CURRICULUM VITÆ
DR. IAN MARC BONAPACE

PERSONAL DATA

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STUDIES

1975-82: "Laurea Universitaria" (Master Degree) in Biological Sciences - 27 January 1982 - score 110/110 - University of Naples, Naples - Italy;

1987-91: Ph.D. in Cellular and Molecular Biology and Pathology - II School of Medicine - "*Federico II University*" - Naples - Italy.

POSITIONS

Current Position

2001 to present **"**Assistant Professor" at the Dept. of Biotechnology and Life Sciences (DBSV) of the University of Insubria, Varese - ITALY.

Former Positions

2005-08 '*Guest Scientist*' at the *Département de Biologie et Génomique Structurales – IGBMC (Institut de Génétique et de Biologie Moléculaire et Cellulaire*) – Strasbourg, France

1997-01 "Postdoctoral Associated" at the European Institute of Oncology – Milan - Italy

1993-94 "Postdoctoral Associated" at the Institute of General Pathology and Oncology of the 2nd University of Naples - Italy

1982 "Postdoctoral Associated" at the "Section of Molecular and Cellular Biology" - Cornell University – Ithaca, NY - USA.

TEACHING ACTIVITY
(Names of the courses per Academic year)

LAST 5 YEARS

2016-17 **A)** "*Pathology"*, Master Degree Program in "*Biomedical Sciences*" at the Dep. of Biotechnology and Lie Sciences, University of Insubria, Varese – ITALY (http://www4.uninsubria.it/on-line/home/cdl-triennale/articolo12244.html).

B) "*Cellular and Molecular Oncology*", Master Degree Program in "*Biomedical Sciences*" at the Dep. of Biotechnology and Lie Sciences, University of Insubria, Varese – ITALY (http://www4.uninsubria.it/on-line/home/cdl-triennale/articolo10015.html).

The Master's program in Biomedical Sciences of the University of Insubria is connected to the Master Degree Programme (MSc) in "*Biomedical Sciences*" at the University of Bonn-Rhein-Sieg of Applied Sciences (Germany) for a Double Degree Program (https://www.h-brs.de/en/anna/biomedical-sciences-msc).

2015-16 **A)** "*Pathology*" Master Degree Program in "*Biomedical Sciences*" at the Dep. of Biotechnology and Lie Sciences, University of Insubria, Varese – ITALY
B) "*Cellular and Molecular Oncology*", Master Degree Program in "*Biomedical Sciences*" at the Dep. of Biotechnology and Lie Sciences, University of Insubria, Varese - ITALY

2014-15 "*Pathology"*, Master Degree Program in "*Applied Biology for Biomedical Research*", at the Dep. of Theoretical and Applied Sciences, University of Insubria, Varese - ITALY

2013-14 "*Pathology"*, Master Degree Program in "*Applied Biology for Biomedical Research*", at the Dep. of Theoretical and Applied Sciences, University of Insubria, Varese - ITALY

2012-13 "*Pathology"*, Master Degree Program in "*Applied Biology for Biomedical Research*", at the Dep. of Theoretical and Applied Sciences, University of Insubria, Varese - ITALY

PREVIOUS TEACHING ACTIVITY

2002-12 "*Pathology"*, Degree Program in "*Biologia Sanitaria*", at the Faculty of Natural Sciences, University of Insubria, Varese – ITALY

2003-06 "*Immunology*" at the Faculty of Natural Sciences, University of Insubria – Varese – Italy.

2004-10 "*Biology of neoplastic transformation*" within the course "*Biology and Pharmacology of neoplastic transformation*" at the Faculty of Natural Sciences, University of Insubria – Varese – Italy.

