
Subject: [EXT] Regarding Dr. Ribas's role in censoring an innovative platform for world-leading lung cancer research

Date: Tuesday, January 27, 2026 at 9:17:27 AM Pacific Standard Time

From: Raj Batra

To: Lana Moralez, David E. Jensen

Attachments: Foti-AACR letter 2019.pdf

CAUTION: This email originated from outside of CIRM.
Do not click links or open attachments unless you recognize the sender and know the content is safe.

Dear Ms Moralez;

This is a message to be shared with the CIRM Scientific Committee, and the public.

In response to the post by Mr Jensen regarding research prioritization by CIRM dated 1/27/2026, I would like to shed light on some history regarding the role played by Dr Ribas in blocking the advancement of an innovative platform for identifying and molecularly categorizing the most aggressive cancer cell endophenotypes in lung cancers, after the proof of concept was already published and well known to administrators at UClA, including Judy Gasson who still sits on the CIRM Board. These powerful and influential figures represent the most reprehensible of scientists in the world.

Please find attached a letter voicing my dissent to the AACR regarding the appointment of Dr Ribas as the president of that August body in 2019. I will also share the email and contents of my own standing from that time period. Those documents can be added to the public disclosure here.

I stand by the concerns I raised, and (still) wish that there was an independent investigation of the charges I raised before they were brushed aside.

As leaders of a profession, I suggest that Ribas's actions, and Gasson's omission of actions, directly led to the premature death of (by orders of magnitude) lung cancer patients than of advancing cures in melanomas since my seminal publication on the investigation of how to unravel the science of intratumoral heterogeneity in lung cancers in 2009.

Please find attached my disclosure to (Honorary) Doctor Foti, CEO of AACR regarding Dr Ribas' actions in matters of influence and ethical authority. Note that David Baltimore and Owen Witte's leadership roles in bending Ribas to their will is also reported-by Ribas' own account.

--
RK (Dani) Batra



Raj Batra <drrajbatra@gmail.com>

Re: Question via AACR Contact Us Form

RAJ BATRA <rbatra@ucla.edu>

Thu, Apr 11, 2019 at 3:50 PM

To: "Beveridge, Michael" <michael.beveridge@aacr.org>, margaret.foti@aacr.org

Cc: "Buck, Rick" <rick.buck@aacr.org>, Raj Batra <drrajbatra@gmail.com>

Dear Dr. Foti;

I am a career physician-scientist who entered my career to make a lasting impact on lung cancer, however who also took much too long to figure out that I was in an academic environment where it was preordained that I wouldn't be granted an opportunity to make that impact.

I will preface my statement with a personal definition of leadership, which in our field I take to mean to be an authoritative position of influence that is founded on content expertise AND character. The role of an organizational leader is to embody and to represent the best our field has to offer. I know from personal experience that although our AACR president-elect is a "nice guy" with a highly promoted name, he has neither the acumen nor character to represent our field, or the AACR.

I provided critical tutelage to Dr. Ribas in his matriculation to his current standing. I and a colleague at the VA in West Los Angeles were instrumental in developing the techniques for both culturing and then gene-modifying dendritic cells (using viral vectors) that led to earlier clinical trials conducted by Drs. Economou and Ribas for the experimental treatment of human melanoma. Grievances were filed with the UCLA academic senate in 2011-2012 (see attached) to complain that the real investigators at the core of that earlier advancement were not being given due credit by the institution (UCLA), and were not being compensated for their services by the institution. The minor role that Dr Ribas played in the narrative at that time was profiled on pages 8, 15, and 24 of the grievance. ***Those statements asserted that much of Dr. Ribas' institutionally promoted content expertise and scientific acumen was "borrowed", and not intrinsic to his person.***

But leadership also requires character. Character is defined by both acts of commission and omission. Not having the courage to speak up against actions one knows to be wrong is an act of omission that defines poor character.

I only came to question Dr. Ribas' character long after filing my formal grievance with the UCLA Academic Senate. Since 2005-2006, I had been independently trying to advance a research program to investigate intratumoral heterogeneity in lung cancer. I had come to learn that my former "mentor" was blocking the advancement of that program by corrupting peer review processes. By his own admission, Dr Ribas' played an active role in the corruption of funding processes that were in place to advance important science to tackle cancer.

Specifically, I was applying for funding to local, state and federal funding mechanisms for my own work. One of those applications was directed towards a seed mechanism through the UCLA-California Institute of Technology consortium. Dr. Ribas was appointed to be a senior reviewer for the allocation of those resources, and after my proposal was rejected, he informed me that although he felt that the theme was important and the investigator highly innovative and competent, he could not bring himself to go against Dr. Owen Witte who demanded that the research not be funded. Dr. David Baltimore sided with Dr. Witte's appraisal. ***Thus, after being appointed to positions of authority, Dr. Ribas displayed a lack of courage and flawed character by not having the courage for going against actions he personally knew to be wrong.***

Only time will tell if Dr. Ribas' acts of commission and omission contributed to a gross misrepresentation of his own scientific expertise, his misallocation of scarce resources, and/or his active blockade (alongside other UCLA and CIT heavyweights) of impactful medical science. Only time will tell whether my personal scientific ideas that were developed nearly 15 years ago, and first published a decade ago (at which time funding was blocked) will ever have an impact on the global landscape of the battle against cancer. But I have personally withstood great hardship to take a stand against the lack of integrity in the advancement of medical research in the fight against cancer, and for speaking out against systemic conflicts of interest. I also believe the AACR should not only stand by me, but to stand for much more than that. ***I believe that its leadership should better embody the mission statement of the organization.*** Unfortunately, many of the recent "leaders" promoted by the organization have not displayed the personal commitment to mission and character befitting their leadership position(s). The appointment of the current president-elect seemingly continues that trend.

Thank you for considering my statement.

With highest regards;

Raj K. Batra M.D.

p.s.: I am attaching my abridged CV and biosketch to assert that this statement is from someone who practices what he preaches.

[Quoted text hidden]

3 attachments

-  **Abridged CV-1-2019 (1).pdf**
599K
-  **BioPharma-cover letter.pdf**
149K
-  **Formal Charge Form-UCLA Academic Senate 5-1-2012.pdf**
152K

Subject: [EXT] Fwd: FW: Question via AACR Contact Us Form
Date: Tuesday, January 27, 2026 at 9:22:16 AM Pacific Standard Time
From: Raj Batra
To: David E. Jensen, Lana Moralez
Attachments: image629000.jpg, image824001.png, image645002.png, image535003.png, image517004.png, image847005.png, image863006.png, Abridged CV-1-2019 (1).pdf, BioPharma-cover letter.pdf, Formal Charge Form-UCLA Academic Senate 5-1-2012.pdf

CAUTION: This email originated from outside of CIRM.
Do not click links or open attachments unless you recognize the sender and know the content is safe.

This email and its attached contents are to be shared with the CIRM-Science Subcommittee to get a glimpse at (perhaps) the worst mistake in appraising the societal value impact of research-project and program content in the history of medical research.

And these are the same figures who CIRM places in positions to assess Merit today...

Raj K Batra MD

----- Forwarded message -----
From: [RAJ BATRA <rbatra@ucla.edu>](mailto:RAJ.BATRA@UCLA.EDU)
Date: Thu, Apr 11, 2019 at 3:51 PM
Subject: Re: FW: Question via AACR Contact Us Form
To: Beveridge, Michael <michael.beveridge@aacr.org>, <margaret.foti@aacr.org>
Cc: Buck, Rick <rick.buck@aacr.org>, Raj Batra <drrajbatra@gmail.com>

Dear Dr. Foti;

I am a career physician-scientist who entered my career to make a lasting impact on lung cancer, however who also took much too long to figure out that I was in an academic environment where it was preordained that I wouldn't be granted an opportunity to make that impact.

I will preface my statement with a personal definition of leadership, which in our field I take to mean to be an authoritative position of influence that is founded on content expertise AND character. The role of an organizational leader is to embody and to represent the best our field has to offer. I know from personal experience that although our AACR president-elect is a "nice guy" with a highly promoted name, he has neither the acumen nor character to represent our field, or the AACR.

I provided critical tutelage to Dr. Ribas in his matriculation to his current standing. I and a colleague at the VA in West Los Angeles were instrumental in developing the techniques for both culturing and then gene-modifying dendritic cells (using viral vectors) that led to earlier clinical trials conducted by Drs. Economou and Ribas for the experimental treatment of human melanoma. Grievances were filed with the UCLA academic senate in 2011-2012 (see attached) to complain that the real investigators at the core of that earlier advancement were not being given due credit by the institution (UCLA), and were not being compensated for their services by

the institution. The minor role that Dr Ribas played in the narrative at that time was profiled on pages 8, 15, and 24 of the grievance. ***Those statements asserted that much of Dr. Ribas' institutionally promoted content expertise and scientific acumen was "borrowed", and not intrinsic to his person.***

But leadership also requires character. Character is defined by both acts of commission and omission. Not having the courage to speak up against actions one knows to be wrong is an act of omission that defines poor character.

I only came to question Dr. Ribas' character long after filing my formal grievance with the UCLA Academic Senate. Since 2005-2006, I had been independently trying to advance a research program to investigate intratumoral heterogeneity in lung cancer. I had come to learn that my former "mentor" was blocking the advancement of that program by corrupting peer review processes. By his own admission, Dr Ribas' played an active role in the corruption of funding processes that were in place to advance important science to tackle cancer.

Specifically, I was applying for funding to local, state and federal funding mechanisms for my own work. One of those applications was directed towards a seed mechanism through the UCLA-California Institute of Technology consortium. Dr. Ribas was appointed to be a senior reviewer for the allocation of those resources, and after my proposal was rejected, he informed me that although he felt that the theme was important and the investigator highly innovative and competent, he could not bring himself to go against Dr. Owen Witte who demanded that the research not be funded. Dr. David Baltimore sided with Dr. Witte's appraisal. ***Thus, after being appointed to positions of authority, Dr. Ribas displayed a lack of courage and flawed character by not having the courage for going against actions he personally knew to be wrong.***

Only time will tell if Dr. Ribas' acts of commission and omission contributed to a gross misrepresentation of his own scientific expertise, his misallocation of scarce resources, and/or his active blockade (alongside other UCLA and CIT heavyweights) of impactful medical science. Only time will tell whether my personal scientific ideas that were developed nearly 15 years ago, and first published a decade ago (at which time funding was blocked) will ever have an impact on the global landscape of the battle against cancer. But I have personally withstood great hardship to take a stand against the lack of integrity in the advancement of medical research in the fight against cancer, and for speaking out against systemic conflicts of interest. I also believe the AACR should not only stand by me, but to stand for much more than that. ***I believe that its leadership should better embody the mission statement of the organization.*** Unfortunately, many of the recent "leaders" promoted by the organization have not displayed the personal commitment to mission and character befitting their leadership position(s). The appointment of the current president-elect seemingly continues that trend.

Thank you for considering my statement.

With highest regards;

Raj K. Batra M.D.

p.s.: I am attaching my abridged CV and biosketch to assert that this statement is from someone who practices what he preaches.

On Thu, Apr 11, 2019 at 6:30 AM Beveridge, Michael <michael.beveridge@aacr.org> wrote:

Dear Dr. Batra:

Thank you for your message. You are welcome to forward your concerns directly to the AACR Chief Executive Officer:

Margaret Foti, PhD, MD (hc)

AACR

615 Chestnut Street, 17th Floor

Philadelphia, PA 19106

margaret.foti@aacr.org

Sincerely,

Michael Beveridge

DIRECTOR, ELECTRONIC COMMUNICATIONS
Communications and Public Relations



American Association for Cancer Research
615 Chestnut Street, 17th Floor | Philadelphia, PA 19106-4404
215-440-9318 Direct | 267-765-1096 Fax
michael.beveridge@aacr.org | www.AACR.org



Title Raj K Batra

Full Name Raj K Batra

E-Mail Rbatra@ucla.edu

Request Type Other

Message Hi I have received notification about the confirmation/appointment of a new president elect. I have concerns from personal experience about both the acumen and character of the new president, and for historical purposes, seek to privately share those concerns with the executive committee of AACR. Who would be my contact for delicate information? Sincerely Raj K Batra MD.

--
RK (Dani) Batra

PERSONAL HISTORY

Raj K. Batra, MD, FCCP

241 S Lapeer Drive

Beverly Hills, CA 90211

Phone: 310-991-1592; e-mail: drrajbatra@gmail.com

Date and Place of Birth: February 4, 1962; India **Citizenship:** USA

Marital Status: Married, wife-Anshu; **Children:** Chanana, Keshav, and Arjun.



