Molecular mechanisms of tumor suppressor functions of HECT-type E3 ligase Smurf2: roles in chromatin organization, dynamics, gene expression and DNA damage response and repair

Fact Sheet

Project Information

SMURF2 IN CANCER

Grant agreement ID: 612816

Project closed

Start date
1 August 2013

End date
31 July 2017

Funded under
Specific programme "People" implementing the Seventh Framework Programme of the European Community for research, technological development and demonstration activities (2007 to 2013)

Total cost
€ 100 000,00

EU contribution
€ 100 000,00

Coordinated by
BAR ILAN UNIVERSITY
Israel

Objective

Cancer is a multifaceted disease in which dysregulated gene expression and other aberrant activities are crucial for neoplastic initiation, progression and tumor cell survival upon the treatment with anticancer modalities.
One of the major mechanisms that regulate these processes is protein ubiquitination. This modification can impose diverse effects on gene products–proteins, ranging from proteolysis to modulation of protein structure, localization and function. Central components of ubiquitination are the E3 ubiquitin ligases (E3s). Among the different types of E3s, HECT-type E3 ligases are largely unexplored. Recently, the anticancer roles of these enzymes have become highly relevant as we discovered prominent tumor-suppressor functions of one member of this family – Smurf2 (Blank M et al., Nature Med 2012). However, many gaps still remain in our understanding of Smurf2 biological functions in cancer, and potentially important therapeutic targets are missed.
The main objective of our research is to delineate the spectrum of molecular mechanisms operating in the cell under the jurisdiction of Smurf2, focusing primarily on those involved in the regulation of chromatin organization and dynamics, gene expression, DNA damage repair and genomic integrity maintenance. They are all interconnected and often compromised in cancer pathways. Using multidisciplinary approaches spanning these areas, we are determined to shed light on roles that HECT-type E3 ligases in general and Smurf2 in particular play in tumor biology and, more importantly, the sensitivity of cancer cells to DNA damage-inducing therapies. Together with surgery, these comprise current first choice in cancer treatment. We believe that the knowledge generated by this study has the potential to provide novel targets in cancer treatment and the design of new, more efficient, therapeutics to combat this devastating disease.

Fields of science (EuroSciVoc)

medical and health sciences > clinical medicine > **surgery**

natural sciences > biological sciences > genetics > **DNA**

medical and health sciences > clinical medicine > **oncology**

natural sciences > biological sciences > biochemistry > biomolecules > proteins > **enzymes**

Programme(s)

FP7-PEOPLE - Specific programme "People" implementing the Seventh Framework Programme of the European Community for research, technological development and demonstration activities (2007 to 2013)

Topic(s)

FP7-PEOPLE-2013-CIG - Marie-Curie Action: "Career Integration Grants"

Call for proposal

FP7-PEOPLE-2013-CIG
See other projects for this call

Funding Scheme

MC-CIG - Support for training and career development of researcher (CIG)

Coordinator

BAR ILAN UNIVERSITY

EU contribution

€ 100 000,00

Total cost

No data

Address

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Activity type

Higher or Secondary Education Establishments

Links

Contact the organisation Website
Participation in EU R&I programmes
HORIZON collaboration network

Last update: 16 December 2021

Permalink: https://cordis.europa.eu/project/id/612816