FUNDING as a PI

2013-18 **Italian Flagship Program - Epigen**
To investigate the epigenetic basis of the apparent paradox of the co-existence of global hypo and site-specific hyper-methylation of DNA during cancer progression. 600.000 €

2010-13 **Italian Association for Cancer Research (AIRC) - Italia.**
Role of UHRF1/2, essential for DNA methylation, in epigenetic gene silencing of tumour-suppressors. 250.000 €

2009-10 **Programmi di ricerca scientifica di rilevante interesse nazionale (PRIN) – Italia (Scientific programs of national interest).**
La cromatina come substrato d'interrelazione nella dinamica tra replicazione del DNA ed espressione genica (Chromatin as a substrate in the dynamic interrelation between DNA replication and gene expression). 119.000 €

2007-09 **Italian Association for Cancer Research (AIRC) - Italia.**
The role of Np95 in pericentromeric heterochromatin formation and stability. 150.000 €

2005-08 **Programmi per l'incentivazione del processo d'internazionalizzazione del sistema universitario - (D.M. 5 agosto 2004 n. 262 - ART. 23) collaborazioni interuniversitarie internazionali. Cooperazione Italo-Francese (**Programs to encourage the process of internationalization of the national university system - International interuniversity Collaborations. Italo-French cooperation)**.**
Il ruolo di Np95/ICBP90, una proteina essenziale alla proliferazione cellulare, nella replicazione eterocromatica, la soluzione della sua struttura 3D e l'individuazione dei suoi domini funzionali (The role of NP95 / ICBP90, a protein essential to cell proliferation, in heterochromatic replication, the solution of its 3D structure and the identification of its functional domains). 24.000 €

2005-07 **Rett Syndrome Research Foundation (RSRF) - USA.**
The isolation of protein complexes involving MeCP2 and Np95: Understanding their roles in the structural organisation of heterochromatin. 100.000 US dollars

2003-06 **Italian Association for Cancer Research (AIRC) - Italia.**
The role of Np95, an essential protein for S phase entry, on chromatin modifications during DNA replication. 105.000 €

FUNDING as Research Unit

2013-15 **Italian Association for Cancer Research (AIRC) - Italia.** Carcinogen–induced Epigenetic Switching in Human Retroelements: a Mechanistic Study on Benzene Effects. 90.000€

2012-14 **Directorate general political and affairs of the foreign affaire, Italy.** Quantitative assessment of gene-environment interaction in cultured nasal epithelial cells exposed to fine air-pollution using an in vitro simulator of nasal breathing. 120.000 €

2005-07 **Fondazione Cariplo – Progetto Nobel. Italia** Genetic and Epigenetic control of genome stability. 30.000 Euro

