Curriculum vitae

Fabio Anastasio Recchia, MD, PhD

Director
Institute of Clinical Physiology, Pisa,
National Research Council of Italy

Professor of Physiology
Scuola Superiore Sant'Anna, Pisa, Italy

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EDUCATION AND TRAINING

1990 Medical Degree
University of Bari, Bari, Italy

1990–1993 Residency in Anesthesiology & Intensive Care
University of Bari, Bari, Italy

1995–1996 Post-doctoral Research Fellowship
Division of Cardiology, Johns Hopkins University, Baltimore, MD, USA (Mentor: David A. Kass, M.D.)

1996–1998 Post-doctoral Research Fellowship
Department of Physiology, New York Medical College, Valhalla, NY, USA (Mentor: Thomas H. Hintze, Ph.D.)

1998 Ph.D. in Physiology
University of Torino, Turin, Italy

ACADEMIC POSITIONS

1999–2002	Assistant Professor of Physiology Department of Physiology, New York Medical College, Valhalla, NY, USA
2002–2016	Associate Professor of Physiology (part time/“a tempo definito”) Sector of Medicine, Scuola Superiore Sant’Anna, Pisa, Italy
2003–2009	Associate Professor of Physiology Department of Physiology, New York Medical College, Valhalla, NY, USA
2004–2014	Adjunct Researcher (“Associato”) of the National Council for Research at the Institute of Clinical Physiology, Pisa, Italy
2009–2012	Full Professor of Physiology Department of Physiology, New York Medical College, Valhalla, NY, USA
2012–2016	Full Professor of Physiology Department of Physiology, Lewis Katz School of Medicine at Temple University, Philadelphia, PA, USA
2017–Present	Adjunct Professor Department of Physiology, Lewis Katz School of Medicine at Temple University, Philadelphia, PA, USA
December 2016–Present	Full Professor of Physiology Institute of Life Sciences, Scuola Superiore Sant’Anna, Pisa, Italy

NON ACADEMIC WORK EXPERIENCES

1992–1993	M.D. Officer in the Air Force Bari, Italy

SERVICE:

Editorial Board Memberships:

- Member of the Editorial Board of the American Journal of Physiology, Heart and Circulation: 2002-2011
- Member of the Editorial Board of the Journal of Pharmacology and Experimental Therapeutics: 2007-2016
- Associate Editor, The American Journal of Physiology, Heart and Circulation: 01/2011-12/2020
- Member of the Editorial Board of the American Journal of Physiology, Heart and Circulation: 01/2021-

Other service:

- Director of the MD/PhD Program at the New York Medical College, NY: 2007-2012
- Coordinator of the PhD Program in “Translational Medicine” of the Scuola Superiore Sant’Anna, Pisa: 2012-2018
- Reviewer for: Circulation Research; Circulation; American Journal of Physiology; Life Sciences; Cardiovascular Research; Heart Failure Reviews; Journal of the American College of Cardiology; Journal of Clinical Investigation; Journal of Physiology; Journal of Cardiac Failure; Journal of Molecular and Cellular Cardiology; Journal of Pharmacology and Experimental Therapeutics; Thrombosis and Haemostasis
- Abstracts grader for the American Heart Association, Scientific Sessions: 2001-present
- Abstract grader for the European Society of cardiology: 2006-present
- Member of “Faculty of 1000-Biology”: 2005-2012
- Member of the AHA Peer Review Study Group, "North-East 1": 2001-2004
- Ad hoc reviewer for the Department of Veterans Affairs Office: 2002 and 2009 and 2019-present
- Member of the Animal Care and Use Committee at the New York Medical College: 2002-2006
- Ad hoc reviewer for Small Business Innovation Research Grants of the National Institute of Health: 11/2001 and 07/2002
- External Member of the Committee for the evaluation of intramural research proposals at the University of Padua, Italy: 2002-2016
- Ad hoc reviewer for the National Institutes of Health “Clinical and Integrative Cardiovascular Sciences (CICS)” study section: 2004-2005
- Temporary (2006-2007) and then regular member (2007-2011) of the National Institutes of Health “Cardiac Contractility, Hypertrophy and Failure (CCHF)” study section
- Scientific Coordinator of the Fondazione CNR-Toscana “G. Monasterio”, Pisa: 2008-2011
- Member of the Animal Care and Use Committee at the Temple University, Philadelphia: 2012-2016
- Reviewer for FIRB and PRIN, Italian Government: 2013-2015
- Ad hoc reviewer for the National Institutes of Health for R01, R15 and F30s grant applications: 2012-present

