BIOGRAPHICAL SKETCH
NAME: RAFFAELLA DI MICCO

POSITION TITLE: GROUP LEADER AT THE SAN RAFFAELE TELETHON INSTITUTE FOR GENE THERAPY, SAN RAFFAELE HOSPITAL
ASSOCIATE PROFESSOR OF PATHOLOGY AT THE SCUOLA UNIVERSITARIA SUPERIORE, IUSS, PAVIA

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Naples "Federico II"	M.Sc	July 2003	Cancer Metastasis
European School of Molecular Medicine (SEMM)	PhD	2004-2008	DNA damage checkpoint activation during oncogene-induced senescence
IFOM Foundation, Milan, Italy (Fabrizio d'Adda di Fagagna's lab)	Postdoc	2008-2010	Interplay between chromatin and DNA damage signaling in senescence and cancer
New York University, New York, USA (Eva Hernando's lab)	Postdoc	2010-2015	Epigenetics and Transcription in stem cells and cancer

A. Personal Statement

The molecular mechanisms of stress responses in stem cells and during cancer progression have always been my main area of interest and fascination. Over the past 15 years I have worked on several different aspects of this incredibly broad field. During my PhD training I investigated the molecular mechanisms by which activated oncogenes induce DNA damage and cellular senescence. I was able to demonstrate that uncontrolled hyper-proliferation of oncogene expressing cells ultimately leads to DNA breaks and the activation of senescence as a tumor suppressor mechanism (*Di Micco et al. Nature 2006*). After graduation, I focused on the study of alterations of chromatin structure during senescence and discovered that heterochromatin formation induced by oncogenes is the consequence of DNA replication stress in the cells. I studied how alterations in chromatin could lead to new targeted therapies aimed to re-establish normal epigenetic patterns in cancer settings *(Di Micco et al., Nat Cell Biol 2011; Sulli et al., Nature Rev Cancer 2012*). In 2010, I moved to United States for a postdoctoral training in the laboratory of Dr. Eva Hernando at NYU School of Medicine to perform research aimed at understanding the epigenetic and transcriptional regulation in stem cell and during cancer development. In the first year of my postdoctoral training I was awarded with a prestigious European Molecular Biology Organization (EMBO) fellowship and in the second year I received a postdoctoral fellowship from Human Frontier Science Project (HFSP). Soon after I joined the lab, I became part of a study focused on the role of miRNA regulation in melanoma metastasis that deserved me a co-authorship in a manuscript published in *Cancer Cell*. Later, by using genetic and chemical-based approaches, both targeted and genome-wide, I identified novel molecular mechanisms involved in stem cell maintenance, with a particular focus on the epigenetic and transcriptional regulator BRD4. These findings were published in *Cell Reports* and as leader of the project I am the corresponding author (Di Micco et al., 2014). This work was supported by the New York Stem Cell Foundation (NYSCF) Druckenmiller postdoctoral fellowship. The fellowship program gave me the opportunity to gain visibility and to establish collaborations and interactions with outstanding scientists in the field of stem cell biology field. More recently, I contributed to a comprehensive study on the mechanisms of BRD4-dependent regulation of enhancer elements in melanoma survival (*Fontanals-Cirera Mol Cell 2017*) that led to the identification of new therapeutic targets for cancer treatment. Seeking for a group leader position, I took part to selective and competitive job interview

processes in leading institutions in Europe and United States. I decided to establish my research team at the San Raffaele-Telethon Institute for Gene Therapy (SR-TIGET) within the San Raffaele Hospital, in Milan in early 2016. The research in my lab capitalizes on the scientific expertise of my PhD and postdoctoral trainings in DNA damage, senescence, epigeneticsand cancer and involves, as a model system, the human hematopoietic stem and progenitor cells (HSPCs). The main goal ofmy lab is to dissect the interplay between chromatin and DNA damage upon stress in normal and aged stem cells and in the context of malignant hematopoiesis with the final aim to develop hypothesis-driven strategies for therapeutic applications. I envision a research career that is pioneering and translational. Our laboratory perfectly integrates within a multidisciplinary team of basic biologists, statisticians, hematologists, clinicians with different yet complementary sets of expertise and takes advantage of relevant human patient samples. Our institute is an internationally recognized research center in the field of normal and malignant hematopoiesis and provided me a fully equipped and renovated lab space, state-of-art technologies and cutting- edge support facilities and infrastructures to promote scientific excellence and innovation.