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PUBLICATIONS

1. Christian Pistore, Elisa Giannoni, Tommaso Colangelo Francesca Rizzo, Elena Magnani, Livio Muccillo, Giorgio Giurato, Monica Mancini, Samantha Rizzo, Mila Riccardi, Nora Sahnane, Del Vescovo Valerio, Kamal Kishore, Martina Mandruzzato, Filippo Macchi, Mattia Pelizzola, Denti Michela A., Daniela Furlan, Alessandro Weisz, Vittorio Colantuoni, Paola Chiarugi and **Ian Marc Bonapace***. *DNA methylation variations are required for epithelial-to-mesenchymal transition induced by cancer-associated fibroblasts in prostate cancer cells*. Oncogene. 2017, In press
2. Mancini M, Mandruzzato M, Garzia AC, Sahnane N, Magnani E, Macchi F, Oulad-Abdelghani M, Oudet P, Bollati V, Fustinoni S, Furlan D, **Bonapace IM**. *In vitro hydroquinone-induced instauration of histone bivalent mark on human retroelements (LINE-1) in HL60 cells*. Toxicol In Vitro. 2016 Dec 13;40:1-10. doi: 10.1016/j.tiv.2016.12.007
3. Ramesh V, Bayam E, Cernilogar FM, **Bonapace IM**, Schulze M, Riemenschneider MJ, Schotta G, Götz M. *Loss of Uhrf1 in neural stem cells leads to activation of retroviral elements and delayed neurodegeneration*. Genes Dev. 2016 Oct 1;30(19):2199-2212.
4. Qin W, Wolf P, Liu N, Link S, Smets M, La Mastra F, Forné I, Pichler G, Hörl D, Fellinger K, Spada F, **Bonapace IM**, Imhof A, Harz H, Leonhardt H. *DNA methylation requires a DNMT1 ubiquitin interacting motif (UIM) and histone ubiquitination*. Cell Res. 2015 Aug;25(8):911-29
5. De Vos M, El Ramy R, Quénet D, Wolf P, Spada F, Magroun N, Babbio F, Schreiber V, Leonhardt H, **Bonapace IM**, Dantzer F. *Poly(ADP-ribose) polymerase 1 (PARP1) associates with E3 ubiquitin-protein ligase UHRF1 and modulates UHRF1 biological functions*. J Biol Chem. 2014 Jun 6;289(23):16223-38
6. De Lerma Barbaro A, Perletti G, **Bonapace IM**, Monti E. *Inflammatory cues acting on the adult intestinal stem cells and the early onset of cancer*. Int J Oncol. 2014 Sep;45(3):959-68
7. Morano, T. Angrisano, S. Bartollino, G. Russo, R. Landi, B. Lee, C. Zucchegna, G. Tell, F. Babbio, C. Pistore, **IM Bonapace**, M.T. Muller, L. Chiariotti, M.E. Gottesman, A. Porcellini and E.V. Avvedimento. *Targeted DNA methylation by homology-directed repair in mammalian cells. Transcription reshapes methylation on the repaired gene.* Nucleic Acids Research, 2013, 1–18
8. F. Babbio, I. Castiglioni, C. Cassina, C. Pistore, M.B. Gariboldi, G. Badaracco, E. Monti, **IM Bonapace***. *Knockdown of Methyl CpG-binding protein 2 (MeCP2) causes alterations in cell proliferation and nuclear lamins expression in mammalian cells*. BMC, 2012 Jul 11;13(1):19
9. Massimo Pancione, Alessandra Fucci, Federica Babbio, Christian Pistore, Ilaria Castiglioni, Lucia Altucci, **Ian Marc Bonapace***, Vittorio Colantuoni*. *The coordinated activity of UHRF1 in the epigenetic silencing of PPARG plays an important role in colorectal cancer pathogenesis.* Oncogene. 2012 (*Corresponding author)
10. Federica Babbio, Christian Pistore, Laura Curti, Ilaria Castiglioni, Kunderfranco Paolo, Laurent Brino, Pierre Oudet, Roland Seiler, George N. Thalman, Manuela Sarti, Sandra Pinton, Maurizia Mello-Grand, Giovanna Chiorino, Carlo V. Catapano, Giuseppina M. Carbone and **Ian Marc Bonapace*** *The SRA protein UHRF1 promotes epigenetic cross-talks and is involved in prostate cancer progression*. Oncogene. 2012 Feb 13. doi: 10.1038/onc.2011.641.
11. Rottach, A., Frauer, C., Pichler, G., **Bonapace, I.M**., Spada, F., and Leonhardt, H. (2010) *The multi-domain protein Np95 connects DNA methylation and histone modification*. Nucleic Acids Res *38*, 1796-1804.

