BIOGRAPHICAL SKETCH

NAME: Francesca Arruga

ORCID number: **http://orcid.org/0000-0001-5298-9314**

POSITION TITLE: Biologist Geneticist (permanent position)

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY	FINAL MARK
University of Turin	Bachelor Degree	2000-2003	Biotechnology	105/110
University of Turin	Master Degree	2003-2005	Medical Biotechnology	110/110 L
University of Turin	PhD	2005-2009	Pharmacology and Clinical and Experimental Therapies	Not applicable
University of Turin	Board Certification	2010-2015	Clinical Pathology	70/70

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Personal statement

Dr. Arruga obtained her Master Degree in Medical Biotechnology, with honors, with a thesis on the inhibition of NFκB pathway as a strategy to overcome resistance to Imatinib in Chronic Myeloid Leukemia (CML) patients. During her PhD she was part of the team working on the generation of a genetic model of CML, based on a transgenic Drosophila melanogaster carrying the fusion transcript BCR-ABL, both in a wild-type (WT) and a mutant form, responsible for therapy resistance (T315I). As a Post-Doc she was awarded the Gigi Ghirotti Foundation fellowship, with a project about the use of *WT1* gene expression to monitor minimal residual disease in patients undergoing allogeneic transplantation.

Since 2011, Dr. Arruga joined the Immunogenetics Research Unit at the Italian Institute for Genomic Medicine (IIGM, formerly Human Genetics Foundation HuGeF), Dept. of Medical Sciences, University of Turin (Head: Prof. S. Deaglio). During these years, Dr. Arruga worked on the analysis and functional characterization of recurrently mutated genes in chronic lymphoproliferative diseases, and applied the more advanced genome editing technologies to recapitulate *in vitro* the most frequent mutations. To this, in 2012 she attended a course at the European Molecular Biology Laboratory (EMBL) in Heidelberg about Zinc Finger Nuclease and CRISPR/Cas9 technologies, both groundbreaking precision genomic systems. Specifically, her work has been focused on *NOTCH1, NOTCH2 e KLF2* genes.

Over the years she became a proficient molecular biologist and acquired experience in the experimental design and set-up of cloning and genome editing strategies, RNA sequencing (library preparation, analysis of differentially expressed sequences, GSEA enrichment, gene ontology enrichment and clustering, statistical analyses and basic R usage), ChIP assays, promoter methylation analyses (bisulfite convertion-based PCR), polysome profiling for the analysis of differentially translated elements. In 2018 she attended a course by University of Insubria to learn sequencing data generation, big data analysis and interpretation through bioinformatic tools.

In 2018, she spent a period as a visiting scientist at the University of Southampton (Reference: Dr. Francesco Forconi) to study the role of *NOTCH1* mutations in the regulation of protein translation in lymphoproliferative malignancies.

Her results were published on the top journals in the field and were also part of a network of collaborators that Dr. Arruga consolidated over the years, and that included Dr. D. Rossi and Prof. G.

Gaidano (University of Eastern Piedmont, Italy), Dr. Piva (Dept. Molecular Biotechnology and Health Sciences), Dr. V. Gattei (IRCCS/Centro di Riferimento Oncologico, Aviano) and Dr. F. Forconi (University of Southampton). Dr. Arruga's research was partly funded by the "Young Investigator Programme" fellowship, granted by Umberto Veronesi Foundation in 2013, and the SIES-Beat Leukemia fellowship, granted by the Italian Society of Experimental Hematology in 2015.

Since 2019, she is a collaborator of the Immunogenetics of Organ Transplantation Unit (Director: Prof. Antonio Amoroso) for the Sanger sequencing validation of genetic variants found in patients eligible for organ transplantation by exome sequencing, and for the monitoring of transplant rejection by measuring circulating donor-derived cell-free DNA with droplet-digital PCR. Furthermore, she is involved in a research project aimed at generating cell line models to functionally validate mutations found in patients affected by polycystic kidney disease.

Over the past years, Dr. Arruga acquired growing independence in experiment and study design, in data analysis and interpretation, also through bioinformatic tools, and in coordinating junior Post-Docs and Lab technicians, as well as in project and paper writing. Furthermore, Dr. Arruga progressively established a number of strong scientific collaborations with italian and foreign researchers, that resulted in several scientific papers.

