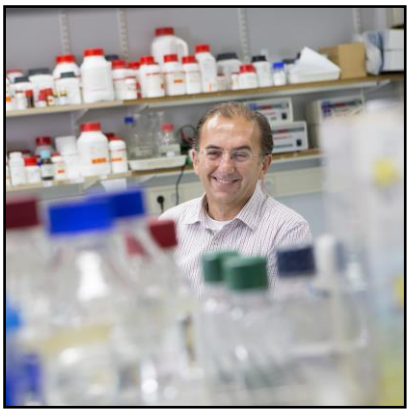

Curriculum vitae prof. dr. Riccardo Fodde, PhD

Date and place of birth : Sept. 16, 1960 ; Cagliari, Italy.

Nationality : Italian and Dutch.
Married (Monique Losekoot) with two sons (Marco and Iosto).

Researcher unique identifier: orcid.org/0000-0001-9839-4324;
Scopus Author ID 7007028278.

URL for web site: http://labriccardofodde.nl

Section A : Biosketch

Education

1980-1984 MSc (*cum laude*). "*Molecular analysis and chromosomal localization of the TK gene co-transfected in mouse cells with satellite DNA*", Oct. 1984. Faculty of Biology, Department of Genetics, University of Pavia, Italy.

1985-1990 PhD. "*Evolution of Multigene Families: Hemoglobins and Haptoglobins*", Nov. 1, 1990. Faculty of Medicine, Department of Human Genetics, University of Leiden, The Netherlands. Promotor: *prof. L.F. Bernini*

Professional Experience

1990 - 1992 post-doc at the Department of Human Genetics, University of Leiden, The Netherlands. Research Field: Molecular genetics of hereditary colon cancer. Supervisor: *prof. P. Meera Khan*

1992 visiting scientist at the Dept. of Molecular Genetics, Albert Einstein College of Medicine, Yeshiva University, New York, USA. Research Field: Generation and Analysis of Transgenic Mouse Models for Colorectal Tumorigenesis. Supervisor: *prof. Raju Kucherlapati*

1992 - 1997 fellow of the Royal Dutch Academy of Science (KNAW). Research Field: Generation and Analysis of Transgenic Mouse Models for Colorectal Tumorigenesis.

1995 - 1996 visiting professor at the Dept. of Human Genetics, University of Newcastle upon Tyne, U.K. Host: *prof. John Burn*.

1997 – 2001 associate professor (UHD) at the Department of Human Genetics, University of Leiden, The Netherlands.

2001 – 2003 full professor of "Cancer Genetics", Department of Human and Clinical Genetics, Leiden University Medical Center, Leiden, The Netherlands.

2003 – to date full professor of "Experimental Pathology", Josephine Nefkens Institute, Erasmus MC Cancer Institute, Erasmus University Medical Center, Rotterdam, The Netherlands.

2017 – to date adjunct professor at Zhejiang University, Hangzhou, Zhejiang Province, P. R. China.

Funding ID (since 2000)

- 2001-2004. The role of base excision repair in intestinal tumor initiation and progression. MWO 04-21. Maag, Lever, Darm Stichting. **MLDS**

- 2002-2006. Microarray analysis of factors determining apoptosis in human rectal cancers. RUL2002-2733. **KWF Dutch Cancer Society**

- 2003-2008. Modulation of stem cell differentiation by Apc/β-catenin signal transduction. 016.036.636. **NWO VICI**

- 2004-2008 Stem Cells in Development and Disease. 03038. **Senter/BSIK** (www.stemcells.nl)

- 2005-2009. Cross-talk between receptor tyrosine kinases and β-catenin signalling during tumor initiation and progression. DDHK 2005-3299. **KWF Dutch Cancer Society**

- 2007-2011. Identification of novel kinases involved in cancer-relevant processes. T3-103. **Top Institute Pharma**

- 2007-2011. Distinct dosages of Wnt/β-catenin signaling levels underlie colon and breast cancer stemness. EMCR 2007-3740. **KWF Dutch Cancer Society**

- 2007-2010. Migrating Cancer Stem Cells in breast and colon cancer. 37297. **EU FP6** (www.mcscs.eu).