I am a translational physician scientist with expertise in lung cancer, and gene/cell based therapies. My working Philosophy is that in our time, ALL effective rationally derived therapeutics will emerge from signatures of disease across a spectrum of expressivity. The new role of Pharma will be to determine the reproducibility of those signatures across a spectrum of clinical diagnoses. This novel role will enable Pharma (and government) to rationally determine the therapeutic index/potential for pharmacotherapy of affected populations. That's the NEW MEDICINE of our time.

EDUCATION

8/1979-6/1983 Ohio State University, Columbus, Ohio, B.Sc.-Biology, Psychology

7/1983-8/1984 Ohio State University, Columbus, Ohio, Graduate Student-Physiology

8/1984-6/1988 University of Toledo College of Medicine & Life Sciences, Toledo, Ohio, MD-Medicine

PROFESSIONAL POSITIONS.

- 6/1988-6/1991 University of Michigan Hospitals, Ann Arbor, Michigan, Internal Medicine, Internship and Residency.
- 6/1991-6/1994 University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, Pulmonary Diseases & Critical Care Medicine, Clinical Fellowship.
- 6/1994-7/95 University of North Carolina at Chapel Hill, Chapel Hill, North Carolina. Pulmonary Diseases-Gene Therapy, Research Fellowship (Dr. DT Curiel & Dr. RC Boucher, Faculty Advisors).
- 7/1995-12/97 Research Instructor, Applications of Gene Therapy in Pulmonary Diseases & Critical Care Medicine, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina. (David T. Curiel and Richard C. Boucher, Mentors).
- 7/1995-12/97 Research Instructor, Multidisciplinary Thoracic Oncology Program, UNC at Chapel Hill, Chapel Hill, North Carolina. (Frank C Detterbeck, Director).
- 1/98-11/2017 Staff Physician; VAGLAHS (also known as WLA-VAMC or the Wadsworth VAMC), Physician-Scientist and VA-Advanced Research Career Development Awardee.
- 6/98-6/2005 Assistant Professor of Medicine and Staff Physician, Pulmonary Diseases & Critical Care Medicine, UCLA/VAGLAHS, Los Angeles, California.
- 6/2005-present Associate Professor of Medicine, Geffen School of Medicine at UCLA and Staff Physician, Pulmonary Diseases & Critical Care Medicine, VAGLAHS, Los Angeles, California. **Dr Batra has abstained from academic promotion requests** (2008 onward).
- 12/2017-present Locum Tenens. Pulmonary and Critical Care Medicine.

LICENSURE:

North Carolina License: 00-34248 California License: G 84851 Indiana License: 01079797A

DEA#: BB2438794, Medicare UPIN#: F06929 ; AMA-ME number: 03843880061; NPI#: 1841302122

BOARD CERTIFICATION

ABIM-Diplomate (137578): Internal Medicine (1991-2001)

ABIM-Diplomate (137578): Pulmonary Medicine (1994; 2005; 2018).

ABIM-Diplomate (137578): Critical Care Medicine (2006; 2018).

RESEARCH INNOVATIONS; PATENTS

2016 UCLA and VA Patent Disclosure: UC Case No. 2016-840, entitled “Discovery of common immunogenic targets in aggressive lung cancer.”

2016 UCLA and VA Patent Disclosure: UC Case No. 2016-803, entitled “PMR for cognitive gains in ASD”.

2015 UCLA and VA Patent Disclosure: UC Case No. 2015-960-1, entitled “Repurposing A1AT replacement therapy”.

2015 UCLA and VA Patent Disclosure: UC Case No. 2015-0615, entitled “A Conceptual Approach To Mediate Atherosclerotic Regression”.

2013 US Provisional Patent; Application number 61/919802; An Approach To Treat Anaerobic Gastroenteritis and To Restore Gut Epithelium

2013 UCLA and VA Patent Disclosure: UC Case No. 2014-411, entitled “An Approach To Treat Anaerobic Gastroenteritis and To Restore Gut Epithelium”.

2013 UCLA and VA Patent Disclosure: UC Case No. 2014-400, entitled “An adjuvant approach to utilize replication competent adenovirus to treat cancer”.

2013 PCT/US2013/050712 entitled Methods of Biomarker Validation and Target Discovery.
<http://patents.justia.com/patent/20150198583>; **Assignment pending.**

2011 US Provisional Patent; Application number 61/524731; Method for Biomarker Validation and Target Discovery in Cancer.

2011 UCLA and VA Patent Disclosure: UC Case No. 2012-171; Method for Biomarker Validation and Target Discovery in Cancer.

2009 <http://newsroom.ucla.edu/releases/ucla-cancer-researchers-develop-94307>

2006-2007 FDA-IND Filing/Approval: “A Phase I Trial of CCL-21 Gene Modified Dendritic Cells in Non-Small Cell Lung Cancer”. (The preparatory work for this submission took place between 1999-2007).

2006 NIH-OBA/RAC Appendix M Submission/Approval: “A Phase I Trial of CCL-21 Gene Modified Dendritic Cells in Non-Small Cell Lung Cancer”. (The preparatory work for this submission took place between 1999-2006).

2004 PCT Patent WO/2002/085286: Methods of using secondary lymphoid organ chemokine to modulate physiological processes in mammals (Application number: 20040175355; Filed: January 13, 2004; Issued: September 9, 2004; Assignee: UC Regents).

2003 PCT Patent WO/2002/085300: Methods of using interleukin-7 to modulate physiological processes in mammalian pulmonary fibroblasts (Application number: 20030175801 Filed: April 18, 2002; Issued: September 18, 2003; Assignee: UC Regents).

2003 VA Disclosure ID No. 03-175 -- “Secondary Lymphoid Tissue Chemokine for Cancer.”

2003 VA Disclosure ID No. 03-174 -- “Novel Cytokine-Based Therapy for Pulmonary Fibrosis.

2003 VA Disclosure ID No. 03-123 -- “Thyroperoxidase-Enhanced NIS Based Radioiodide Concentrator Gene Therapy in the Treatment of Human Cancer.”

2001 US Provisional Patent 60/284,845: Secondary Lymphoid Tissue Chemokine (SLC) Therapy for Cancer

2001 US Provisional Patent 60/286,257: Interleukin 7 inhibits both TGF β production and signaling in pulmonary fibrosis fibroblasts.

.

HONORS & AWARDS

Undergraduate

(Magna) Cum Laude, Phi Eta Sigma; Alpha Lambda Delta, Summa Award in Arts & Sciences.

Medical School

Alpha Omega Alpha Honor Society (Member #: 0065947).

Post-graduate

1994 Wyeth/Ayerst Critical Care Medicine Fellow travel award (SCCM).

1995 Glaxo (Allen and Hanburys) Pulmonary Fellow travel award (ATS).

1996-1997 Thomas Davis Fellowship (ALA-NC).

1997-1998 UNC Lineberger Comprehensive Cancer Center Translational Research Award

1998-2002 Veterans Administration Career Development Award

1999 ACCP/CHEST Foundation Leadership Development Program

2002-AACR/ASCO Workshop on Methods in Clinical Cancer Research

2003 ALA Career Investigator Award

2007 Los Angeles Super Doctor (*Tu Ciudad LA*; Pulmonary medicine; Peer Nominated)

2009 Maple Heights High School Pathfinders Award (A role model for students in an underprivileged school district).

2009 **Investigator spotlight:** <http://newsroom.ucla.edu/releases/ucla-cancer-researchers-develop-94307>

2011 Certificate of Excellence from Editors, *Laboratory Investigation*, for Quality of Reviews.

2011 Hind Rattan Award (For outstanding services and achievements by Indian expatriots).

2013 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

2014 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

2015 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

2015 India International Friendship Society (IIFS) Bharat Gaurav Award.

2015 Top Doctors, Los Angeles Magazine (LOSAMD115).

2015 **Investigator spotlight-VA-ORD;** “One researcher's vision: targeting tumors with multipronged treatment.” <http://www.research.va.gov/currents/winter2015/winter2015-19.cfm>

2016 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

2016 Top Doctors, Los Angeles Magazine (LOSAMD116).

2017 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

2018 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

2019 Southern California Super Doctor (Pulmonary medicine; Peer Nominated)

Personal Statement: I am a *translational physician-scientist* who began studying lung cancer over 20 years ago, with the explicit goal of eradicating the disease. My commitment to that endeavor has not wavered. Based on translational expertise in gene and cell based therapy that was developed at UNC, I was recruited to UCLA in 1998 with a VA-Advanced Career Development Award to build a translational experimental therapeutics program for lung cancer. I was awarded my first NIH-RO1 in 1999 to study viral gene transfer into lung cancer cells, while jointly working as the Chief Architect of the UCLA Translational Gene and Cell Based Therapy program for treating lung cancer. That program led to UCLA being awarded a NCI Specialized Program of Research Excellence (SPORE) grant in Lung Cancer, the development of a clinical grade vector by NIH-Rapid Assistance to Intervention Development (RAID), and regulatory approvals by the NIH/OBA and FDA for a Phase 1 clinical trial in 2007. Unfortunately, the “academic credit” and accrued institutional resources from this work were withheld towards career development and independence for this physician-scientist by the administrative Principal Investigator, an appointed University vice-Chancellor. The “academic politics” were compounded by an administrative blockade of access to University Collaborative and Technical Infrastructure, as well as extramural funding mechanisms. In 2011, the basis for this blockade was attributed to an administrative (re)interpretation that I was “not a University employee.” Effectively, what this meant was that I was **not eligible** to apply for California or (non-VA) Federal Medical Research Funding as a PI.

Nevertheless, our important scientific mission and goals have persevered.

In fact, key observations made in our pre-clinical studies on applications of viral gene therapy changed my entire frame of reference on lung cancer. I realized that gene therapy and Ad-virotherapy strategies can be quite effective if they are applied correctly for treating clinical disease. A key caveat that seemed to lead us astray in achieving translational potential was that our preclinical models were rather inadequate representations of disease *in situ*. By using inadequate models for proof-of-concept, we lost disease complexity, and consequently, predictability of effect. That realization prompted consideration that tumor heterogeneity confounded not only our gene therapy strategies, but possibly the effectiveness of all therapeutics. It made little sense to follow a *status quo* that we foresaw as unsuccessful; a different approach to study lung cancer needed to be developed.

Our work since 2006 is a result of pursuing that novel idea. Our recent publications provide key pilot feasibility data, and a philosophical foundation on which we are challenging existing paradigms. We have developed a rational discovery paradigm for identifying the key molecular drivers of aggressive tumor cell behaviors. We are desirous of implementing this strategy to develop biomarker-signatures of aggressive disease phenotypes, and to jointly identify candidate combinatorial targets for treating aggressive disease. I and members of my team seek funding and strategic investment for advancing this strategy to clinical implementation. To that end, we bring an innovative (world-leading), scientifically sound approach, and comprehensive “*bedside to bench to bedside*” capabilities to the ongoing study and rationally-derived targeted treatment of lung cancer.

A) PEER REVIEWED PUBLICATIONS:

1) **Batra RK**, S Agarwal, H Berschneider, & DT Curiel. Molecular conjugate vectors mediate efficient gene transfer into gastrointestinal epithelial cells. *Cancer Gene Therapy* 1(3), 1994, pp 185-192.

This article is a key concept and process-related cross-reference in the US Patent Applications 5292662; 5328470; 5643579; 5681744; 5786340; 5821235, and Foreign Patent Applications WO 90/11092; WO 93/03769; WO 93/19660; WO 94/25608; WO 96/40081. These applications resulted in the granted US patent 6225290 (Assigned to the Regents of the University of California).

2) **Batra RK**, F Wong-Johanning, E Wagner, RI Garver, and DT Curiel. Receptor-mediated gene delivery employing lectin-binding specificity. *Gene Therapy* 1(4), 1994, pp 255-260.

This was the first demonstration that lectins targeting cell surface receptors could effectively and selectively mediate gene transfer into target cells. I conceived the idea; Dr Curiel provided the reagents and experimental design, which I implemented. Molecular conjugates (now called nanoparticles) that used lectins as targeting ligands were developed and used to transfect target cells. Study provided proof-of-concept that if cells have a specific lectin-binding signature, then those lectins could be used for enabling gene transfer into those cells.

This article is a key concept and process-related cross-reference in the US Patent Application 6569450 (granted patent, May 2003; assigned to Chiron Corporation).

This article is a key concept and process-related cross-reference in the US Patent Application 6869606 (patent issued 2005, Millenium Pharmaceuticals)

This article is a key concept and process-related cross-reference in the US Patent Application 7214384 (Assigned to Novartis Corp.).