B. Positions and Honors

Positions and Employment

2004-2008 Predoctoral Student at the European School of Molecular Medicine (SEMM), IFOM, Milan
2008-2010 Postdoctoral researcher at IFOM, Milan
2010-2015 Postdoctoral researcher at the NYU Langone Medical Center
2015- Group Leader at the San Raffaele Hospital
2024- Associate Professor in Pathology at the School for Advanced Studies (IUSS), Pavia
2025- Coordinator of the curriculum in Gene and Cell Therapies at the National Ph.D program in Science and Technology for Advanced Therapies (STAT) at IUSS

Professional Memberships

2010 Member of American Association Cancer Research (AACR)
2010 Member of International Society for stem cell research (ISSCR)
2013 Member of New York Stem Cell Foundation (NYSCF)
2016 Member of European Society of Cell and Gene Therapy (ESCGT)
2016 Member of the European Hematology Association (EHA)
2017 Member of Società Italiana di Biofisica e Biologia Molecolare (SIBBM)
2019 Member of the American Society of Hematology (ASH)
2019 Member of the European Hematology Association (EHA)
2020 Member of the American Society of Cell and Gene Therapy (ASGCT)
2021 Member of the American Society of Cell and Gene Therapy (ASGCT)
2021 Member of European Society of Cell and Gene Therapy (ESCGT)
2022 Member of the European Hematology Association (EHA)
2022 Member of European Society of Cell and Gene Therapy (ESCGT)
2022 Member of the American Society of Cell and Gene Therapy (ASGCT)
2022 Member of European Association for Cancer Research (EACR)

Reviewer Experience

2015-present Journal Reviewer:	*Cell, Nature Cell Biology, Nature, Nature Genetics, Nature Communications, Molecular Cell, Cell Reports, AgingCell, Cell Death and Disease, Cell Stem Cell, Faseb J, Trends in Cell Biology, Plos One, Developmental Cell, Cells, Frontiers in Oncology, Mechanisms of Ageing and Development, Science Immunology, Nature Cancer.*
2015-present Grant Reviewer:	*Italian Ministry of Health, Polish National Science Centre, French National ResearchAgency (ANR), Swiss National Science Foundation, Atip - Avenir program by INSERM*
2019-present Abstract Reviewer:	*Annual Meeting of the International Society of Stem Cell Research (ISSCR Boston 2020);Annual Meeting of the European Hematology Association (EHA Frankfurt 2020); International Cell Senescence Association (Athens 2019).*
2019	Guest Editor of the special issue: "Advances in Senescence" in *Mechanisms of Ageing and Development*

Honors

2007 Research award P. SCHLECTER e L. CESCATTI, Foundation for Cancer Research
2010 European Molecular Biology Organization (EMBO) postdoctoral fellowship: 'Impact of melanocyte differentiation pathogenesis'
2011 Human Frontier Science Project (HFSP) postdoctoral fellowship: 'Epigenetic regulation of melanocyte differentiation during melanoma pathogenesis'
2013 New York Stem Cell Foundation Druckenmiller fellowship: 'Role of BRD proteins in stem cell regulation and cancer"
2014 Offered a Group Leader position at Research Center for Molecular Medicine of the Austrian academy of Science, CEMM, Vienna. Declined
2014 Offered a Group Leader position at Istituto Nazionale di Genetica Molecolare INGM. Declined
2015 Mobility Research Program of Human Frontier Science Project (HFSP)
2019 Selected for participation to the Interstellar Initiative of the New York Academy of Science and Japan Agency for Medical Research and Development
2019 Awarded "Under 40 in Hematology Award" for Best Translational study form the Italian Society of Hematology
2020 Awarded the New York Stem Cell Foundation Robertson Award
2022 Awarded the ERC Consolidator grant

Patents

1)GT protocols using p38i and anti-inflammatory drugs. WO2023/066735A1
2) 3D scaffolds in GT. WO2024/079644A1

C. Contributions to Science

Early publications during my PhD training directly addressed the impact of activated oncogenes on cellular senescence and on dynamics of DNA replication in normal cells. At that time, oncogene-induced senescence was thought to be a program of mere proliferative arrest. I demonstrated that the uncontrolled hyper-proliferation of oncogene expressing cells ultimately leads to DNA breaks and the activation of tumor suppressor mechanisms. When tumor suppressors and cell cycle inhibitors are inactivated, oncogene-expressing cells can bypass cellular senescence and proliferate bearing DNA breaks and being genomic unstable. This study represents a cornerstone in the senescence and cancer field. This study deserved publication in the prestigious journal *Nature* and has been heavily cited underlining the relevance of my findings. In addition, I demonstrated that the activation of DNA damage-response (DDR) is conserved in murine settings. I also authored a comprehensive review on mechanisms of oncogene-induced senescence in Trends in Cell Biology.