- 2008-2012. TuMIC: an integrated concept of tumor metastasis. 201662. **EU FP7** (http://www.umm.uni-heidelberg.de/inst/cbtm/mbio/tumic/)

- 2008-2012. Cross-talk of sex hormones and Wnt/β-catenin signaling in endometrial cancer. EMCR 2008-4056. **KWF Dutch Cancer Society**

- 2010-2014. Netherlands Institute for Regenerative Medicine (**NIRM**). The Life Sciences & Health innovation program of the Dutch government. www.nirm.nl; www.regeneratieve-geneeskunde.nl

- 2010-2011. Erasmus Stem Cell Institute (**ESI**). (www.erasmusmc.nl/medical_genetics/esi/).

- 2012-2014. Paneth cells in intestinal cancer: supporting act or main feature? EMCR 2012-5473. **KWF Dutch Cancer Society**

- 2014-2018. Cell of Origin and Cancer Stem Cells in Ovarian Cancer. Erasmus MRACE funds.

- 2015-2019. Early alterations of the intestinal stem cell niche underlie sporadic colon cancer driven by "Western style" dietary factors. WCRF 1181. **World Cancer Research Funds International**.

- 2016-2020. EpCAMlow cancer stem cells: the culprit of liver metastasis in colon cancer? FP-1508. **MLDS (Dutch Gastroenterology Society)**.

- 2016-2020, The role of Wnt signaling in high-grade serous ovarian cancer stemness and therapy resistance. EMCR 2015-8090. **KWF Dutch Cancer Society**

- 2018-2022, Secretory Paneth-like cells as the origin of intestinal cancer. EMCR 2018-11407. **KWF Dutch Cancer Society.**

- 2018-2022, Dr. Jekyll and Mr. Hide: phenotypic plasticity and epigenetic control of EMT in oral cancer metastasis. **Mrace Grant 2018**.

International activities

- Visiting professor at the Division of Human Genetics, University of Newcastle upon Tyne, 1995-96.
- Member of the steering group of the CAPP study, a multi-national genetic trial to test the preventive potential of Aspirin and resistant starch among FAP and HNPCC patients.
- Contract Professor at the University of Bologna 1998.
- Director and faculty member of the European School of Cancer Genetics, Sestri Levante (GE) and Bertinoro di Romagna (FO), Italy, 1995-2002.
- Member of the Editorial Board of Human Mutation, Frontiers in Molecular and Cellular Oncology.
- Active reviewer for all major journals including Nature, Science, Cell, Cancer Cell, Cell Stem Cell, Nature Genetics, Nature Cell Biology, Nature Communications, etc.
- Member of the board of directors of the research school "Molecular Medicine"
- Member of the Scientific Board Telethon Italia (2000-2005)
- Member of the Scientific Board of the Dutch Cancer Society (2006-2011)
- Member of the European Molecular Biology Organization (EMBO), since 2002
- Member of the European Research Council (ERC) Starting Grant Commission, since 2015.
- Visiting professor at ZJU (Zhejiang University), Hangzhou, P. R. China, since 2017.

Scientific Leadership Profile

- During my PhD studies (prof. L. Bernini, University of Leiden, 1985-1990), I laid the basis for the elucidation of the genetic basis of thalassemias in The Netherlands where an extremely heterogeneous spectrum of ethnical populations from various regions throughout the malaria belt is present. This required a highly sensitive and comprehensive detection method covering multiple genes, and capable of detecting both point mutations as well as genomic rearrangements. The large number of defects identified allowed the elucidation of the molecular and genetic basis of these conditions in The Netherlands and formed the basis for carrier detection, pre- and post-natal diagnosis, and the application of preventive measures to reduce morbidity and mortality among several ethnical groups which comprise approx. 20% of the Dutch population.
Selected publications: **Genomics** 1990;8(4):732-5; **Blood** 1991;77(4):861-7; **Hum Mutat** 1994;3(2):83-94.