This article is a key concept and process-related cross-reference in the US Patent Application 20110275585 A1 (Assigned to Engeneic Molecular Delivery Pty Ltd.).

This article is a key concept and process-related cross-reference in the US Patent Applications US8669101B2, US8735566, US8956864, US8591862, US8691963 (Assigned to Engeneic Molecular Delivery).

3) Hoganson DK, **RK Batra**, JC Olsen, and RC Boucher. Comparison of the effects of three different toxin genes and their levels of expression on cell growth and bystander effect in lung adenocarcinoma. *Cancer Research* 56, 3/15/1996, pp 1315-1323.

4) **Batra RK**, JC Olsen, DK Hoganson, B Caterson, and RC Boucher. Retroviral gene transfer is inhibited by chondroitin sulfate proteoglycans/ glycosaminoglycans in malignant pleural effusions. *J. Biol. Chem.* 272 (18), May 1997, pp 11736-11743. (PMC 1626586).
<http://www.jbc.org/content/272/18/11736.full.pdf+html>.

*This was the first demonstration that there are soluble inhibitors of (amphotropic and VSVg-pseudotyped) retroviral gene transfer *in situ*. Here, we utilized primary human tissues (malignant pleural effusions or MPE) to identify clinically relevant obstacles to viral gene therapy. I developed the methods to fractionate and primarily culture these biospecimens, and to quantify relevant inhibitory biochemical-components (proteoglycans and glycosaminoglycans) within the MPE-fluid. This discovery provided pioneering insight into unforeseen translational obstacles for clinical applications of viral gene therapy, and set the methodological foundation for (MPE-primary cultures to investigate intratumoral heterogeneity) studies I would conduct 10 years later.*

*Article is a key cross reference regarding the tumor microenvironment *in situ* in PCT/US2013/050712 entitled Methods of Biomarker Validation and Target Discovery filed 2013.*

5) **Batra RK**, DK Hoganson, R Pickels, JC Olsen, and RC Boucher. Transduction of non-small cell lung cancer cells by adenoviral and retroviral vectors. *Am J Respir Cell Mol Biol* 18/3, March 1998, pp 402-410. PMID: 9490658. <http://www.atsjournals.org/doi/pdf/10.1165/ajrcmb.18.3.2784>.

*This was the **first demonstration** that gene therapy for lung cancer using viral vectors may require either disease-specific or cancer-specific vectors (study surmised that **inter-tumoral** heterogeneity would confound the use of a specific viral vector for gene therapy). This work was pioneering in that it first determined that the differences in gene transfer by viral vectors could largely be explained by **pharmacodynamic differences** in the expression of cellular receptors mediating Adenoviral binding (later designated the coxsackievirus-adenovirus receptor or CAR). We later went on to describe a role for CAR-adhesion in critically mediating xenoengraftment efficiency **in vivo** (2004), and recognized it as a key marker of de-differentiated (mesenchymal-type; stem-like) lung cancer cells (2009).*

This article is a key concept and process-related cross-reference in the US Patent Application 7968332 (issued 2011; assigned to Institut Pasteur).

6) Hoganson DK, H Matsui, **RK Batra**, and RC Boucher. Toxin gene mediated growth inhibition of lung adenocarcinoma in an animal model of pleural malignancy. *Human Gene Therapy* 9(8), May 1998, pp 1143-1156.

7) **Batra RK**, DC Guttridge, DA Brenner, AS Baldwin, SM Dubinett, and RC Boucher. IkB α gene transfer is cytotoxic to squamous cell lung cancer cells and sensitizes them to TNF α mediated cell death. *Am J Respir Cell Mol Biol* 21/2, August 1999, pp 238-245.
<http://www.atsjournals.org/doi/pdf/10.1165/ajrcmb.21.2.3470>.

This article is a key process-related cross-reference in the US Patent Application 09/927,091 (Assigned to Board of Regents, University of Texas), and in US Patent application 2003/0108920 A1.

8) Miller PW, S Sharma, M Stolina, L Butterfield, J Luo, Y Lin, M Dohadwala, **RK Batra**, L Wu, JS Economou, and SM Dubinett. Intratumoral administration of adenoviral interleukin-7 gene-modified dendritic cells augments specific antitumor immunity and achieves tumor eradication. *Human Gene Therapy* 11(1), January 2000, pp 53-65.

9) **Batra RK**, SM Dubinett, B Henkle, S Sharma, and B Gardner. Adenoviral gene transfer is inhibited by soluble factors in malignant pleural effusions. *Am J Respir Cell Mol Biol*. May, 2000, 22 (5): 613-619. <http://www.atsjournals.org/doi/pdf/10.1165/ajrcmb.22.5.3970>.

10) S Sharma, M Stolina, J Luo, RM Strieter, M Burdick, LX Zhu, **RK Batra**, and SM Dubinett. Secondary Lymphoid Tissue Chemokine (6Ckine) mediates T cell-dependent anti-tumor responses **In vivo**. *J. Immunology* May, 2000; 164 (9):4558-4563.
<http://www.jimmunol.org/content/164/9/4558.full.pdf+html>.

*This was the **first demonstration** to document the anti-tumor efficacy of slc (CCL-21), an endothelial-derived CC chemokine, in a lung cancer model. This work, along with my personal recognition that **in situ** viral gene transfer still had obstacles to overcome, spawned the **first-in-human** clinical immuno-genetic therapy trial for targeting lung cancer worldwide (at UCLA). Instead of delivering the chemokine directly into the tumor bed, we decided to use autologous gene-modified dendritic cells to carry the slc into the tumor bed.*

Article provides initial proof-of-concept for therapeutic applications for PCT Patent WO/2002/085286: Methods of using secondary lymphoid organ chemokine to modulate physiological processes in mammals (Application number: 20040175355; Filed: January 13, 2004; Issued: September 9, 2004; Assignee: UC Regents).

11) Dohadwala M, J Luo, LX Zhu, YQ Lin, GJ Dougherty, S Sharma, M Huang, M Pold, **RK Batra**, and SM Dubinett. Non-Small Cell Lung Cancer COX-2-dependent Invasion is mediated by CD44. *J. Biol. Chem.*, 276 (24), June, 2001, pp: 20809-20812. (PMC1471882).
<http://www.jbc.org/content/276/24/20809.full.pdf+html>.

This work described a candidate molecular mechanism (upregulation of surface CD44) by which COX-2 expression by lung cancer cells contributed to a lethal malignant phenotype (invasion/metastasis). I would later come back to using CD44 as a marker for selecting aggressive “cancer stem cell” phenotypes in primary MPE-cultures.

12) Huang M, **RK Batra**, T Kogai, YQ. Lin, JM Hershman, A Lichtenstein, S Sharma, LX Zhu, GA Brent, and SM Dubinett. Ectopic expression of the thyroperoxidase gene augments radioiodide uptake and retention mediated by the sodium iodide symporter in non-small cell lung cancer. *Cancer Gene Therapy* (8), August 2001, pp. 612-61. (PMC 1471880).
<http://www.nature.com/cgt/journal/v8/n8/pdf/7700354a.pdf>.

This was the first demonstration that radioiodine uptake via the sodium-iodide symporter required the thyroperoxidase gene product to enable intracellular retention of the therapeutic radio-iodine for killing cancer cells. I conceived the idea and developed the experimental design (with Dr. Hershman’s insight), but the project was funded for a “mentee” (Dr. Huang), and was subsequently reassigned by the lab director. This strategy remains a viable treatment option, if applied optimally.

13) Sharma S, M Stolina, L Zhu, Y Lin, **RK Batra**, M Huang, R Strieter, and SM Dubinett. SLC Reduces Pulmonary Tumor Burden in Spontaneous Murine Bronchoalveolar Cell Carcinoma. *Cancer Research* (61), September 2001, pp: 6406-6412.

Article extended proof-of-concept for therapeutic applications for PCT Patent WO/2002/085286: Methods of using secondary lymphoid organ chemokine to modulate physiological processes in mammals (Application number: 20040175355; Filed: January 13, 2004; Issued: September 9, 2004; Assignee: UC Regents).

14) M Huang, S Sharma, LX Zhu, MP Keane, J Luo, L Zhang, MD Burdick, YQ Lin, M Dohadwala, B Gardner, **RK Batra**, RM Strieter, and SM Dubinett. Interleukin-7 inhibits TGF- β production and signaling in pulmonary fibrosis. *J Clin Invest.* April 2002; 109(7):931-937. (PMC150933).
<http://www.jci.org/articles/view/14685/pdf>.

*This work demonstrated that IL-7 inhibited both TGF- β production, and signaling (via JAK1/STAT1) in pulmonary fibroblasts. The IL-7-mediated inhibition of TGF- β activity was associated with an increase in Smad7. Recombinant IL-7 decreased bleomycin-induced pulmonary fibrosis *in vivo*, providing a pre-clinical rationale for its potential use for the treatment of pulmonary fibrosis.*

Article provides initial proof-of-concept for therapeutic applications for PCT Patent WO/2002/085300: Methods of using interleukin-7 to modulate physiological processes in mammalian pulmonary fibroblasts (Application number: 20030175801 Filed: April 18, 2002; Issued: September 18, 2003; Assignee: UC Regents).

15) Bernal RM, S Sharma, BK Gardner, JT Douglas, JM Bergelson, SM Dubinett, and **RK Batra**. Soluble Coxsackievirus Adenovirus Receptor is a putative inhibitor of adenoviral gene transfer in the tumor

environment. *Clinical Cancer Research* (8), June 2002, 1915-1923.
<http://clincancerres.aacrjournals.org/content/8/6/1915.full.pdf+html>.

16) Dohadwala M, **RK Batra**, J Luo, Y Lin, K Krysan, M Pold, S Sharma, SM Dubinett. Autocrine/paracrine PGE2 production by Non-small cell lung cancer cells regulates MMP-2 and CD44 in COX-2- dependent invasion. *J Biol Chem*. December 2002; 277(52):50828-33. (PMC 1471886)
<http://www.jbc.org/content/277/52/50828.full.pdf+html>.

17) **Batra RK**, Y Lin, S Sharma, M Dohadwala, J Luo, M Pold, and SM Dubinett. Non small cell lung cancer derived soluble mediators enhance apoptosis in activated T lymphocytes through an I κ B-Kinase dependent mechanism. *Cancer Research* February 2003; 63(3):642-6. PMID:12566308
<http://cancerres.aacrjournals.org/content/63/3/642.full.pdf+html>.

18) Heuze-Vourc'h N, L Zhu, K Krysan, **RK Batra**, S Sharma, and SM Dubinett. Abnormal Interleukin 10R{alpha} Expression Contributes to the Maintenance of Elevated Cyclooxygenase-2 in Non-Small Cell Lung Cancer Cells. *Cancer Research* 2003; February 2003, 63(4) 766-70.

19) Sharma S, M Stolina, SC Yang, F Baratelli, JF Lin, K Atianzar, J Luo, L Zhu, Y Lin, M Huang, M Dohadwala, **RK Batra**, and SM Dubinett. Tumor Cyclooxygenase 2-dependent suppression of Dendritic Cell Function. *Clinical Cancer Research* (9), March 2003: 961-968.

20) Sharma S, SC Yang, S Hillinger, LX Zhu, M Huang, **RK Batra**, JF Lin, MD Burdick, RM Strieter, SM Dubinett. SLC/CCL21-mediated anti-tumor responses require IFN-gamma, MIG/CXCL9 and IP-10/CXCL10. *Molecular Cancer*. April 2003; 2(1): 22. (PMC 155639).

21) Qin M, S Chen, T Yu, B Escudero, S Sharma, and **RK Batra**. CAR-expression predicts the efficiency of adenoviral gene transfer into NSCLC-xenografts. *Clinical Cancer Research*. Oct 15;9(13):4992-9. 2003. PMID: 14581374
<http://clincancerres.aacrjournals.org/content/9/13/4992.full.pdf+html>.

22) Sharma S, **RK Batra**, SC Yang, S Hillinger, L Zhu, K Atianzar, RM Strieter, K Riedl, M Huang, SM Dubinett. Interleukin-7 gene-modified dendritic cells reduce pulmonary tumor burden in spontaneous murine bronchoalveolar cell carcinoma. *Human Gene Therapy* 2003 Nov 1; 14(16):1511-24. (PMC1471881).

23) Riedl K, F Baratelli, **RK Batra**, SC Yang, J Luo, B Escudero, R Figlin, RM Strieter, S Sharma, SM Dubinett. Overexpression of CCL-21/Secondary Lymphoid Tissue Chemokine in Human Dendritic Cells Augments Chemotactic Activities for Lymphocytes and Antigen Presenting Cells. *Molecular Cancer*. 2003 Nov 2; 2(1):35. (PMC 270078).