Employments and awards

- 2002: Internal Student, Lab. Of Cellular Biology, Dept. Of Genetics, Biology and Biochemistry, University of Turin, Italy. Supervisor: Prof. G. Tarone.
- 2002-2005: Internal Student, Lab. Of Medicine and Molecular Oncology, Dept. Of Clinical and Biological Sciences, University of Turin, Italy. Supervisor: Prof. G. Saglio, M.D., Ph.D.
- 2005-2009: PhD Student, Lab. Of Medicine and Molecular Oncology, Dept. Of Clinical and Biological Sciences, University of Turin, Italy. Supervisor: Prof. G. Saglio, M.D., Ph.D.
- 2010: **PhD in Pharmacology and Clinical and Experimental Therapies**, University of Turin.
- 2009: Gigi Ghirotti Foundation Fellowship, Lab. Of Medicine and Molecular Oncology, Dept. Of Clinical and Biological Sciences, University of Turin, Italy. Supervisor: Prof. G. Saglio, M.D., Ph.D.
- Jan 2010- Jul 2011: Nippon Inc. fellowship, Lab. Of Medicine and Molecular Oncology, Dept. Of Clinical and Biological Sciences, University of Turin, Italy. Supervisor: Prof. G. Saglio, M.D., Ph.D.
- Sept 2011- Dec 2012: Post-Doc fellowship, Immunogenetics Research Unit, Human Genetics Foundation (HuGeF) and Dept. Of Genetics, Biology and Biochemistry, University of Turin, Italy. Supervisor: Prof. S. Deaglio, M.D., Ph.D.
- 2013: "Young Investigator Programme" fellowship by the Umberto Veronesi Foundation. Dept. Medical Sciences and Human Genetics Foundation (HuGeF), University of Turin, Italy. Supervisor: Prof. S. Deaglio, M.D., Ph.D.
- 2014: Post-Doc Fellowship (Assegno di ricerca), Immunogenetics Research Unit, Human Genetics Foundation (HuGeF), Dept. Medical Sciences, University of Turin, Italy. Supervisor: Prof. S. Deaglio, M.D., Ph.D.
- 2015: SIES-Beat Leukemia fellowship (Italian Society of Experimental Hematology), Immunogenetics Research Unit, Human Genetics Foundation (HuGeF), Dept. Medical Sciences, University of Turin, Italy. Supervisor: Prof. S. Deaglio, M.D., Ph.D.
- 2015: **Board Certification in Clinical Pathology**. University of Turin, final mark 70/70
- 2016-2018: Senior Post-Doc, fixed-term contract. Immunogenetics Research Unit, Italian Institute for Genomic Medicine (IIGM), Dept. Medical Sciences, University of Turin, Italy. Supervisor: Prof. S. Deaglio, M.D., Ph.D.
- 2018: **Visiting Scientist at the University of Southampton**, Cancer Sciences Unit, Haematological Oncology Group, Southampton, UK.

- 01/01/2019-30/06/2022. Senior Post-Doc Researcher. Three-years Assegno di Ricerca. Dept. Medical Sciences, University of Turin, Italy. Supervisor: Prof. S. Deaglio, M.D., Ph.D.
- 2019 -: **National professional qualification as Biologist**
- 01/07/2022-Present: Biologist, full time contract, Immunogenetics and Transplantation Biology, Città della Salute e della Scienza Hospital, Torino

Educational activity and mentoring:

- 2006-2011: Supervisor of 8 internal students (School of Medicine, Biology and Biotechnology) Lab. Of Medicine and Molecular Oncology, Dept. Of Clinical and Biological Sciences, University of Turin, Italy.
- 2013: Supervisor of an internal student (Dr. V. Tani) from the Biotechnology School, University of Florence.
- 2014: Supervisor of an internal student (Dr. D. Fusco) from the Biology School, University of Turin.
- 2012-2013: Supervisor of a PhD student (Dr. B. Gizdic) from University of Zagreb, Croatia.
- 2014-2017: Supervisor of junior post-doctoral fellows (Dr. B. Gizdic, Dr. R. Buonincontri, Dr. C. Bologna, Dr. S. Cignetto)
- 2015-present: Supervisor of post-graduate fellow (Dr. V. Bracciamà)
- 2016: Supervisor of an internal student (Dr. F. Lamberto) from the Biology School, University of Turin
- 2017: Supervision of a Post-graduated Master student (Dr. I. Battaglia)
- 2018: Supervision of an undegraduated medical student (L. Cravero)
- 2011-2017: Lab practice internship in Medical Genetics for students of the School of Medicine (I-II year) at the Italian Institute for Genomic Medicine
- 2015-present: Teaching assistant in Medical Genetics, Nursing School and Pediatric Nursing School, University of Turin; Lecturer: Prof. S. Deaglio, M.D., Ph.D.
- 2019-present: Teaching assistant in Medical Genetics, Biomedical Laboratory Technicians School, University of Turin; Lecturer: Dr. T. Vaisitti, Ph.D.

Professional Membership

Italian Society for Experimental Hematology (SIES)
American Association for Cancer Research
European Haematology Association
Italian Society for Human Genetics (SIGU)

Awards:

2008: Travel grant from the European Haematology Association, 13° Annual Meeting
2017: Travel grant from iwCLL

Reviewer and editorial activities for:

2016: reviewer for Leukemia and Lymphoma Journal
2017: Member of the editorial board of the Journal of Gene Therapy and Research
2017: reviewer for Leukemia Journal
2018: Member of the Editorial board of the journal Annals of Genetics and Molecular Biology
2019: Guest Editor for the Special Issue of Cancers journal on “Chronic Lymphocytic Leukemia”
Reviewer for Frontiers in Oncology
2020: Reviewer for Cancers, IJMS, MDPI
2021: Guest Editor for the Special Issue of Cancers journal on “Latest Development in B-cell Malignancies”