MENTORSHIP

In the USA, from 1999-to date: 16 Post-doctoral Fellows, 2 PhD students (one obtained his PhD in 2004 and the other one in 2018), 7 Master students and > 30 undergraduate student interns.

In Pisa, from 2003-to date: 5 Post-doctoral Fellows, 10 PhD students (5 obtained their PhD in 2007, 2009, 2011, 2017, 2019 and 2020)

HONOURS AND AWARDS

- Scholarship Award from “Ente Nazionale di Assistenza Magistrale” for students of Medicine: 1985-1987
- M.D. graduation cum Laude, Bari, Italy: 1990
- Scholarship Award from “Ente Nazionale di Assistenza Magistrale” for medical residents: 1991
- Scholarship Award for Research from the University of Bari, Italy: 1993-1994
- Fellowship Award from the American Heart Association, Maryland Affiliate: 1994-1996

- Prize of the Italian Physiological Society: 2000
- Established Investigator Award of the American Heart Association: 2007

RESEARCH GRANTS

In the United States

- 2000-2004. R01 from NIH: "Control of Metabolism by NO in the Failing Heart". $ 1.298.000. PI: F. Recchia
- 2000-2001. Contract from CV Therapeutics: "Effects of Ranolazine and of second generation RAN compounds on cardiac metabolism and function in an experimental model of transient myocardial ischemia": $ 199.000.
- 2001-2005. R01 from NIH: "NO and Metabolism in Pregnancy". $ 1.354.674. PI: T. Hintze. F. Recchia at 20% effort.
- 2002-2003. Contract from CV-Therapeutics: "Metabolic and functional effects of the second generation RAN compound CVT-4325 and of the A1 adenosine receptor agonist CVT-510 in normo- and hypo-perfused hearts". $ 160.000.
- 2002-2008. Program Project Grant (PPG) from NIH, Project 2: "Role of NO in heart failure". $ 1.470.003 PI: T. Hintze. F. Recchia at 20 % effort.
- 2003-2014. Program Project Grant (PPG) from NIH, Project 3: "Metabolic Phenotype Switch in Heart Failure". $ 1.482.439 (2003-2008) + $ 1.520.000 (2009-2014). PI of Project 3: F. Recchia
- 2007-2011. Established Investigator Award of the American Heart Association. $ 500.000
- 2012-2016. R01 from NIH: "VEGF receptor-1-mediated protection in dilated cardiomyopathy". $ 1.160.000 PI: F. Recchia
- 2015. Contract from Johnson&Johnson: “Effects of Acute Stresscopin Infusion on Cardiac Contractility in Dogs with Pacing-Induced Compensated Heart Failure”. $ 95.000.
- 2017-2020. R01 from NIH: “Follistatin-like protein 1 in cardiac and systemic metabolism”. $ 2.499.000. F. Recchia is Co-PI of this Multiple PI grant (F. Recchia/Kenneth Walsh)
- 2017-2018. Contract from Amgen: “New drugs for the treatment of atrial fibrillation” $ 256.532.
- 2018-2021. R01 from NIH: “BDNF TrkB- and beta-AR signals in ischemic and non-ischemic cardiomyopathy”. PI: Nazareno Paolocci, Johns Hopkins University. Sub-contract for F. Recchia: $ 648.00
- 2020-2024. R01 from NIH: “Targeting Ketone Metabolism as a Novel Heart Failure Therapy”. $ 2.499.995. F. Recchia is Co-PI of this Multiple PI grant (Daniel Kelly/F. Recchia)

In Italy

- 2005-2008. Research grant from the “Compagnia di San Paolo, Torino” “New

approaches of gene therapy for heart failure". 250.000 euro.