a) Di Micco R., Fumagalli M., Cicalese A., Piccinin S. Gasparini P., Luise C., Schurra C.,Garre' M.,NuciforoP.Bensimon A., Maestro R., Pelicci P.G., and d'Adda di Fagagna F. Oncogene-induced senescence is a DNA-damage checkpoint response triggered by DNA hyper-replication. **Nature**, 2006. *News and views by L. Cao and T. Finkel in Nature Medicine. Faculty of 1000: Highlighted as "Must read". FFa = 9*

b) Di Micco R., Cicalese A., Fumagalli M., Dobreva M., Verrecchia A., Pelicci P.G., and d'Adda di Fagagna F. DNA damage response activation in mouse embryonic fibroblasts undergoing replicative senescence and following spontaneous immortalization. **Cell Cycle**, 2008.

c) Di Micco R., Fumagalli M. and d'Adda di Fagagna F. Breaking news: high speed run away ends in arrest. How oncogenes induce senescence. **Trends in Cell Biology**, 2007.

During the second part of my PhD training and in the following years after graduation, I focused on the study of alterations of chromatin structure during senescence and discovered that heterochromatin formation induced by oncogenes is the consequence of DNA replication stress in the cells. In particular, I found that heterochromatin induced upon genotoxic stress restrains the activation of DDR. Targeted therapy that aims to perturb heterochromatin in oncogene-expressing cells leads to DDR activation and spreading, resulting in cell death. These findings suggest that novel targeted therapies aimed tore-establish epigenetic patterns in tumor cells could be therapeutically exploited in cancer settings. This study deserved a publication in *Nature Cell Biology*. We have also published a comprehensive review in *Nature Reviews Cancer* that analyzed the state of art of the potential interplay between heterochromatin and DDR in senescence and cancer. During that time, I was also involved in other studies elucidating the role of DNA replication stress at telomeres and the impact of reactive oxygen species in mediating oncogene-induced senescence.

a) **Di Micco R.,** Sulli G.,... and d'Adda di Fagagna F. Interplay between oncogene-induced DNA damage response and heterochromatin in senescence and cancer. **Nature Cell Biology**, 2011. *Commentary by P. Adams in Nature Cell Biology.Commentary by T. Halazonetis in Cell Cycle.Faculty of 1000: Highlighted as "Must read". FFa = 12*
b) Suram A, Kaplunov J, …, **Di Micco R,** Mirani N, Gurung RL, Hande MP, d'Adda di Fagagna F, Herbig U. Oncogene-induced telomere dysfunction enforces cellular senescence in human cancer precursor lesions. **EMBO J.,** 2012. *Commentary by K.L. Rudolph in the same issue of EMBO Journal*. *Commentary by J.J. Jacobs in Nature Reviews Molecular and Cellular Biology*
c) Sulli G., **Di Micco R.** and d'Adda di Fagagna F. Crasstalk between chromatin state and DNA damage response in senescence and cancer. **Nat Rev Cancer.** Oct 2012.

d) Ogrunc M., **Di Micco R.**, Liontos M., …, d'Adda di Fagagna F. Oncogene-induced reactive oxygen species fuel hyper-proliferation and DNA damage response activation. **Cell Death and Differentiation**, 2014.

In 2010, I moved to US for a postdoctoral training in the laboratory of Dr. Eva Hernando at NYU School of Medicine.The Hernando's lab focuses on understanding the role of epigenetic regulators (miRNA and chromatin remodelers) in melanoma initiation and progression. Soon after I joined the lab I became part of a study focused on the role of miRNA in melanoma (miR30b/30d) are over-expessed in melanoma and directly regulate the expression of GalNac transferases, to enhance invasion and immunosuppression during melanoma metastasis. As part of this study I deserved a co-authorship in amanuscript published *Cancer Cell.* My main scientific interest remained understanding the epigenetic and transcriptional regulation of stem cell identity and cancer development. I first focused on the role of epigenetic readers, bromodomain and extra-terminal domain proteins (BET) in controlling stem cell identity and cell fate decisions. I discovered that BRD4, a BETfamily member is a critical regulator of stemness. BRD4 chemical and genetic inhibition impairs self-renewal capacity of human embryonic stem cells. Furthermore, BRD4 depletion drives cell differentiation toward the neuroectodermal lineage *invitro* and in teratoma assays *in vivo*. Combining cell and molecular biology approaches, together with next generation sequencing methods, I discovered a novel mechanism by which BRD4 controls the transcriptional elongation of key cell identity genes that are associated with so-called super-enhancers (SE) and regulate lineage specification. These findings are published in *Cell Reports* and as leader of the project I am co-corresponding author. I pioneered the field of epigenetics in a lab with no previous experience and I opened new research lines focused on transcriptional and epigenetic regulation of melanoma genesis and metastasis. In particular, by using an integrative epigenomic approach I demonstrated that BRD4 regulates the expression of important melanoma oncogenes by binding to their enhancer regulatory elements. Conversely, ChIP-seq analysis for histone repressive marks and chromatin repression indicated repressive domains spanning kilobases attumor suppressors genes. This innovative approach led to the identification of AMIGO2 as a novel oncogene and therapeutictarget in melanoma.