Scientific Contributions

1. Identification and functional validation of novel genetic lesions in onco-hematological diseases. The current main topic of Dr. Arruga's research is dedicated to the identification and functional validation of novel genetic lesions in lymphoproliferative diseases, such as Chronic Lymphocytic Leukemia, its transformation to an aggressive lymphoma (Richter Syndrome, RS) and the Splenic Marginal Zone Lymphoma (SMZL). Her activity is focused on the impact of recurrently mutated genes driving disease evolution and transformation to RS, that represent the most aggressive stage of the disease and is characterized by important chemoresistance. As part of a collaboration network, attention has been dedicated on the identification of genetic lesion characterizing high-risk patients. The next steps were focused on the study of the functional impact of these lesions, by dissecting the deregulated molecular mechanisms and identifying novel potential interactors. Specifically, by using the most ground-breaking genome editing systems, Dr. Arruga generated unique cellular and *in vivo* models to recapitulate the disease to in depth study the molecular and biological mechanism that were deregulated upon mutations. She applied genomic and molecular biology tools to characterize the gene expression profile (RNAseq), mechanisms of regulation of transcription (ChIP assays and methylation profile) and translation (polysome profiling) of genetically engineered cell lines and primary leukemic cells. This topic has a strong translational relevance as results may offer a rationale for designing novel targeted therapeutic strategies, to overcome poor responsiveness to conventional treatment options that is frequently observed in this subset of patients. In the last years. Dr. Arruga focused her research on the study of crosstalks between signaling pathways in CLL microenvironment, with specific attention to NOTCH1 pathway and B cell receptor signaling. Furthermore, she actively collaborated to a project aimed at the generation and genetic and functional validation of *in vivo* mouse models to study RS. Dr. Arruga contributed to the establishment of patient-derived xenograft models of the disease and to the RNA-seq mediated analysis of the transcriptome to identify the main pathways involved in the disease transformation and maintenance.

1. Rossi, D., et al., *The coding genome of splenic marginal zone lymphoma: activation of NOTCH2 and other pathways regulating marginal zone development.* The Journal of experimental medicine, 2012. **209**(9): p. 1537-51.
2. Arruga, F., et al., *Functional impact of NOTCH1 mutations in chronic lymphocytic leukemia.* Leukemia, 2014. **28**(5): p. 1060-70.
3. Pozzo, F., et al., *NOTCH1 mutations associate with low CD20 level in chronic lymphocytic leukemia: evidence for a NOTCH1 mutation-driven epigenetic dysregulation.* Leukemia, 2016. **30**(1): p. 182-9.
4. Arruga, F., et al., *Mutations in NOTCH1 PEST-domain orchestrate CCL19-driven homing of Chronic Lymphocytic Leukemia cells by modulating the tumor suppressor gene DUSP22.* Leukemia, 2017.
5. Vaisitti T, Braggio E, Allan JN, Arruga F, Serra S, Zamò A, Tam W, Chadburn A, Furman RR, Deaglio S. *Novel Richter's syndrome xenograft models to study genetic architecture, biology and therapy responses*. Cancer Res. 2018 May 7. pii: canres.4004.2017. doi: 10.1158/0008-5472.CAN-17-4004.
6. Arruga F, Bracciamà V, Vitale N, Vaisitti T, Gizzi K, Yeomans A, Coscia M, D'Aren G, Gaidano G, Allan JN, Furman RR, Packham G, Forconi F, Deaglio S. Bidirectional linkage between the B-cell receptor and NOTCH1 in Chronic Lymphocytic Leukemia and in Richter's syndrome: therapeutic implications. Leukemia. 2019 Aug 29. doi: 10.1038/s41375-019-0571-0. [Epub ahead of print].

2. Analysis of mutations/chromosomal abnormalities with relevance in the clinical practice. Beside the functional characterization of mutated genes, part of Dr.Arruga's work has been dedicated to the analysis of genetic alterations with relevance in the clinical practice. In the laboratories of Medicine and Molecular Oncology she acquired growing independence and became the reference person for the Sanger sequencing analysis of BCR-ABL tyrosine-kinase domain mutations, which represent a prognostic factor influencing therapeutic decision. Similarly, she was in charge of the screening for FIP1L1/PDGFR

translocation, a powerful diagnostic marker for hypereosinophilic syndromes. At the IIGM, she routinely screens for mutations in *NOTCH1* PEST domain by allele specific PCR and will participate in a study to monitor recurrently mutated genes during CLL evolution by droplet digital PCR. In the last years, Dr. Arruga was involved in the screening and genotyping of polymorphisms potentially relevant in the clinical practice. She supervised Dr. I. Battaglia for the genotyping of rs893403, a polymorphism associated with *LIMS1* deletion, which may be potentially relevant in acute kidney rejection. More recently, she contributed to the genotyping analysis of 7 polymorphism in 5 genes (*COMT*, *NOS3*, *GRK5*, *ADRB1*, *ADRB2*) potentially associated with predisposition to myocardial infarction. Since 2019, she is collaborating with the Immunogenetics of Organ Transplantation Unit of the Città della Salute e della Scienza hospital (Director: Prof. Antonio Amoroso) for the Sanger sequencing validation of genetic variants found by exome sequencing in patients undergoing organ failure and eligible for organ transplantation. Furthermore, she is involved in a project using droplet-digital PCR for the monitoring of organs rejection by measuring circulating donor cell-free DNA in transplanted patients. Lastly, she is contributing to a project aimed at functionally validating the role of mutations found by NGS in patients affected by polycystic kidney disease, by genetically modifying cell line models to reproduce variants of interest.

1. Sorbini M, Togliatto GM, Simonato E, Boffini M, Cappuccio M, Gambella A, **Arruga F**, et al. HLA-DRB1 mismatch-based identification of donor-derived cell free DNA (dd-cfDNA) as a marker of rejection in heart transplant recipients: A single-institution pilot study. J Heart Lung Transplant. 2021 Aug;40(8):794-804. doi: 10.1016/j.healun.2021.05.001. Epub 2021 May 14.
2. Migliorero M, Kalantari S, Bracciamà V, Sorbini M, **Arruga F**, et al. A novel COLEC10 mutation in a child with 3MC syndrome. Eur J Med Genet. 2021 Dec; 64(12):104374. doi: 10.1016/j.ejmg.2021.104374. Epub 2021 Nov 2.