12. Meilinger, D., Fellinger, K., Bultmann, S., Rothbauer, U., **Bonapace, I.M**., Klinkert, W.E., Spada, F., and Leonhardt, H. (2009). *Np95 interacts with de novo DNA methyltransferases, Dnmt3a and Dnmt3b, and mediates epigenetic silencing of the viral CMV promoter in embryonic stem cells*. EMBO Rep *10*, 1259-1264.
13. Guarda, A., Bolognese, F., **Bonapace, I.M**.*, and Badaracco, G.* (2009). *Interaction between the inner nuclear membrane lamin B receptor and the heterochromatic methyl binding protein*, MeCP2. Exp Cell Res *315*, 1895-1903.
14. Papait, R., Monti, E., and **Bonapace, I.M**.* (2009). Novel approaches on epigenetics. Curr Opin Drug Discov Devel *12*, 264-275.
15. Papait, R., Pistore, C., Grazini, U., Babbio, F., Cogliati, S., Pecoraro, D., Brino, L., Morand, A.L., Dechampesme, A.M., Spada, F., Leonhardt, H., McBlane, F., Oudet, P., and **Bonapace, I.M**.* (2008a). The PHD domain of Np95 (mUHRF1) is involved in large-scale reorganization of pericentromeric heterochromatin. Mol Biol Cell *19*, 3554-3563.
16. Papait, R., Pistore, C., Negri, D., Pecoraro, D., Cantarini, L., and **Bonapace, I.M**.* (2007). *Np95 is implicated in pericentromeric heterochromatin replication and in major satellite silencing*. Mol Biol Cell *18*, 1098-1106.
17. Nicassio, F., Bianchi, F., Capra, M., Vecchi, M., Confalonieri, S., Bianchi, M., Pajalunga, D., Crescenzi, M., **Bonapace, I.M**.*, and Di Fiore, P.P.* (2005). *A cancer-specific transcriptional signature in human neoplasia*. J Clin Invest *115*, 3015-3025.
18. Citterio, E., Papait, R., Nicassio, F., Vecchi, M., Gomiero, P., Mantovani, R., Di Fiore, P.P., and **Bonapace, I.M**.* (2004). *Np95 is a histone-binding protein endowed with ubiquitin ligase activity*. Mol Cell Biol *24*, 2526-2535.
19. **Bonapace, I.M**., Latella, L., Papait, R., Nicassio, F., Sacco, A., Muto, M., Crescenzi, M., and Di Fiore, P.P. (2002). *Np95 is regulated by E1A during mitotic reactivation of terminally differentiated cells and is essential for S phase entry*. J Cell Biol *157*, 909-914.
20. **Bonapace, I.M**., Addeo, R., Altucci, L., Cicatiello, L., Bifulco, M., Laezza, C., Salzano, S., Sica, V., Bresciani, F., and Weisz, A. (1996). *17β-Estradiol overcomes a G1 block induced by HMG-CoA reductase inhibitors and fosters cell cycle progression without inducing ERK-1 and -2 MAP kinases activation*. Oncogene *12*, 753-763.
21. Addeo, R., Altucci, L., Battista, T., **Bonapace, I.M**., Cancemi, M., Cicatiello, L., Germano, D., Pacilio, C., Salzano, S., Bresciani, F., and Weisz, A. (1996). *Stimulation of human breast cancer MCF-7 cells with estrogen prevents cell cycle arrest by HMG-CoA reductase inhibitors*. Biochem Biophys Res Commun *220*, 864-870.
22. Gallo, A.^, Benusiglio, E.^, **Bonapace, I.M**^., Feliciello, A., Cassano, S., Garbi, C., Musti, A.M., Gottesman, M.E., and Avvedimento, E.V. (1992). v-ras and protein kinase C dedifferentiate thyroid cells by down-regulating nuclear cAMP-dependent protein kinase A. Genes Dev *6*, 1621-1630. ^These authors have equally contributed to this paper
23. **Bonapace, I.M**., Sanchez, M., Obici, S., Gallo, A., Garofalo, S., Gentile, R., Cocozza, S., and Avvedimento, E.V. (1990). Extinction and activation of the thyroglobulin promoter in hybrids of differentiated and transformed thyroid cells. Mol Cell Biol *10*, 1033-1040.
24. Avvedimento, V.E., Musti, A., Fusco, A., **Bonapace, I.M**., and Di Lauro, R. (1988). Neoplastic transformation inactivates specific trans-acting factor(s) required for the expression of the thyroglobulin gene. Proc Natl Acad Sci U S A *85*, 1744-1748
25. AVVEDIMENTO, VE; DIFIORE, PP; BONAPACE, IM. (1986) *Molecular mechanisms of the interference between viral transformation and the thyroglobulin expression*. Annals de endocrinologie Volume: 47 Issue: 6 Pages: 78-78.
26. Polito L.C., Furia M., Cavaliere D. and **Bonapace I.M.** (1980) Analisys of rDNA magnification process in mei9^{bb} mutant of *Drosophila melanogaster*. Genetics Supplement

