CURRICULUM VITAE

PART I: General information

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Place of birth Rome, Italy

Education:

1986	M.D. (cum laude)	University of Rome "La Sapienza", Italy
1990	Medical Specialties, Cardiology	University of Rome "La Sapienza", Italy (cum laude)
1996	Ph.D.	Imperial College, University of London, UK

Postdoctoral Training:

Internship and Residency

1983	Intern, Department of General Medicine, University of Rome "La Sapienza", Italy (pre-doctoral rotation)
1984	Intern, Department of Emergency Medicine & Surgery, University of Rome "La Sapienza", Italy (pre-doctoral rotation)
1985	SHO and Registrar, Division of Cardiac Medicine, Cardiovascular Surgery Institute, University of Rome " La Sapienza", Italy
1986-1990	Resident, Division of Cardiac Medicine, Cardiovascular Surgery Institute, University of Rome " La Sapienza", Italy

Cinical & Research Fellowships

1993-1998	Cinical &Research Fellow, Laboratory of Cardiovascular Pharmacology, Cardiology Department, Cardiovascular Surgery Institute, 1st University of Rome "La Sapienza", Italy
1998-2000	Research Fellow (Medicine), Harvard Medical School
1998-2004	Research Fellow in Medicine, Cardiology Division/Cardiovascular Research Center, Massachusetts General Hospital
2004-	Assistant in Biology (Medicine), Cardiology Division/Cardiovascular Research Center, Massachusetts General Hospital

Licensure and Certification:

1986	NIH Registration, Rome, Italy
1989	NIH Registration, London, UK
1990	Medical Specialties, Cardiology, Rome, Italy

Academic Appointments:

2000-2003	Instructor in Medicine, Harvard Medical School
2003-	Assistant Professor of Medicine, Harvard Medical School

Hospital or Affiliated Institution Appointments:

1990-1993	Honorary Registrar in Cardiology, Royal Brompton Hospital, London, UK
1993-1999	Clinical Cardiologist (Staff), Cardiac Medicine Dept., NIH Hospitals, Rome, Italy
2004-	Assistant in Biology (Medicine), Cardiology Division/Cardiovascular Research Center, Massachusetts General Hospital

Major Committee Assignments

2005-	NIH-NHLBI Member of Committee for K08/K02/K01 Grant Review
2005-	MGH-SRAC Committee Member

Professional Societies:

1987-	Italian Society of Cardiology, member
1991-	International Society for Heart Research, member
1991-	British Society for Cardiovascular Research, member
1993-	Italian Society for Cardiovascular Research, member
2001-	American Heart Association Member of Scientific Council(s) – Basic Cardiovascular Science Council
2003-	Biophysical Society

Community Service Related to Professional Work:

1993	Associazione anni verdi. Cardiology consultant for handicap and social level of poverty cases. NIH Hospitals, Rome, Italy.

Professional Societies:

Ad Hoc Reviewer	Circulation Circulation Research Journal Molecular Cellular Cardiology Cardiovascular Research European Heart Journal

Awards and Honors:

1990-1992	Ph.D. studies funded by competitive Squibb Fellowship for Cardiovascular Research.
1999	1st Prize, Heart Failure Society of America Young Investigator Award
2002	Calderwood Award, Massachusetts General Hospital, Boston, MA
2002	Mentored Clinical Scientist Award, National Institutes of Health
2002	Mentored Clinical Scientist Development Program Award, National Institutes of Aging and Harvard Medical School
2005	FIRB Grant, Harvard Medical School-Armenise Foundation – USA Coordinator

PART II: Research, Teaching and Clinical Contributions

A. Narrative Report

A large area of research and clinical investigation are cellular abnormalities associated with left ventricular hypertrophy and heart failure. Over the last five years, advances in somatic gene transfer, using viral vectors, has provided investigators with the needed tools to investigate signaling pathways contributing to the pathogenesis of heart failure. Ultimately it is the goal of this research is to develop and test the feasibility of being able to manipulate gene expression and devise therapeutic strategies.