3. Novel molecular players in acute lymphoblastic leukemia (ALL) Ph+. Beside the above-mentioned topics, Dr. Arruga was involved in a project, in collaboration with researchers from the Seragnoli Institute (Bologna), to identify and functionally validate novel players in ALL expressing the Philadelphia chromosome [t(9;22)]. This translocation give rise to the BCR-ABL fusion transcript, mainly found in CML, that represents a driver lesion also in ALL pathogenesis. The final result is an aberrantly activated tyrosine kinase leading to proliferation and cell survival as well as a global perturbation of adhesion and migration. Underneath this behavior there is also a genetic instability leading to the accumulation of mutations on other important effectors. The results of these studies led to the identification of splicing alteration of the *Ikaros* gene, encoding an important transcription factor in lymphoid differentiation, and of the activation-induced cytidine deaminase (AID), that drive hypersomatic immunoglobulin mutations. In the first case, splicing aberration lead to disrupted lymphocyte differentiation; in the second case, AID splicing variants may contribute to genomic instability and favor the accumulation of novel mutations.

1. Iacobucci, I., et al., *Expression of spliced oncogenic Ikaros isoforms in Philadelphia-positive acute lymphoblastic leukemia patients treated with tyrosine kinase inhibitors: implications for a new mechanism of resistance.* Blood, 2008. **112**(9): p. 3847-55.
2. Iacobucci, I., et al., *Identification of different Ikaros cDNA transcripts in Philadelphia-positive adult acute lymphoblastic leukemia by a high-throughput capillary electrophoresis sizing method.* Haematologica, 2008. **93** (12): p. 1814-21.
3. Iacobucci, I., et al., *Different isoforms of the B-cell mutator activation-induced cytidine deaminase are aberrantly expressed in BCR-ABL1-positive acute lymphoblastic leukemia patients.* Leukemia, 2010. **24** (1): p. 66-73.

4. Validation of *WT1* expression as a tool in onco-hematological diseases. One of the research topics has been focused on the analysis of *WT1* expression in myeloid diseases. *WT1* is an oncosuppressor associated with Wilm's Tumor development. Its functional role in leukemogenesis is still unclear, however it was found to be overexpressed in several hematological diseases, at variance with non-tumor cells. The final aim of this study was to validate *WT1* expression in a large cohort of patients and to evaluate the possible application as a disease marker. In the current practice, *WT1* expression is useful to monitor disease evolution and to predict relapse before clinical evidences. In addition, its expression levels can be used to early evaluate drug response, by *in vitro* treatment of primary leukemic cells.

1. Cilloni, D., et al., *Sensitivity to imatinib therapy may be predicted by testing Wilms tumor gene expression and colony growth after a short in vitro incubation.* Cancer, 2004. **101**(5):p.979-88.
2. Cilloni, D., et al., *WT1 transcript amount discriminates secondary or reactive eosinophilia from idiopathic hypereosinophilic syndrome or chronic eosinophilic leukemia.* Leukemia, 2007. **21**(7):1442-50
3. Cilloni, D., et al., *Early prediction of treatment outcome in acute myeloid leukemia by measurement of WT1 transcript levels in peripheral blood samples collected after chemotherapy.* Haematologica, 2008. **93**(6):921-4

5. Role of NF-kB in myeloid neoplasias. In parallel, Dr. Arruga's research has been focused on the functional characterization of the role of NF-kB in myelodysplastic syndromes and in CML, as a downstream effector of BCR-ABL tyrosine kinase. It was shown that NF-kB is frequently aberrantly activated in these diseases, particularly in the case of chemoresistance. This pathway is an important mediator of proliferative and anti-apoptotic signals, two conditions frequently associated with disease progression or relapse. For this reason, NF-kB represents a suitable target for therapeutic approaches in combination with conventional treatment options, to overcome resistance.

1. Cilloni, D., et al., *The NF-kB pathway blockade by the IKK inhibitor PS1145 can overcome Imatinib resistance*. Leukemia, 2006. **20**(1):p.61-7.
2. Morotti, A., et al., *NF-kB inhibition as a strategy to enhance etoposide-induced apoptosis in K562 cell line*. Am J Hematol, 2006. **81**(12):938-45
3. Morotti, A., et al., *CD7/CD56-positive acute myeloid leukemias are characterized by constitutive phosphorylation of the NF-kB subunit p65 at Ser536*. Leukemia, 2007. **21**(6):1305-6.
4. Messa, E., et al., *Deferasirox is a powerful NF-kappaB inhibitor in myelodysplastic cells and in leukemia cell lines acting independently from cell iron deprivation by chelation and reactive oxygen species scavenging.* Haematologica, 2010. **95**(8):p.1308-16.