MANUSCRIPTS SUBMITTED

MANUSCRIPTS IN PREPARATION

1. Magnani E, Babbio F, Macchi F, Curti L, Carbone G, Catapano C, and **Bonapace IM*** *UHRF1 represses E-cadherin during the epithelial-mesenchimal transition by regulating the transcription of an anti-sense long ncRNA from the promoter of CDH1.*

2. Magnani E, Babbio F, Mancini M, Monti E, and **Bonapace IM*** *The coexistence of DNA hypo- and hyper-methylation in cancer: two faces of the same coin ?* Review
3. Ferrandi A, Bolognese F, Babbio F, Pistore C, Badaracco G, Barbieri P, and **Bonapace IM*** *The Deinococcus r. SRA containing protein SHP is required for correct response to DNA double strand break damage*.

CURRENT SCIENTIFIC INTERESTS

Since many years, my scientific interest is focused on the role of UHRF1 (*alias* Np95 or ICBP90) and its close structural homologue UHRF2, in DNA methylation and in the epigenetic regulation of onco-suppressors during tumour progression. The SRA (Set and RING-finger-associated domain) domain containing protein UHRF1 is master epigenetic regulator and transcriptional repressor essential to maintain global and local DNA methylation and required for the epithelial to mesenchimal transition (EMT) in tumours,

During the last 5 years, my research group has shown that UHRF1 is deeply implicated in the control of epigenetic modifications during DNA replication and in EMT in prostate and colon cancers. Our current scientific hypothesis is that UHRF1 and UHRF2 are implicated in (at least) prostate and colon cancer pathogenesis and are critical players within the complexes that act at the interplay between DNA replication, chromatin remodelling and transcription to regulate epigenetic modifications and transcriptional silencing of tumour suppressors and other genes during prostate and colon tumour progression.

Three main lines of research are carried out in my laboratory of General Pathology at the University of Insubria in collaboration with three laboratories.

1) DNA hypo/hypermethylation in cancer: decrypt the epigenetic basis of the paradox
2) The involvement of UHRF1 and UHRF2 in prostate and colon cancer pathogenesis
3) The role of UHRF1 and UHRF2 in the epigenetic control of tumour suppressor genes during tumour progression and epithelial to mesenchymal transition

1) DNA hypo/hypermethylation in cancer: decrypt the epigenetic basis of the paradox

An early global DNA hypo- and a later site-specific hyper-methylation are hallmarks of the tumorigenic process as they often co-exist during progression, but answers to this apparent paradox have not been found yet. As the vast majority of the genome is composed of non-coding regions and up to 38% of the human genome consists of Alu and LINE-1 methylated repetitive sequences, DNA hypomethylation affects mainly the genomic regions that contain these non-coding and transposable elements, although demethylation of oncogenes is also observed. Demethylation of these sequences is a source of strong chromosomal instability and an important cause of insertional mutagenesis of genes critical for preventing (tumour-suppressors, both coding and non-coding) or driving malignant transformation (oncogenes, both coding and non-coding). DNA hypermethylation, instead, mainly affects the promoter regions of tumour suppressor genes, which leads to unchecked proliferation, reduced response to apoptotic stimuli and metastasis. The co-existence of DNA hypo- and hyper-methylation in tumours implies that factors required for DNA methylation are still active in transformed cells and that DNA demethylation cannot be simply caused by loss of DNMTs activity. This is exemplified by the finding that UHRF1, a required component of the methylating complexes, and DNMTs are overexpressed in many tumours in which genome-wide hypo- and hyper-methylation of tumour-suppressors genes coexist.

