

Cover page: The Synthetic Lethal Rosette

Aberrant mitotic phenotype found in BRCA1-deficient cells treated with the PLK1 inhibitor Volasertib. Cells become giant and multinucleated and acquire a flower shape, with nuclei arranging in a circular disposition around a cluster of centrosomes. Blue (DAPI: nuclei), Green (FITC-phalloidin: actin cytoskeleton), Red (γ -Tubulin: centrosomes).

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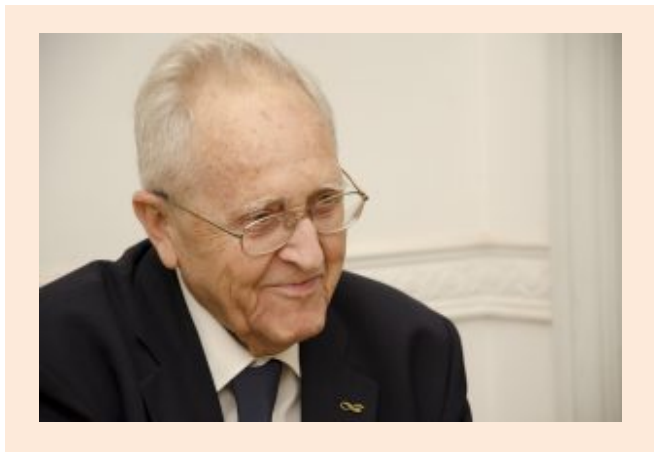
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IN MEMORIAM
HORACIO G. PONTIS
(1928–2019)



Horacio Guillermo Pontis, born in Mendoza (1928), graduated in chemistry and obtained the Ph.D. (1953; Dir.: V. Deulofeu) from the University of Buenos Aires. After working for three years with Dr Luis F. Leloir—where he approached to carbohydrate metabolism—he stayed successively at King College (UK), Durham University (UK) and finally at Karolinska Institutet and University of Stockholm—where his attention turned to enzymology studies. After returning to Leloir’s lab (1960), he embarked on plant biochemistry studies. In his search for clues about fructans, Dr. Pontis’ lab synthesized not only UDP-fructose but also fructose-2-phosphate, which two decades later cleared the way for the chemical synthesis of fructose-2,6-bisphosphate (a key glycolysis modulator).

From 1968 to 1977, he was the director of Dept. of Biology – Fundación Bariloche. In Nov. 1971, Bariloche hosted the SAIB Annual Meeting, being elected Dr. Pontis the President of SAIB (1972).

This reunion was followed by the Symposium “*Biochemistry of the glycosidic linkage*” with the presence of four Nobel Prizes (C. Cori

(1947), G. Cori (1947), F. Lynen (1964), L. F. Leloir (1970)). However, his “*mi mejor experimento y experiencia de formación*” came to a halt-in when the Bariloche lab was closed (1977).

In 1979, Dr. Pontis moved to Mar del Plata where over time his outstanding capacity for innovation launched Instituto de Investigaciones Biológicas (IIB) – U. N. Mar del Plata, Fundación de Investigaciones Científicas (FIBA) and Centro de Investigaciones Biológicas (CIB).

In any site, Dr. Pontis maintained active research groups that trained graduate and post-graduate students generating a steady flow of important contributions to plant biochemistry. The research international community acknowledged these accomplishments, such as American Society of Plant Biologists that named him Correspondent Member. In his scientific activities, Dr. Pontis has been member of the National Research Council of Argentina (1961; CONICET), and Biochemistry Professor –at the UBA and at Universidad Nacional de Mar del Plata. The former and the later institutions recognized his academic performance designating him Emeritus Investigator and Emeritus Professor, respectively.

Dr. Pontis’ story rose from limited beginnings—in Deulofeu’s and Leloir’s labs—to international scientific prestige. In this context, the challenge to overcome adversity during shameful periods in Argentine history honors not only his willingness but also his enthusiasm.

Ricardo Wolosiuk

Schedule	Tuesday November 5		Wednesday November 6	Thursday November 7	Friday November 8
8:30-9:00	WORKSHOPS Workshop Accreditation				
9:00-11:00	Biochemistry Education Workshop	1 st Workshop On Drug Discovery	Oral Communications Room Jacaranda PL-Co1, PL-Co2, PL-Co4 to PL-Co6, PL-Co9, PL-C12, ST-02, BT-Co2 Room Los Ceibos CB-Co1 to CB-Co4, CB-Co7 to CB-C10, ST-Co1	Conferences Room Lapacho <i>Robert Gennis</i> <i>Francisco Barrantes</i>	Oral Communications Room Jacaranda PL-Co3, PL-Co7, PL-Co8, PL-C10, PL-C11, PL-C13, PL-C14, PL-C15 Room Los Ceibos MI-Co1 a MI-Co6, BT-Co1, CB-Co6 Room Lapacho LI-Co1 to LI-Co5, ST-Co3, CB-C11, CB-Co5
11:00-11:30			COFFEE-BREAK		
11:30-12:30			Plenary lecture <i>Bruno Amati</i> Room Lapacho	IUBMB Jubilee Lecture <i>Philip D. Stahl</i> Room Lapacho	“Hector Torres” Plenary Lecture <i>Alejandro Colman Lerner</i> Room Lapacho
12:30-14:30			LUNCH TIME		
14:30-16:30			Symposia <i>Lipids</i> Room Jacaranda <i>Plants</i> Room Los Ceibos <i>Signal Transduction</i> Room Lapacho	Symposia <i>Cell Biology</i> Room Jacaranda <i>RNA</i> Room Los Ceibos	Symposia <i>Microbiology</i> Room Los Ceibos <i>PABMB</i> <i>Young Investigators</i> Room Jacaramda
16:30-17:00			COFFEE -BREAK		
16:30-18:30			POSTERS BT-Po1 to BT-Po6 CB-Po1 to CB-P15 MI-Po1 to MI-P18 PL-Po1 to PL-P15 ST-Po1 to ST-P13	POSTERS BT-Po7 to BT-P12 CB-P16 to CB-P31 EN-Po1 to EN-P11 MI-P19 to MI-P37 PL-P16 to PL-P32	POSTERS BT-P13 to BT-P19 CB-P32 to CB-P47 LI-Po1 to LI-P15 MI-P38 to MI-P49 PL-P33 to PL-P48
			Opening Ceremony Room Lapacho		
			<i>In memoriam of Horacio Pontis</i> Room Lapacho		
18:30-19:30			“Alberto Sols” Plenary Lecture Room Lapacho <i>Encarnación Martínez Salas</i>	EMBO Keynote Lecture <i>F. Gisou van der Goot</i> Room Lapacho	“Ranwel Caputto” Plenary Lecture <i>Maria Elena Alvarez</i> Room Lapacho
			Cocktail 20:00 hs	SAIB Assembly 19:45 hs	Dinner 20:00 hs

