **Gabriele CRUCIANI**, University of Perugia
- ☀️ **Marcello ALLEGRETTI**, Dompé farmaceutici S.p.A.
- ☀️ **Guido FRANZOSO**, Imperial College London



Funding Opportunities

✓ **Fondazione Cariplo & Fondazione CDP**

Deadline: 26/07/2023

✓ **National Institutes of Health (I)**

Deadline: 01/09/2023

✓ **National Institutes of Health (II)**

Deadline: 02/09/2023

✓ **Fondazione Umberto Veronesi**

Post-Doc

Deadline: 14/09/2023



Save the Date!



35th AICC
INTERNATIONAL
MEETING

TRANSLATIONAL AND PRECISION MEDICINE:

FROM PATIENT TO CELL AND BACK



UNIVERSITY
OF L'AQUILA

**4-6
DECEMBER
2023**



"LUIGI ZORDAN" CONGRESS CENTRE - ST. BASILIO MONASTERY, L'AQUILA



Funding Opportunities

✓ European Commission

Deadline: 19/09/2023

✓ Telethon Grant

Deadline: 31/10/2023

✓ ERC-2023-StG

Deadline: 25/10/2023



News from our Partner Community

☼ Società Chimica Italiana - Divisione di Spettrometria di Massa (DMS-SCI)

The 3th INTERNATIONAL PROTEOMICS AND METABOLOMICS CONFERENCE will take place at the University of Piemonte Orientale, Novara, Italy on 2nd-3rd October 2023.

The objective of the conference is to present the recent advances in the proteomics and metabolomics analyses from aging and aging related-diseases, to therapeutic targets detection and clinical translation.

Confirmed invited speakers:

- **Karl Mechtler**, Vienna Biocenter
- **Guido Bommer**, Université Catholique de Louvain
- **Paola Picotti**, ETH Zurich (on-line)

AICC will grant its **patronage** for the event.



Open Positions

1. 18-months Postgraduate/Postdoc position

Institute of Biophysics of
CNR - Genoa

Supervisor: Michael Pusch

Start date: October 2023

Properties and physiological role of the putative neuronal glucose transporter SLC45A1

2. 2-years Postdoc position

Department of Oncology
University of Turin

Supervisor: Chiara Riganti

Start date: February/March 2024

Studying the metabolome of non-small cell lung cancer to identify biomarkers predictive of response to chemo-immuno-therapy

3. PhD Programme in Pharmacological Biomolecular Sciences, Experimental and Clinical

University of Milan

Supervisor: Paola Corsetto

Application Deadline:
26/07/2023

Study of circulating lipid markers to engineer precision diagnostics and targeted pharmacological approaches in cancer



Featured Articles

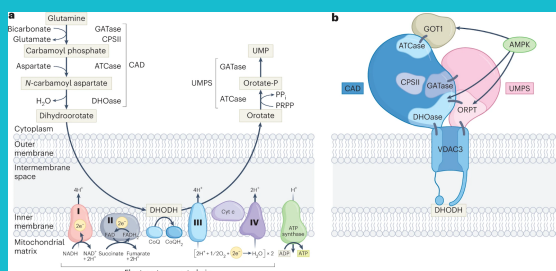
1

De novo pyrimidine biosynthetic complexes support cancer cell proliferation and ferroptosis defence.

Yang C, Zhao Y, Wang L, et al.

Nature Cell Biology 2023

De novo pyrimidine synthesis is a vital process for cell proliferation. It involves multiple enzymes, each one using the product from another as a substrate for the next. However, how these enzymes coordinate to ensure the proper chemical reactions occur remains a mystery. Interestingly, in this study recently published in Nature Cell Biology, Yang and co-workers demonstrated that the key enzymes of de novo pyrimidine nucleotide synthesis and ferroptosis form a dynamic complex called pyrimidinosome, which is controlled by AMPK. Cancer cells low in AMPK expression rely on the pyrimidinosome, suggesting that co-inhibition of AMPK and the pyrimidinosome would potentially represent a novel therapeutic strategy to counteract cancer.



Open Positions

4. National PhD Programme in Precision Medicine

University of Palermo*

*Several Courses available in different locations

Application Deadline:
23/08/2023

5. PhD Programme

University of Palermo*

*Several Courses available

Application Deadline:
17/08/2023

6. PhD Programme in Experimental Medicine

University of L'Aquila

*Several Courses available

Application Deadline:
24/08/2023

Let us know if you are recruiting and we will spread the word!!!

Drop us an email by clicking on the icon below

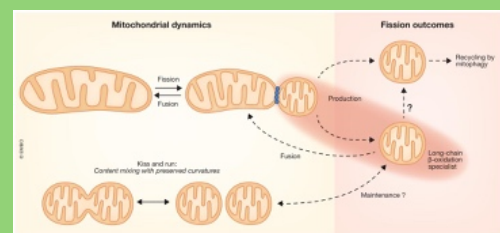


2

Mitochondrial morphology controls fatty acid utilization by changing CPT1 sensitivity to malonyl-CoA.