24) Hillinger S, SC Yang, L Zhu, M Huang, R Duckett, K Atianzar, **RK Batra**, RM Strieter, SM Dubinett, S Sharma. EBV-induced molecule 1 ligand chemokine (ELC/CCL19) promotes IFN-gamma-dependent antitumor responses in a lung cancer model. *J Immunol*. 2003 Dec 15; 171(12):6457-65.

25) Pold M, LX Zhu, S Sharma, MD Burdick, Y Lin, PP Lee, A Pold, J Luo, K Krysan, M Dohadwala, JT Mao, **RK Batra**, RM Strieter, SM Dubinett. Cyclooxygenase-2-dependent expression of angiogenic CXC chemokines ENA-78/CXC Ligand (CXCL) 5 and interleukin-8/CXCL8 in human non-small cell lung cancer. *Cancer Res*. 2004 Mar 1; 64(5): 1853-60.

26) Yang SC, S Hillinger, K Riedl, L Zhang, L Zhu, M Huang, K Atianzar, BY Kuo, B Gardner, **RK Batra**, RM Strieter, SM Dubinett, S Sharma. Intratumoral administration of dendritic cells over-

expressing CCL21 generates systemic antitumor responses and immunity: the importance of IFNg, MIG/CXCL9 and IP-10/CXCL10. *Clinical Cancer Res.* 2004 Apr 15; 10(8): 2891-901

27) Qin M, B Escudero, M Dohadwala, S. Sharma, and **RK Batra**. A novel role for the Coxsackie-Adenovirus Receptor in mediating tumor formation by lung cancer cells. *Cancer Res.* 2004 Sep 15; 64(18):6377-80.(PMID:15374942).

<http://cancerres.aacrjournals.org/content/64/18/6377.full.pdf+html>.

*This was the **first demonstration** that the Coxsackie-Adenoviral Receptor is critical for the efficiency of xenotransplantation by lung cancer cells that constitutively **highly express** this surface adhesion molecule. This demonstration has widespread implications regarding the (patho-) physiological role of CAR (see 2009 *Lab Invest* publication), as well as for anti-tumor Adenoviral Gene Therapy/Virotherapy applications, and the pathogenesis of wildtype subtype C Adenoviral infections of the human airway epithelium.*

28) Qin M, B Escudero, S Sharma, and **RK Batra**. Gene transfer mediated by native versus FGF2-retargeted adenoviral vectors into lung cancer cells. *Am J Respir Cell Mol Biol.* 2005 Mar; 32(3):211-217. (PMID: 15626775)

29) Sharma S, SC Yang, L Zhu, K Reckamp, B Gardner, F Baratelli, M Huang, **RK Batra**, SM Dubinett. Tumor cyclooxygenase-2/prostaglandin E2-dependent promotion of FOXP3 expression and CD4+ CD25+ T regulatory cell activities in lung cancer. *Cancer Res.* 2005 Jun 15; 65(12):5211-20. (PMID:15958566).

30) Sharma S, L Zhu, SC Yang, L Zhang, J Lin, S Hillinger, B Gardner, K Reckamp, RM. Strieter, M Huang, **RK Batra**, SM Dubinett. Cyclooxygenase 2 Inhibition Promotes IFN-{gamma}-Dependent Enhancement of Antitumor Responses. *J Immunol.* 2005 Jul 15;175(2):813-9. (PMID: 16002678).

31) Yang SC, **RK Batra**, S Hillinger, KL Reckamp, RM. Strieter, SM. Dubinett, and S Sharma. Intrapulmonary Administration of CCL21 Gene-Modified Dendritic Cells Reduces Tumor Burden in Spontaneous Murine Bronchoalveolar Cell Carcinoma. *Cancer Res.* 2006, 66: 3205-3213. (PMID: 16540672).

32) Hillinger S, SC Yang, **RK Batra**, RM Strieter, W Weder, SM Dubinett, S Sharma. CCL19 reduces tumor burden in a model of advanced lung cancer. *Br J Cancer.* 2006 Apr 10; 94(7):1029-34. (PMC 2361223).

33) Dohadwala M, SC Yang, J Luo, S Sharma, **RK Batra**, M Huang, Y Lin, L Goodlick, K Krysan, MC Fishbein, L Hong, C Lai, RB Cameron, RM Gemmill, HA Drabkin, SM Dubinett. Cyclooxygenase-2-Dependent Regulation of E-Cadherin: Prostaglandin E2 Induces Transcriptional Repressors ZEB1 and Snail in Non-Small Cell Lung Cancer. *Cancer Res.* 2006 May 15; 66(10): 5338-45. (PMID: 16707460).

34) Hazra S, **RK Batra**, HH Tai, S Sharma S, X Cui, SM Dubinett. Pioglitazone and Rosiglitazone decrease PGE2 in non-small cell lung cancer cells by upregulating 15-hydroxyprostaglandin dehydrogenase. *Mol Pharmacol.* 2007 Jun; 71(6):1715-20. (PMID: 17412838).

35) Baratelli F, H Takedatsu, S Hazra, K Peebles, J Luo, PS Kurimoto, G Zeng, **RK Batra**, S Sharma, SM Dubinett, JM Lee. Pre-clinical characterization of GMP grade CCL21-gene modified dendritic cells for application in a phase I trial in Non- Small Cell Lung Cancer. *J Translational Med.* 2008 Jul 22; 6(1):38. (PMC2507704).

36) Andersson A, SC Yang, M Huang, L Zhu, UK Kar, **RK Batra**, D Elashoff, RM Strieter, SM Dubinett, and S Sharma. IL-7 Promotes CXCR3 Ligand-Dependent T Cell Antitumor Reactivity in Lung Cancer. *J Immunol*. 2009 Jun 1; 182(11):6951-8. (PMID: 19454692).

37) Basak SK, MS Veena, S Oh, G Huang, E Srivatsan, M Huang, S Sharma, and **RK Batra**. The malignant pleural effusion as a model to investigate intratumoral heterogeneity in lung cancer. *PLoS ONE*. 2009, Jun 12; 4(6):e5884. (PMC2697051).
<http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0005884>
<http://newsroom.ucla.edu/releases/ucla-cancer-researchers-develop-94307>

This work is our most important innovation to thought process regarding a disease-perspective to date. Based on past experience, we surmised that using cancer stem cell biomarkers, we could extract behaviorally-distinct aggressive cancer (stem) cell phenotypes directly from MPE-primary cultures. That process would directly enable us to efficiently determine the comprehensive biological basis for behaviorally-lethal "subclones" (or heterotypic conglomerates) in individual tumors. The paper provides the rationale and detailed methods for reliably establishing MPE-primary cultures to culture CSC directly from patient biospecimens, and founds the platform for our Patent application 050712: Methods of Biomarker Validation and Target Discovery.

Article provides reduction of art to practice for PCT/US2013/050712 entitled Methods of Biomarker Validation and Target Discovery filed 2013.

38) Veena MS, M Qin, A Andersson, S Sharma, and **RK Batra**. CAR mediates efficient tumor engraftment of mesenchymal type lung cancer cells. *Lab. Invest.* 2009 Aug; 89(8):875-86. (PMID: 19506548). <http://www.nature.com/labinvest/journal/v89/n8/full/labinvest200956a.html>.

39) Limsukon A, I Susanto, G Soo Hoo, SM Dubinett, and **RK Batra**. Regression of recurrent respiratory papillomatosis with celecoxib and erlotinib combination therapy. *CHEST* 2009 Sept. (136): 924-926. (PMID: 19736197). <http://journal.publications.chestnet.org/article.aspx?articleid=1090050>.

This case report describes the first-in-human demonstration of a rationally derived, clinically effective combination therapy for HPV-induced RRP. A rational combination therapy was devised to extend the life of a patient who was moribund from progressive airway occlusion due to HPV-mediated recurrent respiratory papillomatosis. We sought and received P&T committee approval to try combination COX-2/EGFr-inhibition for compassionate use, reasoning that HPV-infected cells and papilloma tissues highly expressed these targets. Unfortunately, two and a half years after successful therapy, the patient felt well enough to go on a drinking binge with buddies, passed out, and occluded his tracheostomy with his submental pannus. Small consolation was that the trachea and airways were free of papillomas on coroner's autopsy. Nevertheless, this strategy is ripe for more widespread clinical testing for refractory HPV-associated disease of the airways.

40) **Batra RK** and D Warburton. On the derivation and clinical implications of "driver" mutations in lung cancer. *Am J Resp. and Critical Care Med.* 2010 July (182). pp. 4-5, (Invited Editorial). (PMID:20463175) <http://www.atsjournals.org/doi/pdf/10.1164/rccm.201003-0381ED>.

41) Frey MR, Carraro G, **Batra RK**, Polk DB, Warburton D. Sprouty keeps bowel kinases regular in colon cancer, while miR-21 targets Sprouty. *Cancer Biol Ther*. 2011 Jan 1; 11(1):112-114. (PMID: 21124074).

42) Darshni Vira, SK. Basak, MS. Veena, MB. Wang, **RK. Batra**^{*}, and ES Srivatsan^{*} Cancer stem cells, microRNAs, and therapeutic strategies, including natural products. *Cancer and Metastases Reviews*. (*Co-corresponding author). 2012 Dec; 31(3-4):733-51. (PMID: 22752409)

43) **Batra RK**, Soo Hoo GW. Reprogrammed cells for respiratory papillomatosis (Letter). *N Engl J Med*. 2012, Dec 27; 367(26):2553-4. (PMID: 23268676).

44) Basak SK, MS Veena, S Oh, C Lai, S Vangala, D Elashoff, MC Fishbein, S Sharma, NP Rao, D Rao, R Phan, ES Srivatsan, and **RK Batra**. The CD44^{high} tumorigenic subsets in lung cancer biospecimens are enriched for low miR-34a expression. *PLoS One*. 2013 Sep 3; 8(9):e73195. (PMC3790883). <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0073195>

This work is a direct extension of the 2009 PLoS-ONE publication, and directly demonstrates the immense clinical-translational potential of a phenotype based-approach to the discovery of critical therapeutic targets in lung cancer. We show that highly tumorigenic CD44^{hi} "cancer stem cells" can be reliably extracted from MPE-primary cultures, and can be behaviorally validated for a specific malignant (high tumorigenic potential) phenotype. Moreover, using a candidate target discovery approach, we can directly identify and validate a (predicted) high value/ clinically-relevant target (miR34a). This paper extends the rationale for our Patent application 050712: Methods of Biomarker Validation and Target Discovery.

Article extends proof-of-concept for specific target discovery for PCT/US2013/050712 entitled Methods of Biomarker Validation and Target Discovery filed 2013, and identifies miR34a as a critical high value target.

45) Veena MS, R Wilken, JY Zheng, A Ghokar, D Vira, S Ahmed, SK Basak, **RK Batra**, N Kasahara, D Elashoff, MC Fishbein, JP Whitelegge, JZ Torres, MB Wang, ES Srivatsan. p16 recruits gigaxonin for NF κ B ubiquitination in cisplatin induced senescence. *J Biol Chem*. 2014, Dec 12; 289(50):34921-37. [Epub ahead of print Oct 20]. (PMID: 25331947).

46) **Batra RK**, S. Oh and SK Basak. The isolation and molecular characterization of cancer stem cells (aggressive endophenotypes) in individual lung cancers. *IORE Journal of Cancer* Vol 1.1 (Epub 4/26/2016; [http://www.ioreinternational.org/uploads/articles/Dr_Batra_revised\(1\).pdf](http://www.ioreinternational.org/uploads/articles/Dr_Batra_revised(1).pdf); pp 1-16.

47) Shlomi D., N. Peled, YA. Schwarz, GW. Soo Hoo, **RK. Batra**, G. Fink, T. Kaplan, S. Mollan, WR. Burfeind Jr. Non-Invasive Early Detection of Malignant Pulmonary Nodules by FISH-Based Sputum Test. *Cancer Genetics* 2018 Oct;226-227:1-10. (PMID: 30005848).

B) PEER REVIEWED (in preparation/submitted)

Veena MS, SK Basak, E Srivatsan, and **RK Batra**. Differentiation dependent dynamic changes in CAR expression in lung epithelial cell models.