Heart failure is characterized by a number of abnormalities in Ca^{++} cycling. One of the key abnormalities in both experimental and human heart failure is a defect in the sarcoplasmic reticulum Ca^{++}-ATPase pump (SERCA2a). SERCA2a has been shown to account for a reduced Ca^{++} uptake during diastole as well as reduced systolic Ca^{++} levels. Using a genetic approach through adenoviral gene transfer we believed it possible to furnish single cardiac myocytes with abundant normal copies of the gene encoding for SERCA2a. Our study was the first to report that overexpressing SERCA2a using recombinant adenoviruses enhances contractile function and improves intracellular Ca^{++} handling in human cardiac myocytes. Furthermore, it was shown that contractile function could be enhanced in human cardiac myocytes by modulating SERCA 2a through the inhibition of its regulatory protein phospholamban. These two critical experimental findings provided a sound basis for investigating further the molecular mechanisms underlying defects seen in heart failure using genetic manipulation. The research is technically challenging due to the restricted availability of failing and especially non-failing donor hearts. Also to be able to isolate single adult cardiac myocytes from human hearts and to be able to maintain them in a non-de-differentiated state for up to 72 hours is also required.

In addition, the development of surgical animal models of heart failure was required for validation of our hypotheses. The surgical approach was optimized so as to reduce the occurrence of scarring and inflammation, which could easy confound our experimental observations. Viral vectors are known to cause myocardial inflammation. Similarly, the cell isolation and culture procedures for diseased rodent hearts had to be developed and optimized, which they were. By using rodent models of heart failure that were first well characterized at the functional level, we were able to exclude potential negative side effects that might result from overexpression of the SERCA2a gene *in vitro* as well as *in vivo*.

Having successfully tested the key hypothesis that SERCA2a overexpression in heart failure would restore myocardial contractility and prove to be of therapeutic benefit in a rodent model, more recently we have expanded our research to a large animal model. By using a large animal model, we can develop and test new strategies for gene delivery that might be applicable to the human condition. We first tried newer viral vectors i.e., the adenoassociated virus (AAV). We are testing the vectors in single adult swine and human myocytes in order to optimize the conditions and observe potential side effects of the transduction. These *in vitro* studies are followed by *in vivo* large animal studies.

All of our experimental findings point to a key defect in Ca^{++} handling as being a factor in cardiac dysfunction in failing hearts. As a result, we have expanding yet again our experimental investigation and approaches using state of the art techniques i.e., genomic and proteomic analysis. By identifying deregulated genes in failing human hearts we will be able to target genes that impact cardiac muscle function with the ultimate goal of identifying therapeutic targets.

The experimental findings left a key question on the origin of the down-regulation of the Ca^{++} regulating pump in the natural history of heart failure. Along this line in our most recent line of research we expanded the experimental research towards the proteomics characterization of the sub-cellular compartments of the myocytes and in particular of the endoplasmic reticulum (ER/SR), the site of control of oscillating Ca^{++} content in the heart, but also of several vital functions (requiring more stable Ca^{++} concentrations) as it provides a unique oxidizing compartment for the assembly and folding of newly synthesized proteins, post translational modifications, a transport route to direct proteins to their destination as well as quality control of misfolded polypeptides. The ER/SR integrates this dual function as high intraluminal Ca^{2+} is a key determinant for the synthesis and processing of proteins, low cytosolic Ca^{2+} for cell survival and Ca^{++} oscillations for myocyte function.

Various conditions can interfere with ER function and are collectively called ER stress, which in turn can be triggered by alterations of ER Ca^{2+} homeostasis. Upon ER stress induction a series of changes in protein structures may occur (protein misfolding) and, a conserved stress response pathway, the UPR allows cells to tolerate accumulation of misfolded proteins and act to correct the defect. A signal selectively activates the transcription of genes encoding for ER-localized proteins –chaperone proteins - to increase the rate of protein folding. We thus explored the pathways of ER stress in heart failure and shown that in dilated cardiomyopathy protein misfolding can be the mechanism at the origin of the pathogenesis of the disease. We observed signs of ER stress such as activation of the chaperones proteins or inactivation of the ubiquitin-proteasome system as well as signs of misfolding proteins such as the presence of protein aggregates in the myocytes and in between myocytes in failing human hearts, a pathogenetic observation that is common to many other disease including neurodegenerative diseases (Alzheimer dementia), diabetes, cystic fibrosis, many forms of cancer, polycystic liver and kidney etc.