- 2006. Contract with Prassis-Sigma Tau, Milano: "Effects of PST2744 on the efficiency of the normal and failing heart". 34.500 euro
- 2007. Contract with Debiopharm, Lausanne, Switzerland: "Effects of Istaroxime on cardiac function and oxygen consumption in a pig model of heart failure". 65.000 euro
- 2010-2011. Endowment from the "Gruppo Intini-SMA" for the creation of a "Gruppo Intini-SMA Laboratory of Experimental Medicine": 260.000 euro
- 2013-2015. PRIN 2010-2011: "Nuove interazioni tra pathways molecolari favorenti un rimodellamento cardiaco adattativo nella cardiomiopatia da sovraccarico". 108.572 euro. PI of Unit 6: F. Recchia
- 2014-2015. Progetto SMAG - bando unico 2012 - Regione Toscana: "New methods for molecular imaging". Role: PI of the Unit Scuola Sant'Anna. 120.000 euro. PI: F. Recchia
- 2014-2017. Italian Ministry of Health: "CARDIOriGEN". 100.800 euro (Unit Scuola Sant'Anna). F. Recchia is the PI of the Unit Scuola Sant'Anna
- 2017-2020. Research Award from the "Fondazione Pisa": "An innovative therapy for inherited cardiomyopathies: experimental validation". 416.000 euro.
- 2020-2023. Research Award from the Tuscany Government: "Personalized Cardiac Repair (PERCARE)": 235.400 euro (Unit Scuola Sant'Anna). F. Recchia is the coordinator of this multiple Units project.
- 2020-2024. Grant Horizon 2020 from the European Commission: "Restoring cardiac mechanical function by polymeric artificial muscular tissue (REPAIR)". 457.500 euro (Unit Scuola Sant'Anna). F. Recchia is the PI of the Unit Scuola Sant'Anna.
- 2020-2024. Grant Horizon 2020 from the European Commission: "New-generation cardiac therapeutic strategies directed to the activation of endogenous regenerative mechanisms (REANIMA)". 400.000 euro (Unit Scuola Sant'Anna). F. Recchia is the PI of the Unit Scuola Sant'Anna.
- 2021-2023. Fondo Integrativo Speciale per la Ricerca (FISR) from the Italian Ministry of Research: "LEONARDO". 415.520 (Unit Scuola Sant'Anna). F. Recchia is the coordinator of this multiple Units project.
- 2021-2022. Contract with Maria Cecilia Hospital S.p.A: "Inibitori dell'apertura del permeability transition pore complex per il trattamento del danno da riperfusione nell'infarto del miocardio, in modello suino". 92.280 euro.

MEMBERSHIPS IN PROFESSIONAL SOCIETIES

- American Physiological Society: 2000-present
- Fellow of the American Heart Association (FAHA) for the "Council on Basic Cardiovascular Sciences": 2004-present
- Italian Physiological Society: 2011-present

INVITED TALKS AT CONGRESSES AND

- International Meeting on "Integrated Control of Pressure and blood flow in coronary microcirculation". Italian National Council for Research, Pisa, Italy, 2000.