INVITED REVIEWS/ BOOK CHAPTERS:

- 1) Dubinett, SM., PW. Miller, S. Sharma, and **RK Batra**. Gene Therapy for Lung Cancer. *Heme/Onc Clinics of North America* 12(3), June, 1998, pp 569-594.
- 2) Dubinett SM, **RK Batra**, P. Miller, and S. Sharma. Tumor Antigens in Thoracic Malignancy. *Amer. Journal of Resp. Cell and Molecular Biology* May 1, 2000, 22 (5): 524-527
- 3) **Batra RK**, S. Sharma, and SM. Dubinett. New Gene and Cell-based Therapies for Lung Cancer. *Seminars in Respiratory Medicine* 2000, Vol. 21 (5), pp 463-473.
- 4) **Batra RK**, S. Sharma and Lily Wu. Utility of Adenoviral Vectors in animal models of human disease I: Cancer. In *Adenoviral Vectors for Gene Therapy*. Edited by DT Curiel and JT Douglas. Academic Press, USA 2002.
- 5) **Batra RK**, Sherven Sharma, Robert M Strieter, and Steven M. Dubinett. Gene Therapy for Lung Cancer. In *Lung Biology in Health and Disease Series, "Gene Therapy in Lung Disease"*. Edited by Steven M. Albelda and Claude Lenfant. Marcel Dekker NY, NY. 2002.

- 6) S Sharma, M Huang, M Dohadwala, M Pold, **RK Batra**, and SM Dubinett. Cyclooxygenase-2-Dependent Regulation of Antitumor Immunity in Lung Cancer (pp 723-738). In Methods in Molecular Medicine Series, "Lung Cancer," Edited by Barbara Driscoll. Humana Press USA 2003
- 7) S Sharma, SC Yang, **RK Batra**, and SM Dubinett. Intratumoral Therapy with Cytokine Gene-Modified Dendritic Cells in Muring Lung Cancer Models (pp 711-722). In Methods in Molecular Medicine Series, "Lung Cancer," Edited by Barbara Driscoll. Humana Press USA 2003.
- 8) **Batra RK**, S. Sharma and R Bernal. Obstacles to Efficacious Viral gene transfer in vivo; Malignant Pleural Effusions as a Paradigm. (pp 545-560). In Methods in Molecular Medicine Series, "Lung Cancer," Edited by Barbara Driscoll. Humana Press USA 2003.
- 9) **Batra RK**, S. Oh and SK Basak. The isolation and molecular characterization of cancer stem cells (aggressive endophenotypes) in individual lung cancers. Chapter 18 in Stem Cells, Tissue Engineering and Regenerative Medicine. ISBN: 978-981-4612-77-7. Edited by David Warburton. Imperial College Press/World Scientific, London, UK 2014.

I have been an academic pulmonary and critical care physician and a translational physician scientist for the last twenty years, who is now seeking to advance programs within the Bio-Pharma sector. I am well versed in the clinical and basic science (molecular and cellular) biology of lung epithelial malignant transformation.

Past relevant experience:

1999-2007	FDA-IND Filing/Approval: “A Phase I Trial of CCL-21 Gene Modified Dendritic Cells in Non-Small Cell Lung Cancer”. (First-in-Man work for which I was chief architect of the regulatory portfolio to the FDA).
1999-2006	NIH-OBA/RAC Appendix M Submission/Approval: “A Phase I Trial of CCL-21 Gene Modified Dendritic Cells in Non-Small Cell Lung Cancer”. (First-in-Man work for which I was chief architect of the regulatory portfolio to the NIH-RAC)

Key basic discoveries:

- ***First demonstration*** that lectins targeting cell surface receptors could effectively and selectively mediate gene transfer into target cells.
- ***First demonstration*** that endogenous chondroitin sulfate containing Glycosaminoglycans and Proteoglycans inhibit (amphotropic and VSVg-pseudotyped) retroviral gene transfer *in situ*.
- ***First demonstration*** that **inter-tumoral** heterogeneity would confound the use of a specific viral vector for gene therapy in the treatment of lung cancer.
- ***First demonstration*** to document the anti-tumor efficacy of slc (CCL-21), an endothelial-derived CC chemokine, in an animal model of lung cancer.
- ***First demonstration*** that radioiodine uptake via the sodium-iodide symporter (NIS) required the thyroperoxidase gene product for intracellular retention of the therapeutic radio-iodine to kill cancer cells.
- ***First demonstration*** that the cellular attachment receptor for Adenoviruses (CxAdR) is also critical for the efficiency of xenotransplantation by lung cancer cells that constitutively **highly express** this surface adhesion molecule.
- ***First demonstration*** of successfully culturing out “lung cancer stem cells” from clinical lung cancer biospecimens.
- ***First demonstration*** of characterizing miR34a as a key target for replacement therapy to mitigate aggressive behaviors of lung cancer cells.

Key Innovations:

2016	UCLA and VA Patent Disclosure: UC Case No. 2016-840 , entitled “ <i>Discovery of common immunogenic targets in aggressive lung cancer.</i> ”
2016	UCLA and VA Patent Disclosure: UC Case No. 2016-803 , entitled “ <i>PMR for cognitive gains in ASD</i> ”.
2015	UCLA and VA Patent Disclosure: UC Case No. 2015-960-1 , entitled “ <i>Repurposing A1AT replacement therapy</i> ”.
2015	UCLA and VA Patent Disclosure: UC Case No. 2015-0615 , entitled “ <i>A Conceptual Approach To Mediate Atherosclerotic Regression</i> ”.
2013	US Provisional Patent; Application number 61/919802; <i>An Approach To Treat Anaerobic Gastroenteritis and To Restore Gut Epithelium</i>
2013	UCLA and VA Patent Disclosure: UC Case No. 2014-400 , entitled “ <i>An adjuvant approach to utilize replication competent adenovirus to treat cancer</i> ”.

2013 PCT/US2013/050712 entitled Methods of Biomarker Validation and Target Discovery. <http://patents.justia.com/patent/20150198583>. **Assignment pending.**

2011 US Provisional Patent; Application number 61/524731; *Method for Biomarker Validation and Target Discovery in Cancer.*

2011 UCLA and VA Patent Disclosure: **UC Case No. 2012-171**; *Method for Biomarker Validation and Target Discovery in Cancer.*

2004 PCT Patent WO/2002/085286: [Methods of using secondary lymphoid organ chemokine to modulate physiological processes in mammals](#) (**Application number:** 20040175355; **Filed:** January 13, 2004; **Issued:** September 9, 2004; Assignee: UC Regents).

2003 PCT Patent WO/2002/085300: *Methods of using interleukin-7 to modulate physiological processes in mammalian pulmonary fibroblasts* (**Application number:** 20030175801 **Filed:** April 18, 2002; **Issued:** September 18, 2003; Assignee: UC Regents).

Fiscal stewardship:

My group successfully leveraged less than \$500,000.00 of research funds into a program presently valued at over \$10 billion/year.

Raj K Batra M.D.
241 S Lapeer Dr.
Beverly Hills, CA 90211
310-991-1592
drrajbatra@gmail.com.

FORMAL CHARGE FORM: FACULTY MISCONDUCT

University of California, Los Angeles Academic Senate

(1) Provide the following information:

Name of Complainant: Raj K Batra MD

Name of Complainee: Steven M Dubinett MD

University ID #:

University ID #:

Position: Associate Professor, Department of Medicine.

Position: Vice Chair, Department of Medicine, and CTSI Program Director, Associate Vice Chancellor for Translational Science

University Address: Department of Medicine at VAGLAHS, Mail Code 169147.

University Address: 37-131 CHS
Mail Code 169017.

Daytime Phone: 310-268-3418

Daytime Phone: 310-267-2725

(2) Certification by the Complainant:

I hereby submit a formal charge to the Charges Committee. I have attempted to resolve this matter, but there has been no satisfactory resolution. I certify the charge(s) stated here, and appended documentation, are true to the best of my knowledge and belief. Furthermore, I understand that the Form and appended materials will be sent to the accused.

Signature: _____ Date: _____

Please indicate here the name of your counselor from the Grievance Advisory Committee:

Complete page 2, sign, and date the above certification and return to the Charges Committee, Academic Senate Office, 3125 Murphy Hall, University of California, Los Angeles, 90095-1408; for information call (310) 206-2469.

(3) The Charge(s):

I charge that Dr SM Dubinett failed to meet the responsibilities of instruction (mentorship)

I charge that Dr SM Dubinett used his position and powers of influence to coerce others to cause serious harm for arbitrary or personal reasons.

I charge that Dr SM Dubinett participated in intimidation and the orchestration of a severely hostile work environment.

I charge that Dr SM Dubinett violated Ethical Principles in relation to Scholarship by the intentional misappropriation, misrepresentation, and public promotion of work.

I charge that Dr SM Dubinett, in his supervisory capacity, devalued my professional competence not only for an appropriate initial assignment, but fair compensation and subsequent promotion.

The complaint:

I was recruited to UCLA in 1998 to bolster the knowledge base about gene and cell based therapies, and to develop the ways and means **to clinically implement (translate)** the use of these complex biological therapeutics into the clinic. Interviews with key “recruiters” (Drs Dubinett, Gasson, and Economou) will confirm that at the time of my coming to UCLA, I was **the** leading authority in the clinical translation of gene therapy for lung cancer in the world. They will also confirm that at the time, taking such an innovative approach into the clinic was amongst the “holy grails” of translational academic medicine.

By the events that followed, Dr Dubinett effectively exploited and usurped (assumed) **my expertise**, leveraged it to local executive authority, as well as national acclaim, influence and prominence. He then used that new-found fame and influence (obtained by inflated credentials) to suppress (extinguish) the research career advancement of this “trainee”. This was largely accomplished by his structural knowledge of *the system*. He effectively recruited me to “UCLA” into a “faculty appointment” at the VA, and then used his “inside knowledge” of local cost centers to effectively steal intellectual property (know-how) to garner large NIH grants that were then used to “train” others. I was left by the wayside, in a VA system that neither understood nor cared about Translational Biomedical Research, and which ignored (or rewarded) executive malfeasance. Moreover, by promoting my work as his own at UCLA, and by the support provided to him by high level crony executives in the JCCC and the DGSoM Dean’s office, Dr Dubinett was able to secure the opportunity to apply for the UCLA-CTSA, and to secure the UCLA-Vice Chancellorship of “Translational Medicine” as an accompaniment to the award being granted. I contend that this was largely accomplished by deliberate academic subterfuge, rather than his personal scientific and/or medical acumen. He effectively used the system and his cronies within to his great advantage, but at a great cost to the academic career of this complainant.

As if that wasn’t sufficient, Dr Dubinett then utilized the influence and power of his position to block a world-leading program in the study of lung cancer, which this complainant was trying to build after being removed from the “UCLA Lung Cancer Research Program”. In doing so, I suggest that Dr Dubinett’s calculated opportunism for personal gain has come at a great cost to the academic and medical advancement in the field we both study (lung cancer), as well as a great fiscal cost to the University.

As my narrative unfolds, the committee will realize that although I came to recognize his nature long before, my speaking out and fighting this man did not take place until I came to be anonymously informed that he was using his influence to consistently block me from garnering extramural support for a novel and highly innovative approach to the study of lung cancer (first of its kind in the world). Unfortunately, in 2012, this work has

subsequently come to be published by others elsewhere. It is also important for the committee to recognize that the issues I raise date back many years (long before Dr Dubinett came to be designated the Associate Vice Chancellor of Translational Research and PI for the institutional CTSA). In fact, I had specifically sought to come to a resolution **before** Dr Dubinett applied for and received the in institutional CTSA, believing that his attaining that status may corrupt a fair adjudication process.

To paraphrase what I have already indicated to the CHS-Ombudsman and executive leadership (before being referred *again* to the Senate), the facts are that over the years, I got screwed in many ways. But despite presenting the evidence to the leadership, it is not clear from those dialogs exactly what "factual threshold" is required for the administration to believe me, or for acting definitively on my behalf.

Confronted in this manner, I feel backed into a corner where the only way out seems to be to fight relentlessly, irrespective of the end results. The senate is a possible recourse for such justice and fair play. The following are the specific ways I "got screwed":

My IP (defined as "know-how") was used to garner resources, and to contribute to the global institutional research and developmental endeavor in an arena (Gene and Cell Based Therapy for Lung Cancer) where the knowledge base was, frankly, superficial. In return, I was to be provided mentorship and "training". But instead, through the resources that my work garnered, that "mentorship and training" instead went to other "junior faculty", for whatever reasons (or post-hoc rationalizations). And to top it off, the "contractual obligations" regarding the compensation for my work and research effort were not met, and my "faculty appointment" and "academic mission" came to be repeatedly re-characterized. To compound that "academic-hell", all this was done at a time when my "mentor" knew that I was facing extremely difficult life circumstances trying to deal with two newly diagnosed "special needs children" at home.