We are exploring a very new pathogenetic hypothesis and we hope that by identifying pathogenetic pathways that lead to the defect in contractility, our research will result in the development of early diagnostic tools for preventive therapies and the treatment of early stage heart failure with the dream of more curative tools.

B. Funding Information (education & research)

1990-1992 Squibb Fellowship for Cardiovascular Research (competitive), funded Ph.D. studies, "Studies of contractile properties of isolated cardiac myocytes from animal models and human heart failure and hypertrophy"

2002- NIH, K08 HL69842 (PI: Federica del Monte), "SERCA2a abnormalities and chaperones in heart failure"

2005- Giovanni Armenise Harvard Foundation - Harvard Medical International RBIN042Z2Y (USA Coordinator: Federica del Monte), "Chaperone proteins in the development of cardiac hypertrophy and failure

C. Report of Current Research Activities

Excitation-contraction coupling in Heart Failure in human and animal models

Gene transfer in the Cardiovascular System

Ca^{2+} Homeostasis and Energy Reserve in Heart Failure

Ca^{2+} Homeostasis and Myocardial Protection in Ischemia/Reperfusion Injury

Sarcoplasmic Reticulum Protein/Function Analysis Heart Failure

Gene expression analysis in heart failure

Protein expression analysis in heart failure, compartmental proteomics

Protein misfolding in heart failure

D. Report of Clinical/Research Teaching

1. Local contributions

a. Harvard Medical School

1999 "Treatment of Heart Failure: Basic Science and Clinical Aspects", Harvard Medical School. Lecturer. 10 fourth-year medical students, 1 month/year, 1 lecture, prep time=5 hours.

e. Advisory responsibilities:

1995	Thesis advisor for 1 student for MD Degree, 1st University of Rome, Italy
1995	Thesis supervisor of 1 student for Perfusionist Specialty Degree, 1st University of Rome, Italy
1996	Thesis supervisor of 1 student for Internal Medicine Specialty Degree, II University of Rome, Italy
1998-date	Weekly laboratory meeting, 50 hours/year
2002-2004	Supervision of 1 student from MIT, 250 hours/year
2002-2003	Supervision of 1 student from HMS, 250 hours/year
2002-2003	Supervision of 1 visiting fellow, 400 hours/year
2003	Supervision on 1 student, 125 hrs/day
2003-2005	Supervising 1 full time technician
2004-date	Supervising full time post doc
7-8/2005	Supervision on 1 student, 125 hrs/day
2005-date	Supervising 1 part time post doc (collaboration with neurology department)
2006-date	Supervising 1 part time post docs (Collabration with CVRC)
2005-date	Advisory committee on a K08 grant

E. Report of Clinical Activities

1. Teaching Hospital

1993-1998 Royal Brompton National Heart & Lung Hospitals, Imperial College, London, United Kingdom
a. Practice: care delivery, general cardiology, in-hospital patient rounds
b. Time commitment: once/week, 6 hours out-patient clinic

2. Non-Teaching Hospital

1989-1993 NIH Hospitals, Rome, Italy
a. Practice: Coronary Care Unit and Sub-Intensive Care Unit
Resuscitation Unit of the Emergency Room
General Cardiology Ward
Internal Medical Department
b. Time Commitment:
1-2/week 12 hours in-hospital on call (+ night shifts)
1 weekend/month in-hospital on call (+ night shift)
8 hours daily in-patient/out-patient care for general cardiology and non-invasive cardiovascular evaluation service (rotations)

PART III: Bibliography

Original Articles

1. Greco C, Di Piero V, Cavalletti C, Argentino C, D'Agostino R, **del Monte F**, Scopinaro F. Myocardial ischemia during stroke: scintigraphic demonstration. *Cardiologia* 1989; 34 (5): 455-457.