- During my first post-doctoral experience (prof. P.M. Meera Khan, University of Leiden, 1990-1992), I worked at hereditary colorectal cancer syndromes and in particular FAP (familial adenomatous polyposis) and Lynch syndrome (LS or HNPCC). The knowledge and methods acquired and developed during my PhD allowed their rapid application to the genes responsible for these conditions (APC and MYH for FAP and MSH2, MLH1, MSH6 and PMS2 for LS). This, combined with the unique familial colorectal cancer registries previously established in The Netherlands, led to the rapid elucidation of the genetic basis of these hereditary syndromes and the establishment of accurate genotype-phenotype correlations to allow pre- and post-natal diagnosis and presymptomatic predictions of their clinical courses. In fact, these findings were of a much wider societal impact, as the mutation spectrum among Dutch hereditary colorectal cancer kindreds contributed to the elucidation of the genetic basis of FAP and LS worldwide. Exemplary illustrations of this are represented by the identification of a US founder mutation in the MSH2 gene estimated to be responsible for 25% of the LS cases in the United States, and the description of a novel syndrome, hereditary desmoids disease (HDD) in a British family, subsequently validated in several European countries and in the US.
Selected publications: **Nat Genet** 1998;20(4):326-8; **N Engl J Med** 1998;339(8):511-8; **Nat Genet** 1999;23(2):142-4; **Am J Hum Genet** 2003;72(5):1088-100.

- A further extension of my post-doctoral fellowship was provided by the Royal Netherlands Academy of Arts and Sciences (KNAW; 1992-1997. Dept. of Human Genetics, University of Leiden, The Netherlands and Dept. of Molecular Genetics, Albert Einstein College of Medicine, New York, USA) and resulted in the

generation of unique preclinical mouse models for FAP and LS, and for intestinal cancer in general. In particular, the applicant developed the very first knock-out model for the mouse Apc gene ($Apc^{1638N/+}$), in addition to several others which provided further confirmation of the above mentioned genotype-phenotype correlations observed in clinical samples and largely contributed to the elucidation of the molecular and cellular basis of tumor onset in the GI tract. These animal models have been employed by the scientific community at large and are still regarded as unique tools for basic and translational cancer research worldwide.
Selected publications: **PNAS USA** 1994;91(19):8969-73; **Gastroenterology** 1998;114(2):275-83; **Genes Dev** 1999;13(10):1309-21; **Gastroenterology** 2000;119(4):1045-53; **Trends Mol Med** 2001;7(8):369-73; **Oncogene** 2006;25(13):1841-51; **Gastroenterology** 2006;131(4):1096-109. **Carcinogenesis** 2010;31(5):946-52; **Gut** 2011; 60(9):1204-12.

- During a one-year visiting professorship at the University of Newcastle, UK (1995-1996) and in close collaboration with prof. John Burn, a unique genetic trial was established and implemented aimed at the evaluation of the preventive role of Aspirin and Amylose (resistant starch) in HNPCC and FAP. The CAPP studies (Concerted Action for the Prevention of Polyps; http://research.ncl.ac.uk/capp2/) not only demonstrated the preventive role of Aspirin in LS but also opened new avenues to explore aspirin's role as a coadjuvant in cytotoxic cancer therapy. The latter is currently being further investigated in collaboration with the LUMC (dr. G.I. Liefers) and has led to the first (ongoing) prospective trial where aspirin is randomized in colon cancer patients.
Selected publications: **N Engl J Med** 2008;359(24):2567-78. **Lancet** 2011; 378(9809):2081-7. **Br J Cancer** 2012;106(9):1564-70. **JAMA Intern Med** 2014;174(5):732-9.