a) Gaziel-Sovran A, Segura MF, **Di Micco R**, …, Hernando E. miR-30b/30d regulation of GalNAc transferases enhances invasion and immunosuppression during metastasis. **Cancer Cell,** 2011.
b) **Di Micco R.§,** Fontanals-Cirera B., Low V., ..., Aifantis I., Zhou M.M., Tsirigos A.§ and Hernando E.§ Control of embryonic stem cell identity by BRD4-dependent transcriptional elongation of super-enhancer associated pluripotency genes. **Cell Reports**, 2014. §Corresponding author.
c) Fontanals-Cirera B., Hasson, D, Vardabasso, C., **Di Micco R,** Agrawal P, …, Hernando E, Bernstein E. Harnessing BET inhibitor sensitivity reveals AMIGO2 as a melanoma survival gene. **Mol Cell** 2017.

In 2016 I established my own laboratory at the San Raffaele Hospital. The research lines of my lab fully capitalize on my expertise in the field of DNA damage and epigenetics. Given the longstanding leadership of the host institution in the field of genetic engineering of human hematopoiesis and in innovative translational research applied to hematological malignancies, we chose hematopoietic cells as an experimental model system. Research in my lab focuses on understanding the cellular responses to stress in hematopoietic stem cells and in hematological malignancies.Research projects in my lab aim at dissecting the contribution of senescence in the aged hematopoietic niche and at modelingthe role of senescence programs in response to therapy and relapse.

a) Schiroli G.* Conti A.*, …, Naldini L. and **Di Micco R.**[&]. Precise Gene Editing Preserves Hematopoietic Stem Cell Function Following Transient p53-Mediated DNA Damage Response. **Cell Stem Cell** 2019**.** [&] last and corresponding author.
b) Conti A. and **Di Micco R§.** p53 activation: a checkpoint for precision genome editing? **Genome Medicine,** 2018.
c) **Di Micco R.** and Montini E. §. De(bar)coding aged hematopoiesis. **Blood** 2018. § corresponding author
d) **Di Micco R. *§.*** Sensing the breaks: cytosolic chromatin in senescence and cancer.**Trends in Molecular Medicine,** 2017**.**
e) Gnani D, Crippa S, Della Volpe L, …, **Di Micco R.§.** An early-senescence state in aged mesenchymal stromal cells contributes to hematopoietic stem and progenitor cell clonogenic impairment through the activation of a pro-inflammatory

program. **Aging Cell**. 2019. PMID: 30828977. § corresponding author

f) Gambacorta V. Gnani D., Vago L. and **Di Micco R§.** Epigenetic Therapies for Acute Myeloid Leukemia and Their Immune-Related Effects Review in **Frontiers in Cell and Developmental Biology** 2019.§ corresponding author

g) Biavasco R, Lettera E, Giannetti K, …, **Di Micco R§**, Montini E. Oncogene-induced senescence in hematopoietic progenitors features myeloid restricted hematopoiesis, chronic inflammation and histiocytosis. **Nature Communications**. 2021. §corresponding author

h)Marcovecchio GE, Ferrua F, …., **Di Micco R**, Merelli I, Bosticardo M, Villa A. Premature Senescence and Increased Oxidative Stress in the Thymus of Down Syndrome Patients. **Front Immunol**. 2021

i)Mangiameli E, Cecchele A, …, **Di Micco R**, Bachi A, Gritti A. Human iPSC-based neurodevelopmental models of globoid cell leukodystrophy uncover patient- and cell type-specific disease phenotypes. **Stem Cell Reports**. 2021.