2. Alessandri N, Pannarale G, **del Monte F**, Moretti F, Marino B, Reale A. Hypertrophic obstructive cardiomyopathy and infective endocarditis: a report of seven cases and a review of the literature. *Eur Heart J* 1990; 11 (n): 1041-1048.

3. Jones SM, Hunt NA, **del Monte F**, Harding SE. Contraction of cardiac myocytes from noradrenaline-treated rats in response to isoprenaline, forskolin and dibutyryl cAMP. *Eur J Pharmacol* 1990; 191 (2):129-140.

4. Harding SE, Jones SM, O'Gara P, **del Monte F**, Vescovo G, Poole-Wilson PA. Isolated ventricular myocytes from failing ad non failing human heart; the relation of age and clinical status of patients to isoproterenol response. *J Mol Cell Cardiol* 1992; 24 (5): 549-564.

5. Harding SE, Jones SM, Vescovo G, **del Monte F**, Poole-Wilson PA. Reduced contractile response to forskolin and a cyclic AMP analogue in myocytes from failing human ventricle. *Eur J Pharmacol* 1992; 223 (1): 39-48.

6. Monti F, Dawodu AA, Giglio V, Lanti M, **del Monte F**, Sugimoto S, Terracciano C, Schiariti M, Puddu PE, Campa PP. Effetti inotropi e batmotropi della beta stimolazione: studio di confronto tra dobutamina e dopamina nel muscolo papillare di cavia in contrazione isometrica. *Cardiologia* 1992; 37 (19): 635-639.

7. **del Monte F**, Mynett JR, Sugden PH, Poole-Wilson PA, Harding SE. Subcellular mechanism of the species difference in the contractile response of ventricular myocytes to endothelin-1. *Cardioscience* 1993; 4 (3): 185-191.

8. **del Monte F**, Kaumann AJ, Poole-Wilson PA, Wynne DG, Harding SE. Coexistence of functioning β_1- and β_2-adrenoceptors in single myocytes from failing human ventricle. *Circulation* 1993; 88 (3): 854-863.

9. Naqvi RU, **del Monte F**, O'Gara P, Harding SE, MacLeod KT. Characteristics of myocytes isolated from the heart of renovascular hypertensive guinea-pigs. *Am J Physiol* 1994; 266 (5 Pt 2): H1886-H1895.

10. Ferrara N, O'Gara P, Wynne DG, Brown LA, **del Monte F**, Poole-Wilson PA, Harding SE. Decreased contractile responses to isoproterenol in isolated cardiac myocytes from aging guinea-pigs. *J Mol Cell Cardiol* 1995; 27(5): 1141-1150.

11. Sanders L, Lynham JA, Bond B, **del Monte F**, Harding SE, Kaumann AJ. Sensitisation of human atrial 5-HT_4 receptors by chronic β-blocker treatment. *Circulation* 1995; 92 (9): 2526-2539.

12. **del Monte F**, O'Gara P, Poole-Wilson PA, Yacoub M, Harding SE. Cell geometry and contractile abnormalities of myocytes from failing human ventricle. *Cardiovasc Res* 1995; 30 (2): 281-290.

13. Sugimoto S, Puddu PE, Monti F, Dawodu AA, **del Monte F**, Schiariti M, Campa PP, Marino B. Activation of ATP-dependent K^+ channels enhances myocardial protection due to cold high potassium cardioplegia: a force-frequency relationship study. *J Mol Cell Cardiol* 1995; 27 (9): 1867-1881.

14. Lefroy DC, Crake T, **del Monte F**, Vescovo G, Dalla Libera LD, Harding SE, Poole-Wilson PA. Angiotensin II and contraction of isolated myocytes from human, guinea-pig and infarcted rat hearts. *Am J Physiol* 1996; 270 (Pt 2): H2060-H2069.