DNA methylation patterns are better correlated with histone modification patterns rather than with the underlying genome sequence context in all genomic regions, including repetitive sequences, suggesting that these two types of epigenetic modifications are tightly co-regulated. Whether DNA methylation drives histone methylation or *vice-versa* has not been definitely established, although binding of UHRF1 to H3K9me3 is required for DNA methylation. UHRF1 (*ubiquitin-like with PHD and ring finger domains 1*) is a member of a subfamily of RING-finger type E3 ubiquitin ligases required for DNA methylation, as it controls methylation of virtually all CpG sequences in the genome by recruiting all three DNMTs to sites of methylation. Altogether, genome-wide methylation sites almost totally coincide with UHRF1/DNMTs binding sites. Interestingly,

UHRF1/DNMTs are found overexpressed in many tumours where hypo- and hyper-methylation co-exist.
We assume that genes that can be methylated might have a common epigenetic signature that must be red by UHRF1 in order to recruit the methylating complexes. We hypothesise that, following DNA demethylation occurred for whatever reason, UHRF1 and the complexes required for DNA methylation are no longer capable of binding demethylated DNA because of an epigenetic switch. This switch would generate a chromatin configuration that impedes UHRF1 to recruit the methylating complexes to these sites, *i.e.* 'non-competent' chromatin. The methylating enzymes driven by UHRF1 should now be available to relocate to other chromosomal regions that have a 'competent' epigenetic configuration and unmethylated DNA, thereby promoting UHRF1-mediated DNA methylation at these sites.
Our plan is to gain a 'photograph' of the stable end point epigenetic configuration of the binding properties of UHRF1 and DNMTs by performing ChIP-seq, BS-ChIP-seq and high-resolution mass spectrometry analysis of histones, in combination with stable isotope labeling with amino acids in cell culture (SILAC), to quantitatively track with an unbiased approach the differences of histone post translational modifications, possibly identifying new epigenetic markers involved in the binding. We will study the epigenetic events that dynamically occur to allow UHRF1 binding to the chromatin during the epithelial to mesenchymal transition (EMT). We expect to unveil potential epigenetic signatures at genome level that define 'competent' and 'non-competent' chromatin configurations for the binding of UHRF1/DNMTs methylating complexes, that will *de facto* identify positive and negative epigenetic configurations for DNA methylation.
To verify whether a potential 'competent' and 'non-competent' chromatin for DNA methylation does indeed exist *in vivo*, we will investigate if the epigenetic configurations identified in the *in vitro* experiments are also present in human tissues. ChIP from paraffin-embedded tissues (Pat-ChIP) from a well-characterized tumour series previously published will be used to compare samples of normal colorectal mucosa with specimens of adenomas and of carcinomas of colon-rectum.

2) The involvement of UHRF1 and UHRF2 in prostate and colon cancer pathogenesis
In collaboration with with Dr. Carlo Catapano and Dr. Giuseppina Carbone of the Laboratory of Experimental Oncology of the Institute of Oncological Research in Bellinzona (CH), we have studied the pathological role of UHRF1 in prostate cancer. By integrating genomic data and functional assays in normal and prostate cancer cells, we have examined the expression profile of UHRF1 and found that it is over-expressed in prostate tumours and its expression is strictly correlated with the epigenetic effector EZH2. Analysis of a large cohort of tissue microarrays indicated that UHRF1 staining was absent in benign and increased in clinically localized prostate cancer where it was significantly associated with elevated Gleason score (≥8) and poor prognosis.
By immunohistochemistry (IHC) analysis, we discovered that UHRF1 expression was elevated in prostate cancer cell lines more aggressive and androgen independent. UHRF1 knockdown in prostate cancer cells (PC3) reduced proliferation, clonogenic capability, and anchorage independent growth and resulted in re-expression of several tumour suppressor genes frequently repressed and significantly inversely correlated to UHRF1 in prostate tumours. UHRF1 deregulation was associated with clinical outcome in prostate cancer patients. Follow-up data for 203 patients treated with radical prostatectomy and for which UHRF1 protein level had been assessed by IHC in the TMAs, where tested for clinical outcome. We found that patients with highest positivity for UHRF1 had significantly reduced overall survival (Log Rank (Mantel-Cox) p-value=0.07) compared to the patients with low or negative staining. Thus, high levels of UHRF1 might signal tumours more prone to progression and with a negative impact on patient survival. These data may have important clinical implications since UHRF1 may help to identify a subgroup of prostate cancer patients with poor prognosis.
Similar results have been obtained in colon cancer models. By IHC, we have found that UHRF1 expression was high in 60% of the samples of a cohort of 110 primary colorectal tumors, that generally displayed a less differentiated phenotype and a subverted histologic structure. High UHRF1 expression is associated with poorly differentiated and/or undifferentiated CRC (high grade, G3/G4 tumors).