Major collaborations

1. Gianluca Gaidano, Full Professor in Hematology, and Davide Rossi, Dept. Of Translational Medicine, University of Eastern Piedmont "Amedeo Avogadro" (Novara, Italy) for the identification and functional characterization of novel genetic lesions in onco-hematological diseases.
2. Alan G. Ramsay, Associate Professor in Lymphoma Biology, at the King's College (London UK) for the study of the architecture of CLL microenvironment
3. Francesco Forconi, Professor of Hematology, and Graham Packham, Professor of Molecular Oncology, at the University of Southampton (Southampton, UK), for the analysis of the impact of *NOTCH1* mutations on global mRNA translation.
4. Marta Coscia, Division of Hematology, Città della Salute di Torino Hospital; Luca Laurenti, Hematolgy Insitute, Università Cattolica del Sacro Cuore, Roma; and Giovanni D'Arena, Dept. Of Onco-Hematology, IRCCS-Centro di Riferimento Oncologico della Basilicata, Rionero in Vulture, who provide samples and primary leukemic cells.
5. Richard R. Furman, Associate Professor of Medicine, Director of the CLL Research Center at Weill Cornell Medicine (New York, USA), and John Allan, who provide primary leukemic and RS samples.
6. Salvatore Oliviero, Full Professor of Molecular Biology, University of Turin, and Head of the Epigenetics Unit at the Italian Institute for Genomic Medicine, and Matteo Cereda for the analysis of the genetic and genomic profile of sarcoma samples and derived models.
7. Roberto Piva, Dept. Of Molecular Biotechnology and Health Sciences, University of Turin, for the generations of constructs carrying recurrently mutated genes important in the pathogenesis of lymphoproliferative diseases.
8. Valter Gattei, Head of Experimental and Clinical Onco-Hematology Unit, Michele Dal Bo and Federico Pozzo, Centro di Riferimento Oncologico, I.R.C.C.S., Aviano (PN, Italy) for the study of the role of NOTCH1 and its mutations in the development of resistance to monoclonal anti-CD20 antibodies in CLL (Rituximab, Ofatumumab).
9. Nadia Felli, researcher at the National Institute of Health, Rome, for drug library screening on sarcoma models.
10. Josée Golay, Cellular Therapy Center "G. Lanzani", ASST Papa Giovanni XXIII, Bergamo, for the evaluation of the *in vitro* responses of *NOTCH1*–mutated leukemic cells to monoclonal anti-CD20 antibodies.
11. Giovanni Martinelli, Associate Professor of Hematology, and Ilaria Iacobucci, Institute of Hematology and Medical Oncology "Seragnoli", University of Bologna, for the study of molecular alterations in B-ALL.

Research Support

- Gigi Ghirotti Foundation; 2009. "Allotransplantation of Haematopoietical Stem Cells in oncohematology: Total Lymphoid Irradiation as new conditioning strategy". Role: PI, fellowship.

- Nippon Inc.; 2010-2011. "Evaluation of ROS Inhibitors in Chronic Myelomonocytic Leukemia". Role: PI, fellowship.

- "Young Investigator Programme", Umberto Veronesi Foundation; 2013. "Functional analysis of the role of NOTCH1 as a novel genetic lesion characterizing chronic lymphocytic leukemia patients with aggressive disease." Role: PI, fellowship.

- SIES-Beat Leukemia, Italian Society of Experimental Hematology; 2015. "Ruolo funzionale delle mutazione del gene *NOTCH1* nella leucemia linfatica cronica". Role: PI, fellowship.

- Italian Ministry of Health, Young Investigator Grant 2010: “New genetic lesions characterizing high risk chronic lymphocytic leukemia: clinical and functional implications”. Role: participant.
- Italian Ministry of Education, Futuro in Ricerca 2012: “Identification and functional characterization of genomic lesions in lymphoid malignancies”. Role: participant.
- Cariplo Foundation Grant 2012: “Deciphering the molecular basis of splenic marginal zone lymphoma by whole exome sequencing and functional genomics”. Role: participant.
- Human Genetics Foundation Investigator Grant 2011-2013: “Genetic regulation, surface receptors and soluble molecules in the control of tumor/host interactions”. Role: participant.
- Italian Ministry of Health Young Investigator Grant 2011: “Pleiotropic transcriptional control mechanisms of CD49d expression in trisomy 12 chronic lymphocytic leukemia: implications for novel therapeutic approaches”. Role: participant.
- Italian Association for Cancer Research, Investigator Grant 2015: “Understanding tumor-host interactions and therapy resistance in chronic lymphocytic leukemia: moving up a NOTCH?” Role: participant in the PI’s Unit
- Fondazione Italiana di Ematologia ed Oncologica Pediatrica ONLUS (FIEOP): “Functional genomics applied to pediatric cancer: from mutations to functions and therapies”. Role: participant
- Institutional funds from Italian Institute for Genomic Medicine: “Functional genomics applied to recurrently mutated genes in Chronic Lymphocytic Leukemia and Splenic Marginal Zone Lymphoma” Role: participant
- Italian Association for Cancer Research, Investigator Grant 2019: “A ménage à trois involving the B cell receptor, NOTCH1 and NAMPT: therapeutic implications for CLL and RS patients” Role: participant

Career breaks

- Enrico (Sept 12, 2020)
- Bianca (Sept 16, 2023)

Peer-reviewed publications

Publications with Impact Factor (2004-2021): 52
Total IF (JCR 2022): 455.1
Mean IF: 8.752
H-Index (Scopus): 25