15. Harding SE, Brown LA, **del Monte F**, Davies CH, O'Gara P, Vescovo G, Wynne DG, Poole-Wilson PA. Acceleration of contraction by β-adrenoceptor stimulation is greater in ventricular myocytes from failing than non-failing human hearts. *Basic Res Cardiol* 1996; 91 Suppl 2: 53-56.

16. Wynne DG, **del Monte F**, Harding SE. Cyclic AMP levels in ventricular myocytes from noradrenaline-treated guinea-pigs. *Eur J Pharmacol* 1996; 310: 235-242.

17. Harding S.E., Brown L.A., **del Monte F**, Davies C.H., O'Gara P., Vescovo G., Wynne D.G., Poole-Wilson P.A. Acceleration of contraction by β-adrenoceptor stimulation is greater in ventricular myocytes from failing than non-failing human hearts. *Basic Res Cardiol* 1996; 91 Suppl 2: 53-56.

18. **del Monte F**, Harding SE, Schmidt U, Matsui T, Kang ZB, Dec GW, Gwathmey JK, Rosenzweig A, Hajjar RJ. Restoration of contractile function in isolated cardiomyocytes from failing human hearts by gene transfer of SERCA2a. *Circulation* 1999; 100: 2308-2311.

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23 Myamoto MI, **del Monte F**, Schmidt U, Matsui T, Guerrero JL, Gwathmey JK, Rosenzweig A, Hajjar RJ. Adenoviral gene transfer of SERCA2a improves left ventricular function in aortic-banded rats in transition to heart failure. *Proc Natl Acad Sci* 2000; 97 (2): 793-798.

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Reviews

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Book Chapters

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2. Piccolo GP, Macchiarelli AG, Scibilia G, **del Monte F**. Protocolli di comportamento e di condotta del personale addetto alla cura del ricevente nel periodo post-operatorio immediato. In: Marino B, Piccoli G, editors. Il Trapianto Cardiaco Ortotopico. Roma: Masson; 1990. p. 195-198.

3. Jones SM, Hunt NA, **del Monte F**, Harding SE. Reduced contractile response to stimulation by isoprenaline, forskolin and dibutyryl cAMP in isolated myocytes from noradrenaline-treated rats. In: Anonymous, editors. Adrenoceptors: Structure, Mechanisms, Function. Advances in Pharmacological Science. The Pharmacology of Adrenoceptors. E Szabadi: Basel; 1991. p. 331-332.

4. Harding SE, Brown LA, **del Monte F**, O'Gara P, Wynne DG, Poole-Wilson PA. Parallel changes in the β-adrenoceptor/adenylyl cyclase system between the failing human heart and the noradrenaline-treated guinea-pig. In: Nagano M, Takeda N, Dhalla NS, editors. The Cardiomyopathic Heart. Raven Press; 1993. p. 361-374.

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6. **del Monte F**, Puddu P.E., Harding S.E. β-adrenoceptor desensitisation in human heart failure: focus on isolated myocytes. In: Puddu PE, Bing RJ, Campa PP, Poole-Wilson PA, editors. Congestive Heart Failure: From Basic Science to Therapeutics. Roma: Cardioricerca; 1997. p. 39-62.

7. **del Monte**, Harding SE, Hajjar RJ. Manipulation of SERCA2a in the heart by gene transfer. In: Hasensfuss G, Marban E. Molecular Strategy to the Therapy of Heart Failure. Springer; 2000. p. 53-68.

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9. **del Monte F**, Hajjar RJ. Efficient viral gene transfer to rodent hearts in vivo. In: Metzger J, editor. Methods in Molecular Biology: Cardiac Cell & Gene Transfer. Vol. 219. Totowa, NJ: Humana Press; 2002. Chapter 14.

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Thesis

M.D. **del Monte F**. Valore predittivo del test con nitrati nella valutazione preoperatoria in pazienti con aneurisma ventriculare sinistro postinfartuale. Rome, Italy: University of Rome "La Sapienza"; 1986.

Cardiology Degree **del Monte F**. Propieta' contrattili di miociti isolati da cuori umani scompensati. Rome, Italy: University of Rome "La Sapienza"; 1990.