But despite that, as indicated, I was able to develop a highly innovative program to study the genotype-phenotype relationships that lead to aggressive lung cancer. But I found my avenues to self-sustain my research blocked from many different standpoints, over several years, finally leading to the loss of key personnel whom I had trained for this purpose. And when I sought redress and relief from the administration for my legitimate grievances, I was criminally charged in retaliation, and measures were put in place to remove me from my VA-lab.

Suggested Actions:

In the 14 years since I came to LA, UCLA and the VA (in matching funds) has garnered several millions of dollars in federal funds, along with a reputation for cutting edge expertise in lung cancer, in some (large) part by exploiting my expertise. Over the last 5-6 years, I have sought (I believe) "earned" support for independently developing a program for clinical translation. Since my needs have amassed over the years of scientific isolation at the VA, the absence of mentorship, the lack of due compensation, and the administratively orchestrated stalling and/or suppression of innovation, the **needs** are now urgent, and include the following:

- 1) Financial security to mitigate personal financial devastation and to overcome an immense amount of personal (emotional) and marital stress for nearly a decade.
- 2) Academic advancement and a "real appointment" that is commensurate to the knowledge base and vision that I bring to the table.
- 3) Mentorship (I have a persistent and lingering lack of knowledge and support regarding "process" and "mechanisms" for the acquisition of public and/or private funds).
- 4) Restoration of a sense of institutional belonging after years of being made to feel like a pariah.
- 5) Promotional resources for travel and lecturing (to overcome my loss of national credibility in Gene and Cell Therapy, and to promote my new vision for the study and treatment of lung cancer).

6) Infrastructural resources (space, equipment, and personnel) to overcome the decimation of a research program due to a persistent and sustained denial of funding, in which Dr Dubinett's negative influence seems have a key hand.

My **wants** can be considered in accordance with the level of enthusiasm (and the time table) by which UCLA is desirous of supporting our programmatic and specific project development. These will include our innovative programs for the "transcriptional sorting" of lung cancer and "Rehabilitation of motor function in autism", and to the discovery of the biological basis for aggressive tumor cell properties (cancer stem cell program) in lung cancer. For this development (if the institution is enthusiastic), I prospectively seek a position and institutional platform whereby the maximal value can be derived to mutual benefit. That specific position/platform can be negotiated, but should include each of the following elements:

- 1) Position of respect and responsibility.
- 2) Position reflecting autonomy of thought and action.
- 3) Position enabling that autonomy in the context of the institutional vision (executive influence).

The General Timeline to this complaint:

1998-2007: Developed and codified the clinical implementation of the first ever Immunogene Therapy Trial for the Experimental Treatment of Lung Cancer. I was unceremoniously dispatched from the program immediately after the IND-submission for the FDA was completed in 2007. Dr Dubinett is unable to maintain the sustenance of the UCLA Lung Cancer Research program (which I had key roles in developing) through competitive NIH peer-review.

2004-2011: Repeatedly sought mentorship support and resources that I believe I had “earned” by my contributions. This support was never provided by Dr Dubinett (in fact, in retrospect, it appears I was often misguided), and these measures were wholly ineffective. In 2011, I was asked by the administrative leadership to stop interacting with Dr Dubinett.

2006-2012: Repeatedly sought research support from the VA Executive and Research Leadership for overriding Dr Dubinett’s stifling and autocratic (and misguided) control of both VA and UCLA-research resources/collaborative support, and subsequently, for the recognized corruption of the peer-review process. These measures were (and to date, have been) largely ineffective.

2008: Reached out to the JCCC Leadership (Drs Gasson and H Herschman) for support towards the development of an independent program. I was heard, but not answered.

2009: Published a (to be) seminal paper in PLoS ONE about how to culture “cancer stem cells” from clinical samples. The paper received plaudits both locally (from the JCCC), and nationally (from the VA-ORD who funded the study).

2009: I am first (anonymously) informed that the peer-review process at the VA was being corrupted by negative influence that was stemming from members of my local institution, directly or indirectly through Dr Dubinett. (That Dr Dubinett had been surreptitiously corrupting the peer review process had been suspected since 2007). Following an egregiously biased review (for which an appeal was filed, and local VA-R&D administrators and the VA-COS were informed), the VA-ORD funding is suspended. The appeal regarding the peer-review process was summarily dismissed.

2009: Dr Gasson, who assisted in publicizing our seminal proof of concept observations, is informed about the corrupted peer review. Sometime (apparently in 2009), Dr Dubinett is selected to lead the institutional CTSA endeavor.

June 2010: I publish a perspective (with Dr David Warburton, CHLA/USC) that reframes the study of lung cancer. The perspective delineates that for us to make an impact on lung cancer, a phenotype (behavior)-based approach will need to be employed.

June to July 2010: Referred to Dr Dubinett for a “reconciliation” meeting by Dr R Strieter (former Division Chief, Pulmonary and Critical Medicine, UCLA). He admonished that if I was “ever to get funded” in lung cancer work, I had to go to Dubinett. Dr Strieter first informed me that Dr Dubinett had been selected to apply for the institutional CTSA, which he was likely to get. My subsequent meeting with Dr Dubinett was cordial, though ineffective.

2010: Having been stalled again, I recognize that I had to “speak out” to other University Leaders to neutralize Dr Dubinett’s influence. A series of e-mails were sent to Dr Gasson requesting help. Dr Gasson referred me to Dr Fawzy (Academic Dean, UCLA DGSoM). I Met Dr Fawzy in 12/2010 for “airing out my issues,” and given concerns that Dr Dubinett was blocking resources using negative influence, I requested research support that

came from *his* resources in an effort to stop the practice. A “mediation meeting” was scheduled for 1/4/2011, but was cancelled in late December by Dr Dubinett.

January 2011: I am retaliated against by the filing of a wholly fabricated “criminal harassment” charge, in collusion with members of our joint lab who were coerced to file a complaint against me, and with the tacit consent and administrative complicity of the VA-leadership.

February 2011: I am referred to the academic senate by Dr Dean Norman (VA Chief of Staff). I meet with Dr Feig (GAC) in late February, only to be asked about my whereabouts immediately following that meeting by my clinical supervisor (Dr S Santiago). The context raises concerns about how the “independent University Senate review process” may work.

May 2011: A UCOP-TRDRP proposal that had been linked online to a score of 1 (exceptional) does not get funded. Moreover, similar proposals (7 in total) that are sent to regional, state, foundations, and National Review Panels return unfunded without associated scientific justification, or the LOIs are turned down for further consideration without proposal review.

May 2011: Meet with Dr Fogelman for the first time. He provides influence and helps establish bridge support through the DoM and DGSoM Dean’s office for sustaining a key employee (the last of the personnel remaining; the rest had been dispatched or “laid off” due to an induced suffocation of resources).

June 2011: A Merit Review Application returns unfunded without scientific justification for the sixth straight time. Dr Dubinett now has secured the CTSA and the Vice-Chancellorship, so the reasoning behind the ongoing use of negative influence seems irrational and unduly cruel. After informing Dr Fogelman, I approach the Senate GAC again. On Dr Feig’s advice, the Academic Senate leadership (and Dr Fogelman) are presented with a summary grievance (on 7/13/2011), in order for the University to consider a joint response and a gameplan for us to secure support to move forward.

June 2011 to March 2012: Through dialogs with Dr Fogelman regarding potential avenues for research funding, I am advised to seek corporate and business ventures rather than governmental sources. I also learn for the first time that as far as UCLA executive leadership is concerned, I was “not a UCLA employee”. To determine IP rights on the process and approach I had already developed, I am referred back to the Dean’s office, and the UCLA-OIP. I have discussions with several different individuals in the Dean’s office, one of whom (Dr Gordon) refers me to the Ombudsman for mediation. The ombudsman subsequently refers me to Dr Hiatt, the new Academic Dean for DGSoM. A reconciliation meeting (3-27-2012) to resolve past differences and to outline a gameplan moving ahead is ineffective (the focus of discussion is geared towards the evidentiary basis of my acrimony with Dr Dubinett, rather than how to construct an infrastructure to support the innovative program I have developed).

January 2012-March 2012: A study that mimics exactly what we had been proposing to do since 2006-2007 (by individuals whose grant I had reviewed and provided critical feedback to in 2010) is published in January in **Cell**; and the theme (intratumoral heterogeneity) that we had been proposing to pursue for the last 6 years is published as the lead article in March in the **New England Journal of Medicine**. For all these past 5-6 years, I was fighting to be ***the first*** in not only thinking about the disease process (lung cancer) in the novel way that I had reconstructed it, but also to prove that I am correct in that thinking. Dr Dubinett, I believe, was instrumental in preventing that from happening.

The evidence supporting the Narrative:

1) Although there was expertise in immunology and/or COX2-biology in Dr Dubinett's lab, there was little to no expertise in gene therapy (compare CVs from the time of my recruitment). Specifically, there was no preexisting expertise when it came to translational/clinical applications of the complex biological therapeutics (gene transfer vectors). There also was no "UCLA Lung Cancer Research Program". The fact is that I had been doing innovative translational work in Gene Therapy for Lung Cancer since ~1992 with world leaders in the field. The fact is that *from the outset of my training, my work was driven towards clinical application (my models included primary tissues, and work that I began anew in 2006-2007 after being dispatched from the UCLA Lung Cancer Research Program was an extension of work I had initially done at UNC-Chapel Hill).* Thus, prior to coming to UCLA (1998), I had compiled a body of work that was the "**first of its kind**" in the world. If this is an apt definition of "*innovation*", then it was I who was the *innovator* in the group.

2) I had spent over 7 years developing and studying various gene transfer technologies (both non-viral and viral vectors) at UNC-Chapel Hill before coming here. The fact is that whereas Dr Dubinett was the lead UCLA-recruiter, Drs Gasson (JCCC) and Economou (Gene Medicine Program) each invested \$50,000.00 (into Medicine accounts controlled by Dr Dubinett) towards the recruitment. Dr Economou may also recall that during my recruitment interview, I advised him not to pursue a suicide gene therapy strategy for hepatoma that he was developing given information I had learned in my studies, and through interactions with Brian Huber at Glaxo Smith Kline while I was at UNC-Chapel Hill. Dr Economou also welcomed me as a new faculty member in the UCLA "Gene Medicine" program. Thus began my tenure at UCLA in 1998.

3) The fact is that my recruitment was fiscally made possible by an Advanced "Career Development Award" from the VA, on which Dr Dubinett was listed as the "mentor". This award largely provided salary support for the Advanced Trainee; the "mentor" was to provide the infrastructure and tutelage towards career development. In retrospect, my appointment at the VA as an Assistant Professor in Residence in 1999 was clearly not commensurate with my scientific expertise and translational knowledge base, but I concede that I knew no better at the time.

4) The fact is that by the time I was dispatched from the UCLA Lung Cancer Program in 2007, the rest of the professional world in our arena ("Translational Gene Medicine" for lung cancer) attributed my expertise and scientific credibility (my research identity) to Dr Dubinett. How did this happen?

5) The fact is that although I was recruited to "UCLA", I was assigned to work at the VA. This assignment, then and now, was a huge environmental mismatch in terms of the infrastructure that was needed for me to rationally pursue the work of my training and expertise. The evidence will verify that an agreement (a "Memorandum of Understanding") was reached between administrative officials at the WLA-VAMC, and the UCLA Dean's office. That agreement called for 25% of my salary support to come from UCLA for research efforts. Whereas substantially more than 75% overall effort continually went into "UCLA Research Efforts", the agreed upon UCLA-based compensation (the TNS) was transient. Importantly, the interpretation and meaning of the original memorandum of understanding (and consequently, the terms of the UCLA "appointment") repeatedly changed over time. Always, these changes occurred without conferring with me or notifying me, they simply happened. The first key "change" occurred around 2003/2004, when the definition of "UCLA Research Efforts" came to be re-interpreted as me being the PI on the grant (instead of being compensated for the "supportive role" I had been assigned to do by the lab director). Since my *primary job assignment* was to develop the translational program (and not my first RO1 aims), in effect, I was effectively "fired" by UCLA (my salary support was stopped in 2003 without an explanation, aside from Dr Dubinett indicating that "your Delta is your problem"). Meanwhile, since my unwavering career goals were to make a significant impact on lung cancer as an academic physician scientist, I was still substantively contributing to the development and maintenance of the UCLA Lung Cancer Research Program. This would continue until 2007, because Dr Dubinett now firmly held the strings to success in my chosen profession.