1. Cilloni D, Carturan S, Gottardi E, Messa F, Fava M, Defilippi I, **Arruga F**, Saglio G. Usefulness of the quantitative assessment of PRV-1 gene expression for diagnosis of polycytemia vera and essential thrombocythemia patients. Blood 2004, 103 (6): 2428-2429
2. Cilloni D, Messa F, Gottardi E, Fava M, **Arruga F**, Defilippi I, Carturan S, Messa E, Morotti A, Giugliano E, Rege-Cambrin G, Alberti D, Baccarani M, Saglio G. Sensitivity to Imatinib therapy may be predicted by testing Wilms Tumor gene expression and colony growth after a short “in vitro” incubation. Cancer 2004, 101 (5): 979-988
3. Cilloni D, Messa F, Carturan S, **Arruga F**, Defilippi I, Messa E, Gottardi E, Saglio G. Myelodysplastic syndromes. Ann. N.Y. Acad. Sci. 2004, 1028: 400-408
4. Saglio G, Carturan S, Grillo S, Capella S, **Arruga F**, Defilippi I, Rosso V, Rauco M, Marina Liberati A, Cilloni D. WT1 overexpression: a clinically useful marker in acute and chronic myeloid leukemias. Hematology 2005; 10 Suppl1: 76-8
5. Cilloni D, Messa F, **Arruga F**, Defilippi I, Morotti A, Messa E, Carturan S, Giugliano E, Pautasso M, Bracco E, Rosso V, Sen A, Martinelli G, Baccarani M, Saglio G. The NF-kB pathway blockade by the IKK inhibitor PS1145 can overcome Imatinib resistance. Leukemia 2006, 20(1): 61-67
6. Morotti A, Cilloni D, Messa F, **Arruga F**, Defilippi I, Carturan S, Catalano R, Rosso V, Chiarenza A, Pilatrino C, Guerrasio A, Taulli R, Bracco E, Pautasso M, Baraban D, Gottardi E, Saglio G. Valproate enhances imatinib-induced growth arrest and apoptosis in chronic myeloid leukemia cells. Cancer 2006,106(5): 1188-1196
7. Morotti A, Cilloni D, Pautasso M, Messa F, **Arruga F**, Defilippi I, Carturan S, Catalano R, Rosso V, Chiarenza A, Taulli R, Bracco E, Rege-Cambrin G, Gottardi E, Saglio G. NF-kB inhibition as a strategy to enhance etoposide-induced apoptosis in K562 cell line. Am. J. Hematol. 2006, 81(12):938-45
8. Cilloni D,Messa E, Messa F, Carturan S, Defilippi I, **Arruga F**, Rosso V, Catalano R, Bracco E, Nicoli P, Saglio G. Genetic abnormalities as targets for molecular therapies in myelodysplastic syndromes. Ann. N.Y. Acad. Sci. 2006, 1089: 411-423
9. Morotti A, Parvis G, Cilloni D, Familiari U, Pautasso M, Bosa M, Messa F, **Arruga F**, Defilippi I, Catalano R, Rosso V, Carturan S, Bracco E, Guerrasio A, Saglio G. CD7/CD56-positive acute myeloid leukemias are characterized by costitutive phosphorylation of the NF-kB subunit p65 at ser536. Leukemia 2007, 21(6): 1305-6
10. Cilloni D, Messa F, Martinelli G, Gottardi E, **Arruga F**, Defilippi I, Carturan S, Messa E, Fava M, Giugliano E, Rosso V, Catalano R, Merante S, Nicoli P, Rondoni M, Ottaviani E, Soverini S, Tiribelli M, Pane F, Baccarani M, Saglio G. WT1 transcript amount discriminates secondary or reactive eosinophilia from idiopathic hypereosinophilic sindrome or chronic eosinophilic leukemia. Leukemia 2007, 21(7): 1442-50
11. Cilloni D, Messa F, **Arruga F**, Defilippi I, Gottardi E, Fava M, Carturan S, Catalano R, Bracco E, Messa E, Nicoli P, Diverio D, Sanz MA, Martinelli G, Lo-Coco F, Saglio G. Early prediction of treatment outcome in acute myeloid leukemia by measurement of WT1 transcript levels in peripheral blood samples collected after chemotherapy. Haematologica 2008 93(6).
12. Iacobucci I, Lonetti A, Messa F, Cilloni D, **Arruga F**, Ottaviani E, Paolini S, Papayannidis C, Piccaluga PP, Giannoulia P, Soverini S, Amabile M, Poerio A, Saglio G, Pane F, Berton G, Baruzzi A, Vitale A, Chiaretti S, Perini G, Foà R, Baccarani M, Martinelli G. Expression of spliced oncogenic

Ikaros isoforms in Philadelphia-positive acute lymphoblastic leukemia patients treated with tyrosine kinase inhibitors: implications for a new mechanism of resistance. Blood. 2008;112(9)

13. Nicoli P, Defilippi I, Carturan S, Roetto A, Messa F, **Arruga F**, Messa E, Rotolo A, Iacobucci I, Bracco E, Saglio G, Cilloni D. Detection of humoral immune responses against WT1 antigen in patients affected by different hematological malignancies. Acta Haematol. 2008;120(1)
14. Messa E, Cilloni D, Messa F, **Arruga F**, Roetto A, Saglio G. Deferasirox treatment improved the hemoglobin level and decreased transfusion requirements in four patients with the myelodysplastic syndrome and primary myelofibrosis. Acta Haematol. 2008;120(2)
15. Iacobucci I, Lonetti A, Cilloni D, Messa F, Ferrari A, Zuntini R, Ferrari S, Ottaviani E, **Arruga F**, Paolini S, Papayannidis C, Piccaluga PP, Soverini S, Saglio G, Pane F, Baruzzi A, Vignetti M, Berton G, Vitale A, Chiaretti S, Muschen M, Foà R, Baccarani M, Martinelli G. Identification of different Ikaros cDNA transcripts in Philadelphia-positive adult acute lymphoblastic leukemia by a high-throughput capillary electrophoresis sizing method. Haematologica. 2008 Dec;93(12)
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Congress books:

1. Cilloni D, Messa F, Carturan S, Defilippi I, **Arruga F**, Rosso V, Chiarenza A, Catalano R, Messa E, Morotti A, Saglio G: “Marcatori universali di malattia minima residua in oncoematologia”. 40th Congress of the Italian Society of Hematology, Bergamo July, 03-06 2005

Meetings (oral and poster presentations)

1. Italian Society of Experimental Hematology (SIES) VIII annual meeting: “The in vitro treatment with NF-kB inhibitors is able to overcome imatinib resistance in cell lines and CML resistant patients”. September 14-16, 2004, Pavia, Italy. (**oral presentation**)
2. Italian Society of Hematology (SIE) 40° annual meeting. “Gene expression analysis based on real time PCR of 95 genes coding for tyrosyne kinases in Ph negative chronic myeloproliferative disorders”. July 3-6, 2005, Bergamo, Italy. (**poster**)
3. Italian Society of Hematology (SIE) 41° annual meeting. “EPHA3 is constitutively activated in chronic mieloproliferative disorders and can be targeted by Dasatinib or by monoclonal antibodies”. October 14-17, 2007, Bologna, Italy. (**oral presentation**)
4. American Association for Cancer Research (AACR) 99° annual meeting: “FoxO transcription factor is delocalized in CML patients by Bcr-Abl induced PI3K/AKT activation and IKK pathway and can be reactivated by Imatinib treatment”. April 12-16, 2008, San Diego, California, USA, (**poster**).
5. European Hematology Association (EHA) 13° annual meeting: “Pattern of Meningioma 1 gene (MN1) expression in different genetic subset of acute and chronic myeloid leukaemias and its potential use as a marker for minimal residual disease detection”. June 12-15, 2008, Copenhagen, Denmark. (**poster**). **Travel grant awarded**.
6. Italian Society of Experimental Hematology (SIES) X annual meeting: “Pattern of Meningioma1 gene expression in different genetic subsets of acute and chronic myeloid leukemia and its potential use as a marker for minimal residual disease detection”. September 24-26, 2008, Bari, Italy. (**oral presentation**).
7. Italian Society of Experimental Hematology (SIES) X annual meeting: “EphA3 kinase is constitutively activated in Chronic Myeloid Leykemia during accelerated and blast crisis and can be targeted by Dasatinib or by Monoclonal antibodies”. September 24-26, 2008, Bari, Italy. (**oral presentation**).
8. American Association for Cancer Research (AACR) 100° annual meeting:” EphA3 is abnormally expressed in chronic myeloprolipherative disorders and could represent a new molecular target”. April 18-22, 2009, Denver, Colorado, USA. (**poster**).
9. Italian Society of Hematology (SIE) 42° annual meeting. “Gene expression analysis of the kinome and the phosphatome in Chronic Myeloproliferative Diseases”. October 18-21, 2009, Milan, Italy. (**poster**).
10. Italian Society of Experimental Hematology (SIES) XI annual meeting. “Identification of Rab5 as a gene involved in Chronic Myeloid Leukemia (CML) progression and TKi resistance”. October 6-8, 2010, Turin, Italy. (**poster**).
11. American Society of Hematology (ASH) 55° Annual meeting. “Functional Effects Of NOTCH1 Mutations In Chronic Lymphocytic Leukemia Patients”. December 7-10, 2013, New Orleans, Louisiana, USA. (**poster**).
12. Italian Society of Experimental Hematology (SIES) XIII annual meeting. “Microenvironmental inputs mediated by the B cell receptor (BCR) stimulates NOTCH1 activity in CLL cells harboring NOTCH1 mutations”. October 15-17, 2014, Rimini, Italy. (**poster**)
13. American Society of Hematology (ASH) 58° Annual meeting. “Mutations in NOTCH1 PEST-domain orchestrate CCL19-driven homing of Chronic Lymphocytic Leukemia cells by modulating the tumor suppressor gene DUSP22”. December 3-6, 2016, San Diego, California, USA. (**oral presentation**).
14. XVII international workshop on Chronic Lymphocytic Leukemia (iwCLL). “Mutations in NOTCH1 PEST domain confers growth advantage to CLL cells”. May 12-15, 2017, New York. (**oral presentation**).
15. American Society of Hematology (ASH) 60° Annual meeting. “NOTCH1 Stabilization by PEST Mutations Enhances IgM-Mediated Activity in Chronic Lymphocytic Leukemia”. December 1-4, 2018, San Diego, California, USA. (**poster presentation**).

16. XVIII international workshop on Chronic Lymphocytic Leukemia (iwCLL). “Functional interplay between the B-cell receptor and NOTCH1 in Chronic Lymphocytic Leukemia”. September 20-23, 2019, Edinburgh, Scotland (**oral presentation**).