Ph.D. **del Monte F**. Cardiac failure and overload - Contractile changes in single myocytes. London, UK: Imperial College, University of London; 1996.

Trainees

PAST

Name	Type	Dates	Degree	Institution	Project	Current Position
Jang, Monica	HS Stud.	7-9/00-01		Phillips Exeter Academy, New Hampshire	Viral gene transfer	Mediacl student, MIT
Lamers, Frouke	Post Doc	1/7-31/98	MD	Erasmus University of Rotterdam	Adenoviral Vectors for gene transfer	Cardiology Fellow, Erasmus University of Rotterdam
Ranu, Hardeep	Post Doc	11/15/98-1/31/00	PhD	Imperial College School of Medicine	Na/Ca exchanger in Failing Hearts	Post-doctoral fellow, Renal Unit, MGH
Villa-Petroff, Martin	Visiting Scientist	2002	PhD	Centro de Investigaciones Cardiovasculares Facultad de Medicina, Argentina	Gene Transfer of Na/Ca in Failing Hearts	Staff Biologist, Centro de Investigaciones Cardiovasculares Facultad de Medicina, Argentina
Balderas, Isabel	Pre-Doc	2002	BS	Harvard Medical School	Tissue specific promoter following gene transfer	Medical Student, Harvard Medical School
Bernecker, Oliver	Post-Doc	2002	MD	University of Innsbruck	Tissue specific promoter following gene transfer	Cardiology Fellow (Austria)
Dalal, Rishikesh	Pre-Doc	2002-		Harvard University	Transcript Profiling following SERCA2a gene transfer	Medical Student,
Lebeche, Djemal	Post Doc	8/1/99-	Ph.D.	Boston University	Kv Channels & hypertrophy	Instructor in Medicine, Harvard Medical School
Huq, Fawzia	Post-Doc	4/2001	MD	University of Sidney	Gene Transfer in Failing Human Hearts	Geriatric Fellow
Sato, Taku	Post-Doc	2002-2003	PhD	Mitsubishi Pharma	Effect of MCC 135 on Failing Cardiomyocyte Contractility	Senior Scientist. Mitzubishi Pharma, Tokio
Tsuji, Tsuyoshi	Post Doc	9/2001	M.D.	Nara University	Measurement of Metabolic Parameters in heart failure	Clinical Fellow, Nara University. Japan
Susumu Sakata	Post-Doc	2001-2003	PhD	Nara University	Measurement of Metabolic Parameters in heart failure	Research Fellow, Nara University, Japan
Kevin Heist	Post Doc	2003	M.D.	Harvard University	In Vivo Transfer of CAMK	Assistant Professor in Medicine Harvard Medical School
Vaval Alan	Pre-Doc	2003		Penn University	SERCA2a and smooth muscle proliferation	Medical Student UPenn
Prabhu Padmanabhan	Pre-Doc	2003-2004	Master	Harvard University	ER stress and proteomics	Research Scientist
Joachim Chan	Medical student	2005	BS	Imperial College London –U.K.	Transcript profiling in amyloidotic cardiomyopathies	Medical Student Imperial College. London UK

PRESENT

Davide Gianni	PhD Student	2004-pesent	Master	Harvard University	Protein misfolding in heart failure	PhD Student
Maria Carles	Volunteer Fellow	2005-present	PhD	Gwathmey Inc	SERCA2a mutations in human heart failure	Vice President of Scientific Operation. Gwathmey Inc
Bernhard Kuhn	Mentee	2005-present	MD	Children Hospital	Periostin in myocardial regeneration	Instructor in Medicine
Michela Sluccca	Medical Student	2005		University of Bologna, Italy	Imaging of unstable plaque	Medical Student
Katrin Lindenberg	Post-doc	2005-present	PhD	Mass General Hospital	Cardiac function analysis in Huntinghton Disease	Post-Doc
Laura Borrelli	Res Assist	2005-present	BS	Mass General Hospital	Cardiac function in Altzheimer Disease	Research Assistant
Yhu-Shin Chang	Post-Doc	2006-present	PhD	Mass General Hospital	CHOP transcription factors	Post-Doc