6) A recent conversation with the executive Chair of Medicine at the VAGLAHS (Dr G Brent) provides some greater insight into the mechanism of this subterfuge. It appears that the executive narratives that

accompanied our respective “MOUs” at the time of appointment were quite different. Dr Brent was brought into the VA a few years before me, and was duly informed by his mentor (Dr J Hershman) and the then executive Chair (Dr P Guze) that his academic success depended on his individual research. The narrative regarding my mission assignment was distinctly different. It was repeatedly emphasized (by Drs Dubinett, Santiago, Guze, and Gasson) that my academic success and career trajectory was dependent on “team success.” Thus, my whole focus was on enabling “team success.” That “team success” clearly happened, but to my unforeseen individual detriment. Moreover, similar “MOUs” for “in-residence” faculty at affiliated institutions were duly honored by the Dean’s office and executive administration.

7) It appears that the “firing” (withdrawal of support) by UCLA was by design. This is suggested by the fact that when a copy of my original MOU was requested from the administrative officer assigned by the Department of Medicine to the VA campus (Ms Meldis Asis) for this review process, it was no longer available in my academic files or in the records preserved by the administration. The course of events also seems to suggest that the MOU was simply an administrative façade to enable easy usurpation (theft) of intellectual property that I had collected and honed over a decade of prior research experience, before getting rid of me by artificially changing the terms of agreement. The MOU was never intended to be honored.

8) The fact is that following my arrival in 1998, I was assigned to several different University committees and forums to enhance the institutional knowledge base, as well as the facilities and regulatory infrastructure regarding the translation of gene and cell based therapy into the clinic. Interviews with Drs Economou, Gasson, William Quan, Christine King, Felicita Baratelli, and Antoni Ribas will confirm that I was a key contributor towards the original development of the JCCC- cGMP biological therapy facility, and a contributor to the development of the regulatory process for the intramural gene and cell based therapy projects. I had amassed expertise in this regard while at UNC. This leadership will confirm that I argued against developing a cGMP vector production facility at UCLA (advice which the administration heeded), suggesting instead using the National Gene Vector Laboratories (NGVLs). The fact is that the early templates for the implementation of gene therapy for cancer at JCCC/UCLA (especially for lung cancer) were *largely developed by me* during 1998-2001. Interviews with the local IRBs and JCCC-regulatory committees will confirm that I was the key liaison for the regulatory submissions and approvals for our studies, beginning with studies utilizing the Adenovector for IL7 gene transfer.

9) The fact is that translational physician scientists (e.g.: Drs S Dubinett and A Ribas) who benefitted greatly from my tutelage in scientific discussions and forums following my arrival are now globally recognized as being “experts” in their respective fields. However, these individuals have also consistently received considerable “institutional support”, and both currently hold a great amount of influence and institutional resources to independently implement their programs, and to direct their own career trajectories. I contend that the difference in our career trajectories is more so related to the opportunity and tutelage that was provided, rather than to the knowledge base they possessed, or the merit of their scientific endeavors. The question the Senate is asked to consider and explain is why Drs Ribas and Dubinett were selected for high levels of institutional support (a different path in terms of appointment, mentorship, and resource allocation), when a considerable amount of the scientific expertise that they were imbibing and regurgitating on their rise to their academic “success” was mine.

10) The fact is that although I was the key architect of these translational (clinical implementation) strategies (the portfolio that was presented to the FDA to conduct an immune-genetic therapy trial for lung cancer at UCLA; also called the IND, was largely my work), I did not derive the due benefits for their development. The fact is that over the years, this developmental and regulatory work took many hundreds/thousands of hours. The fact is that upon its completion, it was Dr Dubinett who then took the opportunity to present the work to National regulatory agencies (FDA-CBER), and who subsequently leveraged that assumed expertise to serve on their National Advisory Board. The fact is that whereas I was also the principal architect of the submission for the development and regulatory aspects of the cGMP vector that was manufactured through funding by the NIH-RAID mechanism, and the principal author of the IND that was subsequently submitted to the FDA, ***I was administratively left off of the parallel NIH applications that***

were submitted for funding the experimental implementation of the same work at UCLA. Thus, although I contributed greatly to the Development of BOTH Lung Cancer SPORE applications, my role was minimized on the first (as a “cost saving measure”), and I was entirely left off the second, even though the science, the strategy and the language behind the translational implementation of the immunogene therapy trial in lung cancer was largely mine. That work led to UCLA being awarded the first SPORE grant in Lung Cancer Research, the funding by the NIH-RAID Mechanism for the development of a clinical grade vector, and to the NIH-funding for the implementation of the clinical trial. Thus, although I did most of the work, the monies that were garnered for that work were always under Dr Dubinett’s control. Based on this evidence, I contend that I was very severely under-compensated for this effort, and all assurances by my “mentor” that this work was all an investment in my academic and professional promotion were never honored. He has since then used both the resources I had help garner (of which he took complete possession), and the imposed handicap from abdication of mentorship to leverage a position of exploitation and denigration.

11) The fact is that following my arrival on campus in 1998, I freely shared both knowledge and reagents (viral gene transfer vectors) with the UCLA community. These complex biological reagents had been developed, validated and/or procured by me prior to my coming here, and were in my possession when I deposited them in core UCLA facilities. Interviews with Drs Lily Wu, Matthew Rettig, William McBride, and Nori Kasahara will confirm that I freely deposited and/or shared viral vectors and constructs that were in my possession, for the general benefit of the UCLA gene therapy community. Both Frank Pajonk/W McBride (1999) and M Rettig (2005, 2008) published important work using the AdIkB vector that I had helped develop and validated at UNC prior to coming here; co-authorship was not offered or denied on these studies by the principal investigators, with the justification that the recombinant adenoviral vector represented a simple “reagent”, therefore intellectual contributions were negligible. Dr Dubinett, the “mentor”, also did not push the agenda with those investigators for promoting or accrediting his “protégé” for their use.

12) The fact is that I was funded on an RO1 application on Adenoviral Gene Therapy for Lung Cancer in 1999 (on the resubmission of a NIH-First Award that was initially submitted from UNC-Chapel Hill). The fact is that Dr J Economou was a member of the Study Section that funded that application, and that following his departure, he referred Dr Dubinett as a member to that study section. That study section dissolved soon thereafter, but it provides evidence that (in addition to earlier joint publications), the Economou-Dubinett inter-relationship clearly has long standing roots. This inter-relationship has seemingly evolved to mutual benefit to powerful positions within the UCLA executive administration. Both Drs Economou and Dubinett presently reside in the Chancellor’s office, in charge of the UCLA Global Research and Translational Endeavors, respectively. Evidence presented here suggests that at least Dr Dubinett’s rise to that position of power and influence has come about by dubious means.

13) The fact is that since coming here, Dr Dubinett and I have shared 27 publications together. I contend that during this entire service, however, I was not only deliberately left off of NIH-funding proposals for work and language that I created (as described above), but I was also not supported towards developing any “independent” projects through the repeated denial of requested mentorship and collaborations. Accordingly, I was blocked both from garnering the resources I had help accrue, and the promised mentorship was also not provided. The question the Academic Senate will be asked to consider is in what capacity was my work following my recruitment here undertaken, especially when my work assignment was sold to me as a “team venture” for which I had the lead at the outset. It was only after the resources were in hand, however, that Dr Dubinett variably characterized my participation in the program as that of a “trainee” or a “collaborator.” Unfortunately, the bottom line is that I did not receive mentorship, nor “earned” resources.

14) Whereas Dr Dubinett was funded on a 13 million dollar SPORE in 2000, the “compensation” for my intellectual input was being funded on a “pilot project” that he already knew was going to be supported by the American Lung Association. Although he did not reveal to me his intentions directly, the fact is that he “reassigned” my project to Dr Reckamp (around 2001-2002). I was nonchalantly relegated to a position of scientific isolation at the VA. Being denied the developmental support through resources I had “earned”, and being denied mentorship posed severe hardships.

15) The lack of mentorship and limited access to (incompetent) administrative support was a significant obstacle for me to independently garner research funds between 2003-2009. Due to the lack of mentorship, the most relevant funding mechanisms and the scientific peer review process was a black box (in which only Dr Dubinett had influence). As noted above, sharing my concerns with the administrative hierarchy at the VA and to the JCCC-leadership was ineffective (in retrospect, it is possible that the administration was already aware of the planned exploitation, and that their prescribed role was to stall and/or provide rhetorical empathy and/or disparage the work I was doing, depending on context). By abdicating mentorship and by blocking effective collaboration at the time it was most needed, Dr Dubinett imposed a severe handicap (while simultaneously incorporating the great fiscal and professional-advancement benefit derived from my specialized expertise in gene therapy into his own portfolio).

16) Whereas my work was initiated and performed under the belief (instilled by Dr Dubinett) that these were "our projects" in which I had the lead (as explicitly stated on two distinct occasions, several years apart; see detailed narrative), I was financially supported with salary support only ONCE (upon demand in 2006-2007) during our entire relationship (see narrative). I requested financial compensation under dire personal circumstances (induced by his and the administration not honoring the MOU agreed upon during my hiring). However, even then, the promised reassurances (that I would be assigned the lead on the clinical implementation of the trial) were not met (after Dr Reckamp left, that lead was reassigned to Dr JM Lee).

17) Since the NIH-applications for funding the work I had undertaken at UCLA did not include a percent effort for me, my contributions were administratively devalued not only within the institution, but also at the NIH (even though the underlying scientific expertise was mine). Thus, in addition to not being duly compensated for that work according to the terms of the MOU between UCLA and the VA, it appears that there was a deliberate effort to deny me both the credit and promotion for that work in NIH-peer-review. Moreover, because he sequentially attributed my work in professional speaking forums to Drs Reckamp and Lee, I was also denied the opportunity to independently promote the work as mine. This was easily accomplished by denying me of an opportunity to interact with National leaders in Lung Cancer Research through the SPORE mechanism (the funds were instead used to promote the careers of Drs Reckamp and Lee). This administrative orchestration of events limited not only my local prospects, but prevented me from seeking positions commensurate with my expertise elsewhere as well.

18) The fact is that Dr Dubinett had free access to and effective control of all the resources that were garnered by me at all times as well. Thus, not only was I blocked from resources which came from my work in his name, *but that he often usurped funds that were granted to me* without pre-authorization. This usurpation of resources probably occurred right from the outset (I was administratively rather naïve), and was most easily accomplished with the VA funds, where the local and central R&D administrations were coaxed and/or being intimidated into complying with his needs (see narrative). As recently as 2008-2010, monies from grants made to me were absconded by the VA Research administration for other purposes. The total amount of funds absconded in this manner over the years is not clear, but anywhere from \$70,000.00-\$150,000.00 were inappropriately removed from my Merit Review Funds between the years 2007-2010.

19) Nevertheless, with less than \$500,000.00 in my name between 2005-2009 (as indicated, many of my funds were redistributed elsewhere by the VA R&D administration), my group and I developed a novel (paradigm-shifting) approach to the study of lung cancer. I venture to suggest that our publication in 2009 has already tallied a greater readership and long term impact on the study of lung cancer than the collective works of Dr Dubinett (with twenty to fifty times higher levels of directed intramural and extramural support).

20) Except for \$15,000 (of the promised \$30,000) in bridge support in 2005 (towards VA-Merit Funding), between 2005-2011, the UCLA Dean's Office and JCCC did not support (fund) any of the many projects I submitted for internal "competitive review" (can be confirmed by interviews with Drs Gasson, Herschman, Dubinett, Rome and Economou). Even after the JCCC leadership promoted the publication of our innovative work through a news release in 2009, and clearly recognized our translational vision in 2010, requests for tangible support for extending the work were inexplicably denied. Furthermore, all extramural avenues for funding (VA, UCOP-TRDRP, CIRM, and possibly select study sections within the NIH) have also been blocked.

By 2009, it became clear that Dr Dubinett was surreptitiously corrupting the peer review process (by negatively biased interactions with a network of panel members) at the foundation and national levels, and irrespective of how I presented my work to the reviewing audience in many different forums, it was unlikely to get fundable scores (see narrative).

21) With respect to the VA Merit program, the fact is that Dr Dubinett has long held sway with the local and central VA Research Authorities. He has been the Chairman of the local R&D Research Committee, and was also the Chair of the National VA Medical Research Advisory Board for Oncology Research from 1999-2003. Moreover, his influence with the central administration may have played a role in my initial “recruitment to UCLA,” and his stature there was likely considerably heightened by his usurping promotional credit for the innovative translational trials that were undertaken at UCLA (work that I had developed). Interviews with VA officers and Dr Sharma will confirm that his influence at the VA is unquestioned, and that he is “revered” in that advisory capacity. I contend that he took advantage of that position to deny funding through the VA for my independent research efforts (see narrative). In addition, I contend that in 2011, his direct/indirect influence denied funding on a revised (A2) application submitted to the CA-TRDRP, even though the application was linked to a score indicating that it was an “exceptional” proposal for several weeks.