Invited seminars:

1. Mutational Course and BMH meeting, May 2009, Madrid, Spain.
2. CML Meeting: Latest Update on CML. September 2009, Turin, Italy
3. Italian Society of Experimental Hematology “Discutiamone Insieme”, “Generazione di un modello genetico basato sulla Drosophila melanogaster per lo studio della leucemia mieloide cronica” November 2009, Florence, Italy
4. VII Brainstorming on CLL. “*NOTCH1* mutations: functional aspects”. February 2014, Aviano (PN), Italy
5. VIII Brainstorming on CLL. “Genome editing approaches to study the functional role of NOTCH1”. February 2015, Aviano (PN), Italy
6. 5° International workshop of Experimental Hematology. “Unraveling the role of NOTCH1 in Chronic Lymphocytic Leukemia”. October 2015, Pisa, Italy
7. IX Brainstorming on CLL. “NOTCH1-regulated molecular circuits in CLL”. February 2016, Aviano (PN), Italy
8. Seminar at the Molecular Biotechnology Center. “NOTCH1 orchestrates the homing of CLL cells by modulating the tumor suppressor gene DUSP22: implications for disease progression”. October 2016, Turin, Italy
9. X Brainstorming on CLL. “Novel insights about NOTCH1 mutations in CLL”. February 2017, Aviano (PN), Italy
10. Seminar at the Molecular Biotechnology Center. “Functional interplay between NOTCH1 and the B cell receptor in Chronic Lymphocytic Leukemia and in Richter’s syndrome”. June 2019, Turin, Italy
11. Seminar at the Molecular Biotechnology Center. “The TIGIT/CD226/CD155 immunomodulatory axis is expressed by chronic lymphocytic leukemia cells and contributes to B-cell anergy”. June 2021, Turin, Italy

Attended Meetings

1. Workshop: Real Time PCR & Genomic Assays. April 2003, Turin, Italy
2. Proteomic Seminar Tour. July 2003, Milan, Italy
3. Italian Society of Experimental Hematology “Discutiamone Insieme”, November 2003, Florence, Italy
4. I° Convegno Biotecnologi- sezione Piemonte. February 2004, Turin, Italy
5. Italian Society of Experimental Hematology (SIES) VIII annual meeting, September 2004, Pavia, Italy
6. 2nd Italian RNA Interference Symposium: in vitro and in vivo models. November 2004, Busto Arsizio, Italy
7. Italian Society of Hematology (SIE) 40° annual meeting, July 2005, Bergamo, Italy
8. Mielodysplastic syndromes. October 2005, Turin, Italy
9. Italian Society of Experimental Hematology (SIES) IX annual meeting, September 2006, Naples, Italy
10. Italian Society of Hematology (SIE) 41° annual meeting, October 2007, Bologna, Italy
11. 99th American Association for Cancer Research (AACR) Annual Meeting, April 2008, San Diego, California, USA
12. 13th Congress of European Hematology Association (EHA), June 2008, Copenhagen, Denmark
13. Italian Society of Experimental Hematology (SIES) X annual meeting, September 2008, Bari, Italy
14. 100th American Association for Cancer Research (AACR) Annual Meeting, April 2009, Denver, Colorado, USA

15. Italian Society of Hematology (SIE) 42° annual meeting, October 2009, Milan, Italy
16. Italian Society of Experimental Hematology (SIES) XI annual meeting, October 2010, Turin, Italy
17. Italian Society of Experimental Hematology “Discutiamone Insieme”, November 2010, Florence, Italy
18. 14° Convegno di Patologia Immune e Malattie Orfane, January 2011, Turin, Italy
19. 15° Convegno di Patologia Immune e Malattie Orfane, January 2012, Turin, Italy
20. Workshop: Targeted Genome Editing Using Zinc Finger Nuclease, November 2012, Heidelberg, Germany
21. 16° Convegno di Patologia Immune e Malattie Orfane, January 2013, Turin, Italy
22. XXVI Symposium IACRLRD, September 2013, Turin, Italy.
23. 4° International workshop of Experimental Hematology, November 2013, Bologna, Italy
24. 55th American Society of Hematology (ASH) meeting, December 2013, New Orleans, LA, USA.
25. 17° Convegno di Patologia Immune e Malattie Orfane, January 2014, Turin, Italy
26. VII Brainstorming on CLL, February 2014, Aviano (PN), Italy
27. Italian Society of Experimental Hematology (SIES) XIII annual meeting. October 2014, Rimini, Italy
28. 56th American Society of Hematology (ASH) meeting, December 2014, San Francisco, CA, USA
29. VIII Brainstorming on CLL, February 2015, Aviano (PN), Italy
30. 5° International workshop of Experimental Hematology, October 2015, Pisa, Italy
31. IX Brainstorming on CLL, February 2016, Aviano (PN), Italy
32. 58th American Society of Hematology (ASH) meeting. December 2016, San Diego, California, USA.
33. XVII iwCLL. May 2017, New York, NY, USA.
34. International Symposium on Chronic Lymphocytic Leukemia: Advances in pathogenesis and treatment. March 8-10 2018, Venice, Italy.
35. Basic Next Generation Sequencing (NGS) procedures. March 12-13 2018, Busto Arsizio (VA), Italy.
36. XVIII iwCLL. September 20-23, 2019, Edinburgh, Scotland
37. XXVIIIAIBT (Associazione Italiana di Immunogenetica e Biologia dei Trapianti) Annual Meeting, October 20-22 2022, Parma Italy

Turin, 24/01/2025