3) The role of UHRF1 and UHRF2 in the epigenetic control of tumour suppressor genes during tumour progression

Analysis of a gene expression data set from human normal prostate and organ-confined prostate cancer samples and in prostate and colon cancer cells, showed that UHRF1 is a key epigenetic regulator and promotes epigenetic cross-talks at the promoters of tumour suppressor genes and is implicated in prostate and colon cancer progression:

a) UHRF1 is required for the methylation of DNA and H3K9me3 on the promoters of at least three oncosuppressors (CDH1, RARb and Nkx3.1) in prostate cancer cells
b) UHRF1 is required for the silencing of the PPARg in colon cancer cells RKO and recruits Suv39H1 to determine methylation of H3K9 at the promoter that tumour suppressor.
c) UHRF1 is required for the recruitment of Dnmt3a to the promoter of CDH1 in prostate cancer cells
d) UHRF1 and EZH2 are co-overexpressed in prostate and in colon cancer cell lines and in tumours of patients
e) UHRF1 is among the transcripts with statistically significant increase in tumours compared to normal prostate, with about 50% of tumour samples having elevated UHRF1 mRNA compared to normal prostate and with very high expression in about 20% of the cases.
f) UHRF1 also inversely correlates with a large number of many known tumour suppressor genes. An inverse correlation was also observed for prostate-specific genes, like microseminoprotein (PSP94), prostatic acid phosphatase (PAcP/ACPP), secretoglobin proteins (SCGB-A1, -1D1 and -1D2), acireductone dioxygenase 1 (ADI1) and human prostate-specific transglutaminase (TGM4).

PRINCIPAL COLLABORATIONS

1. Kirsten EDEPLI, Division of Liver Diseases Department of Developmental and Regenerative Biology. Icahn School of Medicine at Mount Sinai - New York, USA
2. Prof. Vittorio COLANTUONI, D.S.B.A. - Università degli Studi del Sannio, Benevento
3. Dr. Carlo CATAPANO e Dr.ssa Giuseppina CARBONE, Laboratory of Experimental Oncology – Istituto Oncologico della Svizzera Italiana, Bellinzona, Svizzera.
4. Dr. David MOLLEVI , Translational Research Laboratory, Institut Català d'Oncologia-ICO, IDIBELL. Barcelona, Spain
5. Dr. Tiziana BONALDI, IFOM-IEO Campus – Milan - Italy
6. Prof. Enrico AVVEDIMENTO, Dipartimento di Biologia e Patologia Cellulare e Molecolare, Università di Napoli
7. Dr. Magdalena GÖTZ, Helmholtz Zentrum Munich Institute of Stem Cell Research - Ludwig-Maximilians-Universität München (LMU); Munich, Germany.
8. Dr. Denti Michela, 'Centre for Integrative Biology', Università di Trento
9. Dr. Fabio SPADA and Prof. Heinrich LEONHARDT, Ludwig-Maximilians-Universität München (LMU); Munich, Germany.
10. Dr. Françoise DANTZER, Departement Intégrité du génome UMR 7175 – ESBS. Illkirch Strasbourg. France
11. Dr. Laurent BRINO and Prof. Oudet Transfected Cell Array platform, CEGBS-IGBMC, Strsbourg France