ACADEMIC INSTITUTIONS (selected)

- International Congress of the European Society of Cardiology: Szeged, Hungary, 2002
- 59° Congress of the Italian Society of Anesthesia, Analgesia and Intensive Care., Bari, Italy, 2005
- Congress of the Heart Failure Association (European Society of Cardiology), Helsinki, Finland, June 2006
- World Congress of Cardiology 2006 (European Society of Cardiology), Barcelona, Spain, September 2006
- Satellite congress on "Heart Failure in Children", organized by the American Heart Association: Denver, Colorado, 2008
- Congress of the "Society for Heart and Vascular Metabolism": Boston, 2008
- Division of Cardiology of the Northwestern University, Chicago, 2009
- Division of Cardiology of the Mount Sinai Medical School, New York, 2009
- Department of Pharmacology of the Cornell Medical School, New York, 2009
- Congress of the "International Society for Heart Research (ISHR)": Baltimore, 2009
- Department of Cell Biology, University of Medicine and Dentistry of New Jersey, Newark, New Jersey, 2011
- International Congress "Frontiers in Cardiac and Vascular Regeneration", Trieste, 2012
- 2013 Cardiac Physiome Workshop, Bar Harbor, Maine, 2013
- Congress of the Society for Heart and Vascular Metabolism, Tromso, Norway, 2014
- Symposium and Training XXIII, University of Texas Southwestern, Dallas, 2015.
- Frontiers in CardioVascular Biomedicine (*FCVB*) of the European Society of Cardiology, Vienna, 2018
- Department of Cardiology of the Charité University Medicine, Berlin, 2019

Contribution to Science

My research work has been mostly centered on integrative cardiac physiology, pathophysiology, metabolism and experimental therapy, with particular regard to heart failure. Most of my studies have been performed in chronically instrumented, conscious dogs, a model widely recognized as physiologically and clinically relevant. Other studies have been performed in pigs assessed with magnetic resonance imaging and positron emission tomography. Four are the areas in which my contribution has been particularly significant and they can be summarized as follows:

1. **Identification of the role of endogenous and exogenous nitric oxide (NO) and vagal stimulation in the modulation of cardiac function and fatty acids and carbohydrate utilization.** Cardiac energy production depends entirely on the capacity of myocardium to oxidize substrates such as long-chain free fatty acids, lactate and glucose. My group was the first to identify the role played by endogenously produced NO, a major biological mediator, and by pharmacological NO donors, which are among the most utilized cardiovascular drugs, in the modulation of cardiac energy substrate utilization in normal and ischemic hearts, *in vivo*. NO can also be released in response to vagal stimulation:

Post H, d'Agostino C, Lionetti V, Castellari M, Kang EY, Altarejos M, Xu X, Hintze TH, Recchia FA. Reduced left ventricular compliance and mechanical efficiency after prolonged inhibition of NO synthesis in conscious dogs. *J Physiol*. 2003; 552:233-9.
Lei B, Matsuo K, Labinskyy V, Sharma N, Chandler MP, Ahn A, Hintze TH, Stanley WC, Recchia FA. Nitric oxide and cyclic GMP reduce glucose transporters translocation and lactate production in ischemic myocardium, in vivo. *Proc Natl Acad Sci USA*. 2005;102:6966-6971.
d'Agostino C., Labinskyy V, Lionetti V, Chandler MP, Lei B, Matsuo K, Bellomo M, Xu X, Hintze TH, Stanley WC, Recchia FA. Altered cardiac metabolic phenotype after prolonged inhibition of NO synthesis in chronically instrumented dogs. *Am J Physiol*. 2006; 290:H1721-6.

Vimercati C, Qanud K, Ilsar I, Mitacchione G, Sarnari R, Mania D, Faulk R, Stanley WC, Sabbah HN, Recchia FA. Acute vagal stimulation attenuates cardiac metabolic response to beta-adrenergic stress. 2012; *J Physiol*. 590:6065-74

2. **Characterization of the altered energy substrate metabolism in the failing heart**. It had been known for decades that the failing heart displays metabolic alterations, which likely contribute to its progressive functional derangement. However, no studies, prior to ours (early 2000s), had provided direct evidence of a shift to higher cardiac glucose oxidation and lower fatty acids oxidation in decompensated heart failure. We showed this profound metabolic alteration and identified some of the underling molecular mechanism both in large animal models (dogs and pigs) and in patients. We also provided initial evidence that, in the failing heart, the increased glucose utilization is associated with an upregulation of the pentose phosphate pathway, which can paradoxically fuel oxidative stress:

Osorio JC, Stanley WC, Linke A, Castellari M, Diep QN, Panchal AR, Hintze TH, Lopaschuk GD, Recchia FA. Impaired myocardial fatty acid oxidation and reduced protein expression of retinoid X receptor alpha in pacing-induced heart failure. *Circulation*. 2002; 106:606-612.
Gupte SA, Levine R, Young ME, Gupte RS, Lionetti V, Ojaimi C, Labinskyy V, Floyd B, Bellomo M, Wolin MS, Recchia FA. Glucose-6-phosphate dehydrogenase-derived NADPH fuels superoxide production in the failing heart. *J Mol Cell Cardiol*. 2006;41:340-9.
Neglia D, De Caterina A, Marraccini P, Natali A, Ciardetti M, Vecoli C, Gastaldelli A, Ciociaro D, Pellegrini P, Gorini I, Testa R, Menichetti L, L'Abbate A, Stanley WC, Recchia FA. Impaired myocardial metabolic reserve and substrate selection flexibility during stress in patients with idiopathic dilated cardiomyopathy. *Am J Physiol*. 2007;293:H3270-8. (accompanied by editorial)
Taegtmeyer H, Young ME, Lopaschuk GD, Abel ED, Brunengraber H, Darley-Usmar V, Des Rosiers C, Gerszten R, Glatz JF, Griffin JL, Gropler RJ, Holzhuetter HG, Kizer JR, Lewandowski ED, Malloy CR, Neubauer S, Peterson LR, Portman MA, Recchia FA, Van Eyk JE, Wang TJ; American Heart Association Council on Basic Cardiovascular Sciences. Assessing Cardiac Metabolism: A Scientific Statement from the American Heart Association. *Circ Res*. 2016;118:1659-701

3. **Characterization of the therapeutic effects of metabolic modulators on the failing heart.** Investigators have debated for years on whether the reduced free fatty acid oxidation and increased glucose oxidation represent an adaptive or a maladaptive change in the failing heart. This question has important clinical implications, since pharmacological metabolic modulators might prove efficacious for the treatment of heart failure, a major pathophysiological condition for which new therapies are absolutely needed. We performed a series of studies with metabolic modulators, showing how a direct action on fatty acid and/or glucose oxidative pathways leads to relevant changes in function and remodeling of the failing heart. In particular, we recently tested, for the first time, the beneficial effects of ketone body supply in a large animal model of heart failure:

Lionetti V, Linke A, Chandler MP, Young ME, Penn MS, Gupte S, d'Agostino C, Hintze TH, Stanley WC, Recchia FA. Carnitine palmitoyl transferase-I inhibition prevents ventricular remodeling and delays decompensation in pacing induced heart failure. *Cardiovasc Res*. 2005;66:454-461. (accompanied by editorial)
Mitacchione G, Powers J, Grifoni G, Woitek F, Lam A, Ly L, Settanni F, Makarewich C, McCormick R, Trovato L, Houser SR, Granata R, Recchia FA. The gut hormone ghrelin partially reverses energy substrate metabolic alterations in the failing heart. Circ Heart Fail. 2014 ;7:643-51.
Seki M, Powers JC, Maruyama S, Zuriaga MA, Wu CL, Kurishima C, Kim L, Johnson J, Poidomani A, Wang T, Muñoz E, Rajan S, Park JY, Walsh K, Recchia FA. Acute and Chronic Increases of Circulating FSTL1 Normalize Energy Substrate Metabolism in Pacing-Induced Heart Failure. *Circ Heart Fail*. 2018;11(1):e004486
Horton JL, Davidson MT, Kurishima C, Vega RB, Powers JC, Matsuura TR, Petucci C, Lewandowski ED*, Crawford PA*, Muoio DM*, Recchia FA*, Kelly DP*. The failing heart utilizes 3-hydroxybutyrate as a metabolic stress defense. JCI Insight. 2019;4(4). pii: 124079. (* co-senior authors)