- The above studies spanning from basic to translational and clinical research allowed me to integrate the results towards the elucidation of the molecular and cellular basis of intestinal cancer. Highlights of this comprehensive research strategy are the "just-right model" to redefine Knudson's the two-hit hypothesis for the APC tumor suppressor gene; the relevance of dosage in Wnt signaling and its consequence for tumor susceptibility; the APC gene as primary cause of chromosomal instability in colorectal cancer; and the role of Wnt in defining self-renewal and differentiation potential of stem cells. These results also led to the prestigious VICI grant from the Dutch Research Council (NWO) to study stemness in homeostasis and cancer at a time when the current cancer stem cell "hype" had not emerged yet. The VICI recognition also coincided with my first professorial chair ("*Cancer Genetics*", University of Leiden; 2001). Shortly after (from 2003 on), I moved my research group from Leiden to the Erasmus University Medical Center in Rotterdam where I took the chair of "*Experimental Pathology*" at the Dept. of Pathology. The move to Rotterdam allowed me to place my research in a more multi-disciplinary and high-standard environment where to implement a novel research line on stem cells in homeostasis and cancer.
Selected publications: **Nat Rev Cancer** 2001;1(1):55-67; **Nat Cell Biol.** 2001;3(4):433-8; **Hum Mol Genet** 2002;11(13):1549-60; **Nat Genet** 2002;32(4):594-605; **Am J Pathol** 2007;170(1):377-87; **PLoS Genet** 2009;5(7):e1000547; **Cell Stem Cell** 2011;9(4):345-56; **PLoS Genet.** 2013;9(5):e1003424.

- During the last years the focus of my research has been centered on the role played by stem cells and their niche not only in intestinal neoplasia but also in mammary, endometrial, oral, ovarian, esophageal, and, most recently, oral cancer. In the particular case of the intestinal (cancer) stem cell niche, my research has been focusing on Paneth cells because of their dual role as niche cells which secrete niche-essential ligands and growth factors such as Wnt3a and phospholipases, and as quiescent stem cells which are activated upon tissue insults (e.g. inflammation). Paneth cells reside exclusively in the small intestine and as such are apparently irrelevant to colon cancer. However, in chronic inflammatory conditions such as ulcerative colitis (inflammatory bowel syndromes, IBD), metaplastic Paneth cells are present in the normal and tumor-associated colonic mucosa, allegedly to support Lgr5-expressing cancer stem cells. Our latest and most promising line of research is aimed at the identification and functional characterization of these Paneth-like cells in the colon and of different members of the secreted phospholipases (PLA2G2A and PLA2G10) as stem cell niche factors and genetic modifier of IBD-associated colon cancer. This also include the characterization of their metabolic status when compared with the actively proliferating $Lgr5^{+}$ stem cells, and their capacity to de-differentiate upon inflammatory insults.

Selected publications: **PLoS One.** 2012;7(7):e40691. **PLoS One.** 2012;7(6):e38965. **Cell Commun Adhes.** 2011, 18(3):33-44. **EMBO Rep.** 2011,12(6):483-4. **Cancer Res**. 2015, 75:3608-22. **Cell Stem Cell**, 2016, 19(1):38-51. **Nature,** 2017, 543:424–427. **Cell Reports**, 2018, 24(9):2312-2328

- Presently, the laboratory is following two main research lines touching on different cancer types and focusing on the cellular and molecular mechanisms underlying 1. the very onset of cancer and the role played by extrinsic risk factors (e.g. inflammation, diet, dysbiosis) on the stem cell niche and its microenvironment; and 2. phenotypic plasticity as the main hallmark of the metastasizing cancer cell.