l) Giordano AMS, Luciani M, …, **Di Micco R**, …, Kajaste-Rudnitski A. DNA damage contributes to neurotoxic inflammation in Aicardi-Goutières syndrome astrocytes. **J Exp Med**. 2022.

m) Di Micco R. §, Krizhanovsky V. Baker D. and d'Adda di Fagagna[§]. Cellular senescence in ageing: from mechanisms to therapeutic opportunities Nature Reviews Molecular and Cellular Biology (Special Issue on Aging). § corresponding author

n)Gambacorta V, Beretta S, Ciccimarra M, Zito L, …, **Di Micco R§**, Vago L. Integrated Multiomic Profiling Identifies the Epigenetic Regulator PRC2 as a Therapeutic Target to Counteract Leukemia Immune Escape and Relapse. **Cancer Discov.** 2022 Jun 2. PMID: 35255120. **§**corresponding author

o) Milardi G, …, **Di Micco R**, Ponzoni M, Aiuti A, Cicalese MP, Fousteri G. Follicular helper T cell signature of replicative exhaustion, apoptosis, and senescence in common variable immunodeficiency. **Eur J Immunol**. 2022.

p) Ferrari S, Jacob A, Cesana D, …., **Di Micco R**, Kajaste-Rudnitski A, Montini E, Penaud-Budloo M, Naldini L. Choice of template delivery mitigates the genotoxic risk and adverse impact of editing in human hematopoietic stem cells. **Cell Stem Cell**. 2022. Review

q) Montaldo E, Lusito E, Bianchessi V, …, **Di Micco R**, Ditadi A, …, Ostuni R. Cellular and transcriptional dynamics of human neutrophils at steady state and upon stress. **Nat Immunol.** 2022.

r)Crippa S*, Conti A*, .., Naldini L, **Di Micco R**, Bernardo ME. Mesenchymal stromal cells improve the transplantation outcome of CRISPR-Cas9 gene-edited human HSPCs. **Mol Ther**. 2023.

s)della Volpe L. Midena F, Vacca R, Conti A,..., **Di Micco R**. Inhibition of p38-MAPK counteracts culture stress induced by ex vivo expansion of HSPCs for efficient genetic engineering. **Cell Reports Medicine 2024**§ corresponding author

t)Conti A, De Marco R, Tavella T, …, I, **Di Micco R**. Senescence and Inflammatory Programs are Unintended Consequences of CRISPR-Cas9 Gene Editing in human HSPCs. **Cell Reports Medicine 2025**§ corresponding author

u)Della volpe L., Vacca R., **Di Micco R. STAR Protocols 2025**§ corresponding author

v)Lettera E, Scala S, Tavella T, Conti A, …, A, **Di Micco R**§. Molecular and phenotypic blueprint of human hematopoiesis links proliferation stress to stem cell aging. **Journal of Experimental Medicine.** ***2026*** § corresponding author

x)Midena F, Alessandrini L.,, **Di Micco R**§. Nanoengineered 3D culture substrate enables superior long-term and polyclonal engraftment of genetically engineered hematopoietic stem cells. **Cell Stem Cell.** ***2026*** § corresponding author

Advanced manuscripts

a) Gilioli D., Fusco S, …… **Di Micco R**§. Therapy-Induced Senescence Promotes Immunogenicity in Acute Myeloid Leukemia Blasts through loss of PRC2-Mediated Gene Regulation. **Nature Communications.** ***Under invited revision.***

b) Vacca R., della Volpe… **Di Micco R**§. p38 MAPK inhibition safeguards genome integrity during gene editing of hematopoietic stem and progenitor cells. **Molecular Therapy.** ***Under review.***

c) Farina G. … **Di Micco R**§. Metabolic Control of Chromatin Accessibility Reverses Age-Related Dysfunction in Human Hematopoietic Stem and Progenitor Cells. In preparation for **Nature Aging**

d) Sconocchia … **Di Micco R**§. Selective elimination of hematopoietic senescent cells via CAR-T as a therapeutic strategy for oncogene-induced hematopioetic clonal neoplasms. In preparation for **Nature Cancer**

D. Past and Ongoing Research Support

2016-2019	OSR Pilot and Seed grant 2015 (2016-2019; 210K): Identification and targeting of epigenetic drivers of immune evasion in AML relapses after allogeneic HSCT
2016-2021	Telethon Grant (2016-2020; 965K): Hematopoietic Stem Cell aging in physiology and disease
2019-2020	Leukemia Research Foundation (100K): Targeting of epigenetic drivers of immune evasion in acute myeloid leukemia relapses after allogeneic HCT.