4. **Gene transfer for the therapy of dilated cardiomyopathy and myocardial infarction.** My laboratory has been long interested in gene therapy as a valuable tool to explore in large animals, which are not routinely

genetically modified like mice, the pathophysiological role played by biological mediators. VEGF-B, a member of the vascular endothelial growth factor family, displays very interesting characteristics: despite its name, it does not exert pro-angiogenic actions, while it is a very powerful cytoprotective agent. By testing adeno-associated virus-mediated VEGF-B gene transfer, we found that it did not affect the function of normal hearts, whereas it exerted marked beneficial effects on the failing heart. In addition, in pig models of myocardial infarction, we tested the pro-regenerative effects of human placenta-derived mesenchymal stem and, very recently, newly discovered microRNAs that promote cardiac repair (to my knowledge, this is the only large animal study of experimental cardiology -that adds to another one of experimental cardiac surgery- ever published in Nature until 2019):

Pepe M, Mamdani M, Zentilin L, Csiszar A, Qanud K, Zacchigna S, Zoltan Ungvari Z, Puligadda U, Moimas S, Xu X, Edwards JG, Hintze TH, Giacca M, Recchia FA. Intramyocardial VEGF-B167 gene delivery delays the progression towards congestive failure in dogs with pacing-induced dilated cardiomyopathy. *Circ Res*. 2010;106:1893-903.
Simioniuc A, Campan M, Lionetti V, Marinelli M, Aquaro GD, Cavallini C, Valente S, Di Silvestre D, Cantoni S, Bernini F, Simi C, Pardini S, Mauri P, Neglia D, Ventura C, Pasquinelli G, Recchia FA. Placental stem cells pre-treated with a hyaluronan mixed ester of butyric and retinoic acid to cure infarcted pig hearts: a multimodal study. *Cardiovasc Res.* 2011;90:546-56.
Woitek F, Zentilin L, Hoffman NE, Powers JC, Ottiger I, Parikh S, Kulczycki AM, Hurst M, Ring N, Lam A, Wang T, Shahikh F, Gross P, Singh H, Kolpakov MA, Linke A, Houser SR, Rizzo V, Sabri A, Madesh M, Giacca M, Recchia FA. Intracoronary cytoprotective gene therapy: A study of VEGF-B167 in a pre-clinical animal model of dilated cardiomyopathy. *J Am Coll Cardiol.* 2015;66:139-153. (accompanied by editorial)
Gabisonia K, Prosdocimo ZG, Aquaro GD, Carlucci L, Zentilin L, Secco I, Ali H, Braga, L, Gorgodze N, Bernini F, Burchielli S, Collesi C, Zandonà L, Sinagra G, Piacenti M, Zacchigna S, Bussani R, Recchia FA, Giacca M. MicroRNA therapy stimulates uncontrolled cardiac repair after myocardial infarction in pigs. *Nature*. 2019; 569:418-422. (Recchia and Giacca co-senior authors)

FULL LIST OF PUBLICATIONS

Peer reviewed articles:

134 peer-reviewed articles from 1995 to date. H index = 48 and >7900 citations (Scopus). 7 articles were accompanied by editorials.

1. Giuliani R., L. Mascia, **F. Recchia**, A. Caracciolo, T. Fiore, V.M. Ranieri. Patient-ventilator interaction during synchronized intermittent mandatory ventilation. Effects of flow triggering. *Am. J. Respir. Crit. Care Med*. 1995; 151(1): 1-9.

2. Saeki A., **F. Recchia**, D.A. Kass. Systolic flow augmentation in hearts ejecting into a model of stiff aging vasculature. Influence of myocardial perfusion-demand balance. *Circ. Res*. 1995; 76: 132-141.

3. Saeki A., **F.A. Recchia**, H. Senzaki and D.A. Kass. Minimal role of nitric oxide in basal coronary flow regulation and cardiac energetics of blood-perfused isolated canine heart. *J. of Physiol. (London).* 1996; 491.2:455-463.