1. As shown by our recent publications, the elucidation of the mechanisms through which common etiology cancer risk affect specific components of the intestinal stem cell niche represent the earliest alteration leading to the formation of a tumor. By employing state-of-the-art *in vivo* models we are now focusing on inflammation and on western-style dietary factors as the main predisposing factors in colon cancer. Overall, it appears that tissue insults are invariably accompanied by a depletion of resident and high-turnover stem cells (Lgr5+) and by the de-differentiation and acquisition of stem-like properties by a number of post-mitotic and fully differentiated cell lineages. The trade-off effect of the recruitment of alternative stem cell sources and the resulting improved regenerative response is an increased chance of tumor-initiating mutations to occur.
2. We are investigating phenotypic plasticity in specific subpopulation of cancer cells with stem-like, EMT, and chemo-resistant features, thought to underlie distant metastasis in colon, ovarian and oral cancer. This has led, among others, to the elucidation of the molecular basis of malignant transformation in oral cancer and the role of the Nurd chromatin remodeling complex in EMT/MET, in collaboration with prof. Peter Verrijzer. Similar research is being conducted on colon and ovarian cancer towards the identification and characterization of CTCs in patient-derived blood and ascites as future diagnostic, prognostic, and therapeutic tools.

Section b: Track-record

1. Selected publications, as first and senior author

- Schmitt M, Schewe M, Sacchetti A, Feijtel D, van de Geer WS, Teeuwssen M, Sleddens HF, Joosten R, van Royen ME, van de Werken HJG, van Es J, Clevers H, Fodde R. Paneth cells respond to inflammation and contribute to tissue regeneration by acquiring stem-like features through activation of the SCF/c-Kit signaling axis. **Cell Rep** *in press* (2018)
- Schewe M, Sacchetti A, Schmitt M, Fodde R. The Organoid Reconstitution Assay ORA) for the Functional Analysis of Intestinal Stem and Niche Cells. **J Vis Exp**. 2017 Nov 20;(129). doi: 10.3791/56329.
- Rodríguez-Colman MJ, Schewe M, Meerlo M, Stigter E, Gerrits J, Pras-Raves M, Sacchetti A, Hornsveld M, Oost KC, Snippert HJ, Verhoeven-Duif N, Fodde R, Burgering BM. Interplay between metabolic identities in the intestinal crypt supports stem cell function. **Nature** 2017, Mar 16, 543:424–427
- Fodde R, Schmitt M, Schewe M, Augenlicht LH. Modelling western dietary habits in the mouse: easier said than done. **HepatoBiliary Surg Nutr** 2017, 6(2):138-140
- Mohd-Sarip A, Teeuwssen M, Bot AG, De Herdt MJ, Willems SM, Baatenburg de Jong RJ, Looijenga LHJ, Zatreanu D, Bezstarosti K, van Riet J, Oole E, van Ijcken WFJ, van de Werken HJG, Demmers JA, Fodde R, Verrijzer CP. DOC1-Dependent Recruitment of NURD Reveals Antagonism with SWI/SNF during Epithelial-Mesenchymal Transition in Oral Cancer Cells. **Cell Rep**. 2017 Jul 5;20(1):61-75
- Schewe M, Franken PF, Sacchetti A, Schmitt M, Joosten R, Böttcher R, van Royen ME, Jeammet L, Payré C, Scott PM, Webb NR, Gelb M, Cormier RT, Lambeau G, and Fodde R. Secreted phospholipases A2 are stem cell niche factors with distinct roles in homeostasis, inflammation and cancer. **Cell Stem Cell**, 2016 Jul 7;19(1):38-51.
- van der Zee M, Sacchetti A, Cansoy M, Joosten R, Teeuwssen M, Heijmans-Antonissen C, Ewing-Graham PC, Burger CW, Blok LJ, Fodde R. IL6/JAK1/STAT3 Signaling Blockade in Endometrial Cancer Affects the ALDHhi/CD126+ Stem-like Component and Reduces Tumor Burden. **Cancer Res.** 2015 Sep 1;75(17):3608-22.
- Atlasi Y, Noori R, Gaspar C, Franken P, Sacchetti A, Rafati H, Mahmoudi T, Decraene C, Calin GA, Merrill BJ, Fodde R. Wnt signaling regulates the lineage differentiation potential of mouse embryonic stem cells through Tcf3 down-regulation. **PLoS Genet.** 2013 May;9(5):e1003424. doi:10.1371/journal.pgen.1003424. Epub 2013 May 2. PubMed PMID: 23658527; PubMed Central PMCID: PMC3642041.

