ERC-StG
ERC-PoC
An applicant’s perspective

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INTRO INTO RESEARCH FOCUS

HOW THE ERC CAME INTO MY LIFE

MY ERC STG STORY

MY ERC POC STORY

WHAT I WOULD DO THE SAME ALL OVER AGAIN

THINGS I WOULD DO DIFFERENTLY
Malignant Pleural Effusion (MPE)
Adverse Event Vs Ca Hallmark

Hanahan D, Weinberg RA. Cell 2011
Stathopoulos GT, Kalomenidis I. Am J Respir Crit Care Med 2012
A paradigm for tumor-host interactions?

Antunes G et al. Thorax 2003
Stathopoulos GT, Kalomenidis I. Am J Respir Crit Care Med 2012


MPE

A molecular culprit of KRAS-driven paracrine signaling?

Ji H et al. Oncogene 2006;25:2105–12
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Prof. GK Kostopoulos MD PhD
Chair, Dpt. Of Physiology, Med, UoP
Prof. TS Blackwell
MD
Chair, Dpt. Of APCCM, Med, VUMC

Figure 1. Mutant Kras is linked with the ability of tumor cells to form a MPE. (A) MPE volume (B) photographs of MPEs taken via the diaphragm, and (C) survival of C37BL/6 mice with MPEs induced by various syngeneic tumor cells. Lung (LLC) and colon (MC38) adenocarcinomas cause MPE formation, in contrast to AE17 pleural mesothelioma and B16 skin melanoma cells. MPE-competent tumor cells display Kras mutations and constitutive Ras signaling (D), which is linked with constitutive alternative, but not canonical NF-kB activation (E). *P < .001 compared with other cell lines.

Figure 2. Kras promotes alternative NF-kB activation and MPE formation in mice. (A) Compared with a control plasmid (pCMV), forced transduction of a wild-type (wt) Kras and GFP-encoding bicistronic plasmid (pCMV.Kras.GFP) in B16 skin melanoma cells causes enhanced Ras activation in unstimulated conditions, increased alternative NF-kB activation, and renders these MPE defective cells capable of MPE formation. (B) Compared with a control plasmid encoding random shRNA (pU6), forced transduction of a bicistronic plasmid encoding specific shRNA targeting Kras mRNA at position 727 and RFP (pU6.Ksh727.RFP) in MC38 cells limits Ras pathway activation in unstimulated conditions, inhibits constitutive alternative NF-kB activation, and renders these MPE-competent tumor cells incapable of MPE formation. * P < .05 and P < .001 compared with other cell lines.
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http://www.ekt.gr/
NCP
(CP)

https://erc.europa.eu/
Guide for applicants
Guide for reviewers
Status At ERC Application

EXPATRIATE (US), DEPENDENT RESEARCH

NO NATURE, SCIENCE, CELL PAPER, BUT SEVERAL 1ST AUTHOR MID-RANGE (IF 5-15) PUBLICATIONS INCLUDING JNCI, PNAS, AJRCCM, CANCER RES, ETC.

GENERATED NEW/EXPANDED EXISTING FIELD

CLINICAL RELEVANCE

ERS MAURIZIO VIGNOLA AWARD

UPCOMING REPATRIATION ASST. PROF. POSITION (NOT DEPENDENT ON ERC FUNDING, NO INSTITUTIONAL START-UP)

NO OTHER WAY TO GO
What happened...

JUL 2009: HEARD OF ERC
NOV 2009: APPLIED FOR StG
FEB 2010: 1ST ROUND
MAY 2010: INTERVIEW
OCT 2010: FINAL RESULTS
DEC 2010: GA
FEB 2011: PREFINANCING
APR 2011: START DATE
OCT 2011: EXPERIMENTS
Applying for and (even more so) getting an ERC StG is like entering an arena with lions

Pressure to get things going
Pressure to publish high impact and to garner major accomplishments
Pressure to consolidate the group
Pressure for continued funding
Pressure escalates as approaching the CoG schemes
Who u r competing against...

ERC-CoG results 2014
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Why the heck would you apply???
(for a PoC if you have a StG or CoG or AdG)

Top-up money (so not true, need to design and steer separate project)

Commercialize an idea (so not true, just seed money)

Fund an idea to make it happen (the truth: to start making it happen)

Because it’s a piece of cake compared to the big ERC grants (so not true, technical writing as compared with scientific writing, assessment criteria)

Because it requires less writing (so not true, 10 pages fro 150 K as compared with 20 pages for several M)

Because it has double or triple the success rate as the big ones (so not true, the denominator is ERC grantees)

The speaker got his StG at once, his PoC after 3 failures
Why the heck would you not apply???
(for a PoC)

Ha, ha, because I already got one (so not true, can apply for up to two projects as long as they do not overlap in time)

Cause by ERC ran out last year (so not true, can apply up to two years post-ERC)

Cause I do not like doing business, I am a basic scientist (the truth, it is a worldwide trend: science-application-money)

Because I already have enough money with my ERC grant (so not true, there is no better time to apply for additional grants than when you’re ERC-funded)

Because I only like working alone (so true, best partner up with liaison before applying)

Because I do not understand the managerial jargon in the call (so true, you need a liaison to write it)
2013-14: 3 REJECTS FOR A COMPLEX IDEA (1 WITH LIAISON)

DEC 2014: IDENTIFIED NEW LIAISON

FEB 2015: APPLIED FOR NEW PROJECT (IDIOT PROOF)

MAY 2015: GOT IT (150 K)

NOV 2015: PREFINANCING

NOV 2015: START

DEC 2015: NEGOTIATE WITH LIAISON

JAN 2016: FULL DEPLOYMENT
What is it about (idea 2p)

Syngeneic lung tumor models: LLC

Naturally induced lung tumors

Cell lines

Floxed cell lines

Tools for the CR community
Expected impact (2p)

Economic and/or societal benefits

Commercialisation process and/or any other exploitation process

Proposed plans for:

- Competitive analysis
- Testing, technical reports
- IPR position and strategy
- Industry/sector contacts
A Marazioti
M Spella
T Agalioti
I Lilis
I Giopanou
NI Kanellakis
N Spyropoulou
M Papageorgopoulou
G Ntalliarda
G Giotopoulou
A Krontira
V Armenis
D Kati

M Vreka
LV Klotz
K Arendt