4. Kass D.A., A. Saeki, R.S. Tunin, **F.A. Recchia**. Adverse influence of systemic vascular stiffening on cardiac dysfunction and adaptation to acute coronary occlusion. *Circulation*. 1996; 93:1533-41.

5. **Recchia F.A.**, H. Senzaki, A. Saeki, B.J. Byrne, D.A. Kass. Pulse pressure-related changes in coronary flow in vivo are modulated by nitric oxide and adenosine*. Circ. Res*. 1996; 79:849-856.

6. **Recchia F.A.**, P.I. McConnell, R.D. Bernstein, T.R. Vogel, X.B. Xu, T.H. Hintze. Reduced nitric oxide production and altered myocardial metabolism during the decompensation of pacing-induced heart failure in the conscious dog. *Circ. Res*. 1998; 83:969-979. (with accompanying editorial)

7. Zhang XP., **F.A. Recchia,** R.D. Bernstein, X.B. Xu, A. Nasjletti, T.H. Hintze. Kinin-mediated coronary nitric

oxide production contributes to the therapeutic action of ACE , NEP inhibitors and amlodipine in the treatment of heart failure*. J. Pharmacol. Exper. Ther*. 1999; 288: 742-751.

8. **Recchia F.A**., Byrne J.B., Kass D.A. Sustained vessel dilation induced by increased pulsatile perfusion of porcine carotid arteries in vitro. *Acta Physiol Scan*. 1999; 166: 15-21.

9. **Recchia F.A.**, P.I. McConnell, K.E. Loke, X.B. Xu, M. Ochoa, T.H. Hintze. Nitric oxide controls cardiac substrate utilization in the conscious dog. *Cardiovasc. Res*. 1999; 44: 325-332.

10. Pagliaro P., H. Senzaki, N. Paolocci, T. Isoda, G. Sunagawa, **F.A. Recchia**, D.A. Kass. Specificity of synergistic coronary flow enhancement by adenosine and pulsatile perfusion in the dog. *J. of Physiol. (London).*1999; 520-1: 271-280.

11. **Recchia F.A.**, R.D. Bernstein, P.B. Sehgal, N.R. Ferreri, T.H. Hintze. Cytokines are not a requisite part of the pathophysiology leading to cardiac decompensation. *Proc Soc Exp Biol Med*. 2000; 223:47-52.

12. Loke K.E, P.R. Forfia, **F.A. Recchia**, X.B. Xu, J.C. Osorio, M. Ochoa, M. Gawryl, T.H. Hintze. Bovine polymerized hemoglobin increases cardiac oxygen consumption and alters myocardial substrate metabolism in conscious dogs: role of nitric oxide. *J. Cardiovas. Pharmacol*. 2000; 35: 84-92

13. Tada H., C.I. Thompson, **F.A. Recchia**, K.E. Loke, M. Ochoa, C.J. Smith, E.G. Shesely, G. Kaley, T.H. Hintze. Myocardial glucose uptake is regulated by nitric oxide via ecNOS in Langendorff mouse heart*. Circ. Res*. 2000; 86:270-274.

14. Recchia F.A., T.R. Vogel, T.H. Hintze. NO metabolites accumulate in erythrocytes in proportion to carbon dioxide and bicarbonate concentration. *Am. J. Physiol*. 2000; 279:H852-H856.

15. Peng X., **F.A. Recchia**, B.J. Byrne, I.S. Wittstein, R.C. Ziegelstein, D.A. Kass. In vitro system to study realistic pulsatile flow and stretch signaling in cultured vascular cells. *Am. J. Physiol*. 2000; 279: C797-C805.

16. Osorio J.C., **F.A. Recchia**. The role of nitric oxide in metabolism regulation: from basic sciences to the clinical setting. *Intensive Care Med*. 2000; 26: 1395-1398.