- Luis TC, Naber BA, Roozen PP, Brugman MH, de Haas EF, Ghazvini M, Fibbe WE, van Dongen JJ, Fodde R*, Staal FJ. Canonical wnt signaling regulates hematopoiesis in a dosage-dependent fashion. **Cell Stem Cell.** 2011 Oct 4;9(4):345-56. doi: 10.1016/j.stem.2011.07.017. PubMed PMID: 21982234.
- Gaspar C, Franken P, Molenaar L, Breukel C, van der Valk M, Smits R, Fodde R. A targeted constitutive mutation in the APC tumor suppressor gene underlies mammary but not intestinal tumorigenesis. **PLoS Genet.** 2009 Jul;5(7):e1000547. doi: 10.1371/journal.pgen.1000547. Epub 2009 Jul 3. PubMed PMID: 19578404; PubMed Central PMCID: PMC2697381.
- Fodde R, Brabletz T. Wnt/beta-catenin signaling in cancer stemness and malignant behavior. **Curr Opin Cell Biol.** 2007 Apr;19(2):150-8. Epub 2007 Feb 16. PubMed PMID: 17306971.
- Kielman MF, Rindapää M, Gaspar C, van Poppel N, Breukel C, van Leeuwen S, Taketo MM, Roberts S, Smits R, Fodde R. Apc modulates embryonic stem-cell differentiation by controlling the dosage of beta-catenin signaling. **Nat Genet.** 2002 Dec;32(4):594-605. Epub 2002 Nov 11. PubMed PMID: 12426568.
- Fodde R, Smits R, Clevers H. APC, signal transduction and genetic instability in colorectal cancer. **Nat Rev Cancer.** 2001 Oct;1(1):55-67. Review. PubMed PMID: 11900252.
- Fodde R, Kuipers J, Rosenberg C, Smits R, Kielman M, Gaspar C, van Es JH, Breukel C, Wiegant J, Giles RH, Clevers H. Mutations in the APC tumour suppressor gene cause chromosomal instability. **Nat Cell Biol.** 2001 Apr;3(4):433-8. PubMed PMID: 11283620.
- Smits R, Ruiz P, Diaz-Cano S, Luz A, Jagmohan-Changur S, Breukel C, Birchmeier C, Birchmeier W, Fodde R. E-cadherin and adenomatous polyposis coli mutations are synergistic in intestinal tumor initiation in mice. **Gastroenterology.** 2000 Oct;119(4):1045-53. PubMed PMID: 11040191.
- Wijnen J, de Leeuw W, Vasen H, van der Klift H, Møller P, Stormorken A,Meijers-Heijboer H, Lindhout D, Menko F, Vossen S, Möslein G, Tops C, Bröcker-Vriends A, Wu Y, Hofstra R, Sijmons R, Cornelisse C, Morreau H, Fodde R. Familial endometrial cancer in female carriers of MSH6 germline mutations. **Nat Genet.** 1999 Oct;23(2):142-4. PubMed PMID: 10508506.
- Smits R, Kielman MF, Breukel C, Zurcher C, Neufeld K, Jagmohan-Changur S, Hofland N, van Dijk J, White R, Edelmann W, Kucherlapati R, Khan PM, Fodde R. Apc1638T: a mouse model delineating critical domains of the adenomatous polyposis coli protein involved in tumorigenesis and development. **Genes Dev.** 1999 May 15;13(10):1309-21. PubMed PMID: 10346819; PubMed Central PMCID: PMC316713.
- Wijnen J, van der Klift H, Vasen H, Khan PM, Menko F, Tops C, Meijers Heijboer H, Lindhout D, Møller P, Fodde R. MSH2 genomic deletions are a frequent cause of HNPCC. **Nat Genet.** 1998 Dec;20(4):326-8. PubMed PMID: 9843200.
- Wijnen JT, Vasen HF, Khan PM, Zwinderman AH, van der Klift H, Mulder A, Tops C, Møller P, Fodde R. Clinical findings with implications for genetic testing in families with clustering of colorectal cancer. **N Engl J Med.** 1998 Aug 20;339(8):511-8. PubMed PMID: 9709044.
- Smits R, van der Houven van Oordt W, Luz A, Zurcher C, Jagmohan-Changur S, Breukel C, Khan PM, Fodde R**.** Apc1638N: a mouse model for familial adenomatous polyposis-associated desmoid tumors and cutaneous cysts. **Gastroenterology.** 1998 Feb;114(2):275-83. PubMed PMID: 9453487.
- Eccles DM, van der Luijt R, Breukel C, Bullman H, Bunyan D, Fisher A, Barber J, du Boulay C, Primrose J, Burn J, Fodde R**.** Hereditary desmoid disease due to a frameshift mutation at codon 1924 of the APC gene. **Am J Hum Genet.** 1996 Dec;59(6):1193-201. PubMed PMID: 8940264; PubMed Central PMCID: PMC1914868.
- Fodde R, Edelmann W, Yang K, van Leeuwen C, Carlson C, Renault B, Breukel C, Alt E, Lipkin M, Khan PM, et al. A targeted chain-termination mutation in the mouse Apc gene results in multiple intestinal tumors. **Proc Natl Acad Sci USA.** 1994 Sep 13;91(19):8969-73. PubMed PMID: 8090754; PubMed Central PMCID: PMC44728.