2019-2021	European Hematology Association Advanced Research Grant (160k euros): Dissecting cell-autonomous and paracrine functions of cell senescence in AML response to therapy
2019-2022	Human Frontier Science Program (300K): Elucidating the biological impact of precise genome editing in hematopoietic stem cells.
2019-2020	Interstellar Initiative on Healthy Longevity from New York Academy of Sciences and Japan Agency for Medical Research and Development
2020-2025	My First AIRC grant(500K) Italian Association for Cancer Research: Targeting epigenetic drivers of leukemia Catalyst Award on Healthy longevity from Japan Agency for Medical Research and Development (30K)
2021-2026	New York Stem Cell Investigator Robertson Award (1.5M USD)
2021-2023	NC3Rs Crack-IT challenge (Multi-PI). CleanCut: Developing a human in vitro model to assess the safety of gene edited human haemopoietic stem cells. (1M euros)
2022-2027	European Research Council Consolidator Grant 2020 (2M Euros)
2023-2026	RF-2021-12373598 Ministry of Health: Uncover and overcome senescence and dysfunction of genetically engineered T lymphocytes for cancer immunotherapy (collaborator for the grant funded to Prof. C.Bonini)
2022-2025	PNRR-MAD-2022-1237667.Dissecting the molecular and cellular pathophysiology of sarcopenic obesity (MultiPI). (1M Euros)
2022-2026	X-PAND project: Exploiting ex vivo expansion and deep multiomics profiling to bring novel, efficient and safer hematopoietic stem cell gene therapies to clinical application. (3,8M Euros for 6 partners)
2023-2026	PNRR-MCNT2-2023-12377474 Dissecting the molecular and cellular pathophysiology of sarcopenic obesity in the elderly (MultiPI).(1M euros)
2023-2026	PNRR-AgeIT Ageing well in an ageing society (PI) (170,000 euros)
2024-2027	Marie Curie ITN Doctoral Network: IMMERGE (254,000 euros)
2025-2028	Marie Curie ITN Doctoral Network: UNION (254,000 euros)
2025-2028	Marie Curie ITN Doctoral Network: MIRACLE (254,000 euros)

E. Past and Ongoing Research Support to laboratory members

2017-2019	Anastasia Conti	Postdoctoral AIRC-FIRC fellowship: Transcription and epigenetic regulation of transposable elements in acute myeloid leukemia pathogenesis (75,000 Euros)
2018	Daniela Gnani	Postdoctoral IBSA Foundation fellowship: Transcriptional regulation of senescence induced by oncogenes during tumor progression (30,000 Euros)
2020	Anastasia Conti	EHA-ASH TRTH Program 2020 winner: Transposable Elements and cell senescence are new molecular players in AML onset and response to therapy (20,000 Euros)
2020	Anastasia Conti	Postdoctoral International Award for Research in Leukemia by The Lady Tata Memorial Trust: Elucidating the functional role of cell senescence and transposable elements reactivation in acute myeloid leukemia response to therapy (35,000 Pounds)
2021-2022	Lucrezia della Volpe	Postdoctoral Fondazione Fronzaroli fellowship: Gaining mechanistic insights to advance hematopoietic stem cell based gene and cell therapies (50,000 Euros)
2022-2024	Antonella Santoro	Postdoctoral EMBO fellowship: Harnessing senescence-primed immune effects to eradicate leukemia and overcome resistance to therapy (132,000 Euros)
2023-2025	Simona Fusco	Predoctoral AIRC-FIRC fellowship for Italy: Dissecting the role of cellular senescence in Acute Myeloid Leukemia response to therapy (75,000 Euros)
2023	Antonella Santoro	HORIZON-MSCA-2022-PF-01-01: SLIM "Harnessing senescence-primed immune responses to eradicate leukaemia and overcome resistance to therapy" (260,000 euros) AIRC-FIRC postdoctoral fellowship (105,000 euros)
2024	Antonella Santoro	EHA Advanced Research Grant (240,000 euros)
2025	Anastasia Conti	Junior Researcher at School for Advanced Studies, IUSS, Pavia
2025	Antonella Santoro	Fondazione Roche per la Ricerca Indipendente Grant (50,000 euros)
2025	Anastasia Conti	Giovani Ricercatori per la Ricerca Finalizzata by the Italian Ministry of Health
2026	Anastasia Cont	(450,000 euros)