Ngo J, Choi DW, Stanley IA. *et al.*

The EMBO Journal 2023



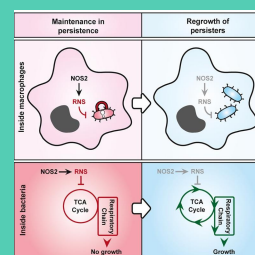
The relationship between mitochondrial shape and substrate-specific metabolism has been long debated. Here, using cellular models representing a wide variety of mitochondrial shapes, Ngo and colleagues show a strong linear correlation between mitochondrial fragmentation and increased fatty acid oxidation rates. In particular, using respirometry and metabolic tracing approaches, the authors evidenced that while forced mitochondrial elongation determines an impairment of FAO, induction of mitochondrial fragmentation promotes an increase of FAO, so demonstrating that mitochondrial architecture plays a key role in regulating metabolism.

3

Decline in nitrosative stress drives antibiotic persister regrowth during infection.

Ronneau S, Michaux C and Helaine S.

Cell Host & Microbe 2023



As known, certain bacteria – thanks to their slow growth – can survive antibiotic treatment also in the absence of specific mechanisms of resistance. A typical example is represented by *Salmonella enterica* serovar Typhimurium that, actively growing inside macrophages, are sensitive to the antibiotic cefotaxime. Despite this, some bacteria subgroups have been found to survive antibiotic treatment, thus leading to the onset of chronic infections. In this recent work, Ronneau and colleagues found that host reactive nitrogen species (RNS) derived from the nitric oxide produced by nitrogen oxide synthase 2 (Nos2) promoted the *Salmonella* persister state inside macrophages. RNS does not induce a growth-arrested persister state but maintains it by altering the bacterial tricarboxylic acid (TCA) cycle. Treating infected macrophages with metabolites that stimulate respiration independently of the TCA cycle caused intracellular bacteria to resume growth and sensitized them to cefotaxime. Nos2 and NO increase in macrophages after infection with *Salmonella* and induce nitrosative stress in the bacteria. As host NO production abates, some of the bacteria resume growth and become sensitive to cefotaxime, but others remain in the growth-arrested state. Treatment of infected macrophages with a pharmacological inhibitor of Nos2 forces the intracellular persisters to resume growth. In this way it is possible to enhance the antibiotic sensitivity of bacteria, dramatically reducing infection recurrence in patients.

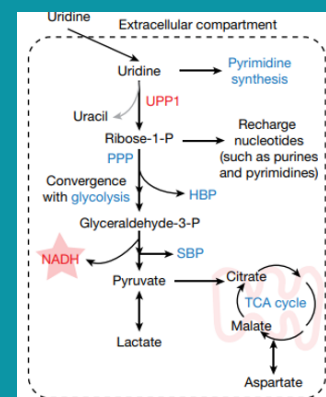
4

Uridine-derived ribose fuels glucose-restricted pancreatic cancer

Nwosu, Z.C., Ward, M.H., Sajjakulnukit, P. *et al.*

Nature 2023

This recent study published in Nature identified uridine-derived ribose as the fuel for pancreatic ductal adenocarcinoma (PDA) in glucose-deprived conditions. The researchers applied a high-throughput in vitro nutrient screening to 21 pancreatic cell lines to analyze the ability of the cancer cells to metabolize over 175 nutrients under nutrient-restricted conditions and assessed the correlation between metabolite utilization patterns and the expression of associated genes. The study illustrates the complexity of nutrient availability in the tumor microenvironment and suggests a model in which cells inside and outside PDA tumors fuel cancer metabolism with uridine. Moreover, these findings suggest a novel metabolic axis for PDA therapy.



5

Ferroptosis surveillance independent of GPX4 and differentially regulated by sex hormones

Liang, D., Feng, Y., Zandkarimi, F., *et al.*

Cell 2023

Ferroptosis is a cell death process driven by iron-dependent phospholipid peroxidation that has been implicated in various diseases. Mounting evidence suggests ferroptosis acts as an innate tumor suppressive mechanism mediating the anticancer activity of multiple tumor suppressors. In their recent publication, Liang D. and colleagues utilized a whole-genome CRISPR activation screen and identified membrane bound O-acyltransferase domain containing 2 (MBOAT2), a lyso-PL acyltransferase (LPLAT), as a ferroptosis-suppressing gene. Interestingly MBOAT2 is directly upregulated by androgen receptor (AR) and estrogen receptor (ER) suggesting that sex hormone signaling restrains cancer cells from ferroptosis through MBOAT1/2-mediated phospholipid remodelling. These regulatory events represent an interesting new avenue that can be explored for the treatment of cancers.