Complete publication list.
See: http://www.ncbi.nlm.nih.gov/pubmed/?term=Fodde+R
Or http://www.labriccardofodde.nl

Citation Report.

Total Publications:	253 (1998-2017)
h-index:	61
Average citations per item:	63,06
Sum of Times Cited:	15.953
Without self-citations:	15.370
Citing articles:	11.992
Without self-citations:	11.836

2. International Prizes/Awards/Academy memberships

- Short-term European Molecular Biology Organization (EMBO) fellowship, 1985
- Fellow of the Royal Dutch Academy of Sciences (KNAW) 1992-1997
- NWO (Dutch Research Council) VICI-laureate, 2003
- Frieda den Hertog Jager Prize. Dutch Society of Gastroenterology, 2004
- EMBO (European Molecular Biology Organization) full member since 2005
- MD Anderson Cancer Center, Houston, TX, USA. *Distinguished Lecturer Science in Medicine.* 2016
- Chinese University of Hong Kong, Hong Kong, China. *Honourable Speaker "Cancer: from Biology to Treatment"*. 2016
- The 18th Royan International Award for Regenerative Medicine, Tehran, Iran, September 2017.
- ZJU Global Lecture Prize, Hangzhou, Zhejiang, P.R. China, May 21, 2018.

3. Major contributions to early careers of excellent researchers

To date, I trained a total of 16 PhD students who completed and defended their thesis under my supervisions, and more than 40 among MSc students and post-doctoral fellows. Among them, some are now well-established and internationally recognized scientists (e.g. Dr. R. Smits first at Genentech, San Francisco CA, USA, and subsequently at the Erasmus MC; Dr. P. Hohenstein first at MRC UK, The Roslin Institute, University of Edinburgh, UK; and now at the LUMC in Leiden, The Netherlands; Dr. Y. Atlasi, at the Radboud University Medical Centre, Nijmegen, NL; Dr. Claudia Gaspar, Faculdade de Medicina de Lisboa, Instituto de Medicina Molecular, Lisboa, Portugal). See also http://labriccardofodde.nl