17. Sun D., A. Huang, **F.A. Recchia**, Y. Cui, E.J. Messina, A. Koller, G. Kaley. Nitric oxide-mediated arteriolar dilation after endothelial deformation. *Am. J. Physiol*. 2001; 280: H714-H721.

18. **Recchia F.A.** J.C. Osorio, M.P. Chandler, X. Xu, A.R. Panchal, G.D. Lopaschuk, T.H. Hintze, W.C. Stanley. Reduced synthesis of NO causes marked alterations in myocardial substrate metabolism in conscious dogs. *Am. J. Physiol*. 2002;282:E197-E206.

19. **Recchia F.A.** Role of nitric oxide in the regulation of substrate metabolism in heart failure. *Heart Fail Rev*. 2002; 7:141-8.

20. Osorio J.C., W.C. Stanley, A. Linke, M. Castellari, Q.N. Diep, A.R. Panchal, T.H. Hintze, G.D. Lopaschuk, **F.A. Recchia**. Impaired myocardial fatty acid oxidation and reduced protein expression of retinoid X receptor alpha in pacing-induced heart failure. *Circulation*.2002; 106:606-612.

21. Pagliaro P., A. Chiribiri, D. Gattullo, C. Penna, R. Rastaldo, **F.A. Recchia**. Inhibition of fatty acid utilization impairs Frank-Starling mechanism and Gregg effect, but not catecholamine response, in isolated rat hearts. *Acta Physiol. Scan*. 2002;176:167-176.

22. Linke A., **F. Recchia**, X. Zhang, T.H. Hintze. Acute and chronic endothelial dysfunction: implications for the development of heart failure. *Heart Fail Rev* 2003;8:87-97.

23. Linke A. G. Zhao, **F.A. Recchia**, J. Williams, X. Xu, T.H. Hintze. Shift in metabolic substrate uptake by the heart during development of alloxan-induced diabetes. *Am J Physiol.* 2003; 285:H1007-H1014.

24. Post H., J. Kajstura, B. Lei, W.C. Sessa, B.J. Byrne, P. Anversa, T. H. Hintze, **F.A. Recchia**. Adeno-associated virus mediated gene delivery into coronary microvessels of chronically instrumented dogs. *J. Appl. Physiol.* 2003; 95:1688-94.

25. Post H, D 'Agostino C, Lionetti V, Castellari M, Kang EY, Altarejos M, Xu X, Hintze TH, **Recchia FA**. Reduced left ventricular compliance and mechanical efficiency after prolonged inhibition of NO synthesis in conscious dogs. *J Physiol*. 2003; 552:233-9.

26. Trochu JN., S. Mital, XP. Zhang, X. Xu, M. Ochoa, J.K. Liao, **F.A. Recchia**, T.H. Hintze. Preservation of NO production by statins in the treatment of heart failure. *Cardiovasc. Res.* 2003; 60: 250-258. (with accompanying editorial)

27. Kinugawa S., H. Post, P.M. Kaminski, X. Zhang, X. Xu, H. Huang, **F.A. Recchia**, M. Ochoa, M.S. Wolin, G. Kaley, T.H. Hintze. Coronary microvascular endothelial stunning after acute pressure overload in the conscious dog is caused by oxidant processes: the role of angiotensin II type 1 receptor and NAD(P)H oxidase. *Circulation*. 2003; 108: 2934-2940.

28. Hintze TH, **Recchia FA**. Misplaced eNOS in the dystrophic myocardium: a good enzyme turns ugly. *J Mol Cell Cardiol*. 2004; 36:205-6.

29. Lei B, Lionetti V, Young ME, Chandler MP, D' Agostino C, Kang E, Altarejos M, Matsuo K, Hintze TH, Stanley WC, **Recchia FA**. Paradoxical downregulation of the glucose oxidation pathway despite enhanced flux in severe heart failure. *J Mol Cell Cardiol*. 2004;36:567-76.

30. Lionetti V, **Recchia FA**, Ranieri VM. Overview of ventilator-induced lung injury mechanisms. *Curr Opin Crit Care.* 2005;11:82-86.

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