and salt stresses. Altogether, these data suggest that ERF-SR regulates the expression of genes involved in ABA- and JA- signal-transduction pathways.

SIGNAL TRANSDUCTION

ST-C01

ROLE OF THE P53 TARGET ICMT IN METASTASIS: POST-PRENYLATION PROCESSING AT THE CENTER OF THERAPEUTIC STRATEGIES IN CANCER

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The mevalonate pathway catalyzes the *de novo* synthesis of cholesterol, but also provides isoprenoids that may be used for protein modification through a complex process known as prenylation. Some proteins involved in oncogenic processes, such as Ras and Rho GTPases are among the targets of this modification. Pioneering studies with statins have suggested that mevalonate pathway alteration cooperates with tumor aggressiveness. More recently, other evidence has confirmed that alteration of this pathway may promote aggressive phenotypes and pointed out at enhanced protein prenylation as a possible mechanism underlying this effect. ICMT plays a central role in this posttranslational modification process by catalyzing the last step, carboxymethylation of the prenylated C-terminus in target proteins. We have recently unveiled a link between post-prenylation processing and the p53 pathway by showing that *ICMT* expression is repressed by wt p53, but enhanced by cancer-associated p53 point mutants. Moreover, our analysis of Breast and Lung cancer databases showed a negative correlation between *ICMT* expression and wt p53 status. Moreover, we found a significantly decreased metastasis-free survival frequency in patients with high *ICMT* expression. Basing on these results, we wondered if alteration of ICMT levels enhances metastasis development. To answer this question, we studied the effect of ICMT overexpression on metastasis *in vivo*, using Triple Negative Breast Cancer cells in an immunocompetent mouse model. We extended our previous analysis on breast cancer patients and we found that p53 status affects the impact of ICMT overexpression on clinical outcomes. Besides, our studies on the regulation of *ICMT* expression showed that other p53 family members affect its transcription. Our results suggest that ICMT levels are affected by alterations in the functional equilibrium between different members of this family during tumor progression. To explore the potential of pharmacological manipulation of ICMT function, we analyzed the impact of the ICMT inhibitor Cysmethynil to affect tumor-associated phenotypes. We found that ICMT inhibition affects clonogenic potential, as well as phenotypes associated with metastatic cells, such as migration and invasion *in vitro*. In an effort to develop novel ICMT inhibitors and inspired on Salirasib (S-trans-trans-farnesylthiosalicylic acid) we synthesized novel thiosalicylic acid derivatives. As a preliminary characterization, we analyzed the antiproliferative activity of our compounds *in vitro* on MDA-MB-231 cells. Our results suggest that ICMT overexpression affects tumor progression and that molecules interfering with the function of prenylated proteins are potentially useful in therapeutic strategies.

ST-C02

NITRIC OXIDE AND AUXIN REGULATE ROOT MERISTEM DURING GRAVITROPISM IN *ARABIDOPSIS THALIANA*

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Nitric oxide (NO) is a gaseous redox-active molecule with a role in different physiological auxin-mediated processes including gravitropism. Roots bend in response to gravity by the formation of an asymmetric distribution auxin pattern between the upper and the lower sides of elongation and meristematic zones of the root. However, the mechanisms by which auxin and NO interplay during the gravitropic response are not still fully understood. In this work, we focus on deciphering the spatio-temporal pattern of auxin and the functional contribution of NO in the meristematic cells during early events of gravitropism in *Arabidopsis* roots. In *Arabidopsis thaliana*, the meristematic root zone comprises all the cells that undergo mitotic divisions and stretches longitudinally up to 350 μm from the quiescent center (QC). By using the auxin sensor DII-VENUS we demonstrated that within the first 30 min of a 90° gravity stimulus, the hormone was distributed asymmetrically between the upper and lower sides in lateral root cap, epidermal and cortical cells extending up to 120 μm from the QC in the meristematic region of the root. In addition, we demonstrated that NO is accumulated asymmetrically between the lower and upper sides of the root meristem. Next, we measured the length of individual epidermal cells along the meristematic zone. Scavenging of endogenous NO affects the characteristic epidermal cell length observed during the gravitropic response. Therefore, we hypothesize that the disturbance of the interaction between auxin and NO signals could affect the meristematic cell size pattern which leads to an agravitropic response in *Arabidopsis* roots. Since cyclins play a vital role in controlling cell cycle progress, one of our current challenges is to investigate how auxin- and NO-mediated regulation affects the dynamic and functionality of root meristem during gravitropism. *Supported by UNMdP, CONICET, ANPCyT.*

ST-C03