22) It is critical for the Senate to comprehend the value of the work for which we have been seeking institutional and extramural resources since 2006. Requests were made for developing 1) a “Transcriptional Sorting” approach for the study of lung cancer, and 2) for promoting the study of intratumoral heterogeneity and emergence of a “Lung Cancer Stem Cell” program.

23) For the “transcriptional sorting” approach, we applied for funds from the JCCC in 2008, and then the NIH and CIRM in 2008/2009. Regrettably, a study similar to the one that we had proposed in 2008 was published in the Journal ***Nature*** in 2011 (Drs Gasson, Fogelman and/or Economou can confirm this). With respect to the theme of Intratumoral heterogeneity, we started soliciting resource support in 2006, and we published a proof-of-concept model on this theme in ***PLoS ONE*** in 2009. The approach we had developed was published by others in ***Cell*** in January of 2012, and the theme came roaring into the limelight by a high impact ***New England Journal of Medicine*** publication in March of 2012. I contend that with a fair and prudent resource allocation process, it should (and would) have been UCLA Investigators reporting those observations and discoveries, under a non-biased and vision-oriented scientific executive leadership structure.

24) Anecdotally, one of the (suspected) reviewers of our original 2008 JCCC proposal (?Dr Hong Wu?) regarding our “transcriptional sorting approach” recently invited the senior author of the 2011 ***Nature*** publication from MIT (Dr Tyler Jacks) for a seminar to UCLA. In addition, I am also informed that a similar approach is being developed by investigators at Boston University, where Dr Dubinett currently has key collaborations. Given the course of events, it is not unreasonable to speculate that the intellectual property I had envisioned developing may have been openly or inadvertently shared with professional cronies, who then took up the projects as their own.

25) Anecdotally, shortly after our publication in ***PLoS ONE*** in 2009, I was asked to review a scientific proposal by a Singapore Ministry that manages research funds for A-STAR for a project similar to mine. Although I had some technical concerns, I was impressed by how much institutional support the Investigators had been able to garner. In January, 2012, these investigators published a game changing article for lung cancer in ***Cell*** (Zhang, WC et al.; ***Cell*** 148, January 20, 2012). Meanwhile, I am still languishing and fighting for my academic survival at UCLA. What this anecdote indicates is that *any innovator anywhere in the world* can make a big impact, but that such discovery requires a vision, an opportunity, and institutional nurturing. From a personal perspective, it appears such institutional nurturing at UCLA has not been handed out on the basis of the merits of the endeavors or the potential impact the work may have.

26) Collectively, what these anecdotal facts also indicate is that the work that I had been proposing to UCLA officials and leaders for support was *consistently cutting edge and of high caliber*. However, it appears that administrative officials approached me and my work with a preconceived mindset of rejection. I contend that these (denial of opportunity) actions have seriously harmed me in my chosen profession, but may also

have harmed the pursuit of translational science for the benefit of society. Is that pursuit not the supreme mission for the health sciences component of our educational institution?

27) The fact is that even though University officials acknowledged and understood the value of our innovative work as early as 2008-2009, no overtures were made by Dr Dubinett to correct the course of our conflict, to meet my recurrent requests for mentorships and collaborative support, or to resolve our differences through negotiation. The fact is that I first “publically” aired our differences to cancer center leaders in 2008, and **in writing** (to Drs Gasson and Fawzy in late 2010). In these discussions and reports, I alluded to our long standing difficult interrelationship, and a culture of unchecked exploitation. That disclosure was made to seek administrative redress and relief (to move forward with our important new approach to studying lung cancer), but instead it put me in a “catch-22”; **severe retaliatory efforts were brought to bear**. I went from simply being a university pariah who could be ignored to an institutional enemy who needed to be removed. **An overtly hostile work environment was created, fabricated criminal charges were filed, and measures to evict my from my VA-lab were brought into play.** These measures were implemented by and through UCLA administrators with palpable antipathy, who worked in collusion with high ranking VA administrators. The process was orchestrated at the highest levels of administrative bureaucracy. These measures continue, as indicated by communication to the UCLA administrative hierarchy and the Senate in 3/2012.

28) The lack of clarity regarding my University “appointment” has also taken on added complexity. In July/August of 2011, I was informed by Dr Fogelman that I “was not a University Employee”. This revelation suggests that I was hoodwinked from the start. Until this revelation, it simply made no sense why the administration was hell bent on killing my career or my program that they clearly recognized as important. After that revelation, I was forced to file for protection of IP rights on my own (see letter to VA-OGC/VA-OIP dated 11/7/2011), and to piece together the circumstances that have befallen me. The University has not supported my ventures because (despite the rhetoric that recruited me here), it did not view it as “its own.” For me, it appears that the University has no interest in me or the IP I have developed, and the actions of the executive officials over several years support that conclusion. Perhaps, the University was deliberately allowing me to lose a competitive edge, and was creating an environment to force me to leave.

29) **If** that is the case, **then** it appears that between 1998-2011 (when I found out that I wasn’t “a UCLA employee”), the circumstances and conditions of my University “appointment” were **explicitly designed to exploit (steal) IP related to gene therapy (and perhaps, after my induced departure, to steal the novel approach for the study of intratumoral heterogeneity in lung cancer), without regard to the harm done to this victimized (and then vilified) career-investigator.**

30) Alternatively, it is also possible that the executive administration (Dean’s office) simply made a mistake in honoring an established MOU for an “In Residence” faculty member at its academic affiliate, the VA. If that is the case, then I have been a “university employee” and a “tenured In Residence Faculty” throughout this time period (I certainly received UCLA-W2s earlier, and over the long timeline of my grievance, in my interactions with Dubinett, I have consistently maintained that our agreed upon terms of service and appointment had not been honored). But if that is the case, then the University has acted arbitrarily in not meeting it’s contractual obligations to this “employee” (other contracts in similar situation were honored), and compensatory/punitive damages are warranted.

THE DETAILED PERSONAL NARRATIVE:

The issues/concerns presented here date back to my hiring. Concerns regarding academic misconduct first arose in or around 2001-2002, when I first questioned the reassignment of my standing research endeavors to Dr Reckamp. Since then (until now), the concerns were repeatedly presented and repeatedly ignored by both Dr Dubinett, and then by both VA and UCLA administrations. Thus, ***issues that originally arose during my “training” have lingered, and significantly impact career advancement and work environment today.***

In reading through the historical narrative, I hope that the reviewers will recognize that it is ***because*** of my established expertise and translational competence that I was first recruited here. I suspect it was also ***because*** of this established competence (known only to my former “mentor” and members of the then JCCC-administrative hierarchy) that I came to be re-characterized as a threat.

I first recognized (in or around 2004-5) the attacks an unopposed and biased negative narrative that was gossipy in nature. This evolved into my being made a pariah at the institution I called my own, with my research career being targeted for elimination. I contend that in my quest for justice and fair play, these fundamental pillars of academic medicine have been repeatedly denied.

Perversely, it seems that ***Dr Dubinett has instead been increasingly shielded by the institution, despite serious leadership missteps along the way.*** Whereas he has been *promoted* for his mistakes, *my career and livelihood have been grievously hurt by his missteps*, because *I* played a major role in the acquisition of the initial UCLA lung cancer SPORE, and did my part towards the submission of the second, but did not derive the deserved credit. Thus, it appears that whereas the administration has been generously forgiving regarding Dr Dubinett, it has consistently ignored or aggressively acted out against me, despite the value I had brought to the institutional endeavors, and the recognized potential value I currently hold.

A. I charge that Dr SM Dubinett violated Ethical Principles in relation to Scholarship by the intentional misappropriation, misrepresentation, and public promotion of work.

I was recruited to UCLA in 1997 to develop a Gene and Cell Based Therapy program for lung cancer. I began my work in Gene Transfer Technologies in 1992 with Dr David Curiel, and continued my studies under the mentorship of Drs Richard Boucher (Chief, Pulmonary Medicine and Director, UNC-CF center), and Frank Detterbeck (Director, Multidisciplinary Thoracic Oncology Program) after Dr Curiel's departure from UNC. These circumstances are important, because they highlight that with respect to the medical and clinical translation science of Genetic therapies for lung cancer, it was *I* who possessed this expertise. *I* brought the expertise and several of the viral gene transfer vectors that I had developed and/or acquired with me, which were then widely disseminated in the UCLA community. Dr Dubinett's lab expertise was in immunology, and he was developing an expertise in COX2 biology in lung cancer with Dr H Herschman's support.

I was amongst the first investigators in the world to systematically apply a suite of viral and non-viral vectors to establish their gene transfer efficiencies into lung cancer cells *in vitro*. In addition, at UNC, *I* also first developed the protocols to primarily culture advanced stage lung cancers (I would return back to this work in 2006, in order to model realistic tissue culture environments, which simulated the complex *in situ* disease) for my studies on gene transfer technologies. *I* was the first to identify several different pre-existing hurdles to adenoviral and retroviral/lentiviral gene transfer for *in situ* gene delivery in lung cancer. Many of these studies were the first of their kind in the world.

Because my intent at the outset was to take gene therapy into the clinic, I developed a working knowledge of the translational and regulatory aspects of Gene Therapy. In 1996 and 1997, I attended NIH/FDA-CBER workshops that detailed the regulatory aspects of Phase 1 vector development, and the

acceptable safety and efficacy surrogate measures that were applicable for gaining regulatory approval of a Biological Product through the FDA-CBER branch.

Accordingly, I had both substantive lab-based experience, and practical regulatory experience, for the clinical implementation of Gene Therapy before my recruitment to UCLA. Dr Dubinett, the mentor and lab director, did not have this background, and did not comprehend the nuances of clinically applying this technology until after I arrived.

I was initially offered a “privileged membership” in the UCLA academic community. The recruitment was bolstered by an investment by the JCCC and UCLA Gene Medicine Program, and a “Memorandum of Understanding” between the administrative staff at the VA, as well as by the Dean’s office at the UCLA School of Medicine. I had a pre-existent salary support built in through the advanced research career development (ARCD) award from the VA, following a proposal that was submitted from UNC-Chapel Hill. Using the VA-ARCD-mechanism was possibly Dr Dubinett’s brainchild; and this administrative contribution may have subsequently allowed him to hold great sway with the central VA-R&D administration with respect to my career trajectory. If so, perhaps he felt that *my* innovative contributions were *his*, even though the expertise wasn’t.

Dr Dubinett recruited me to “UCLA” (I do not recall visiting the VA during the “recruitment phase”). The Gene Medicine Program and the Jonsson Comprehensive Cancer Center each contributed \$50, 000 into Dr Dubinett’s accounts towards the recruitment. I was assigned an “academic in-residence position” at the VAGLAHS. Nuanced differences between “in-Residence” appointments versus state-supported FTE were inquired about, but I was reassured that *this position was in equal standing with a position at the University Hospital*. A memorandum of understanding (signed off by the VA Chief of Staff, and the VA representative of the UCLA SoM Dean’s office) indicated that 25% of research effort would be supported by UCLA (**see Appendix; MOU**).

From UNC-Chapel Hill, I brought with me and freely shared all cell lines, gene transfer vectors, reagents to assess transgene expression, supplies (from funds of an ongoing ALA grant that I had at the time), and expertise in implementing gene transfer technologies. I began my work in January, 1998, with an urgency to please my mentor and senior administration, being grateful for the opportunity.

I had most of the funding for salary support through the VA-ARCD, but I was asked to submit an application for a NIH-RO1. I succeeded in getting that award in 1999 (on a resubmission of a “First Award Application” from UNC). The study section that reviewed that RO1 application was an experimental therapeutics section on which Dr Economou sat at the time, and on which Dr Dubinett was asked to serve on later. Later, I came to recognize that getting this RO1 was really a curse in disguise. Although it established my expertise in Gene Therapy Applications and allowed the administration to support my MOU-associated “Delta” (the agreed upon research salary support from UCLA was 25%), it jointly gave my VA-ARCD mentor the opportunity to simultaneously characterize me as an “independent” investigator. However, under his direction and supervision, my ***principal institutional task assignment*** was entirely different from the work proposed on the VA-ARCD and NIH-RO1.

Before my recruitment in 1998, although Dr Dubinett had co-published in the emerging field of Gene Therapy, there was ***no established expertise or infrastructure at UCLA to implement a clinical trial in gene therapy of lung cancer***. Based on his limited experience in the field, Dr Dubinett also did not possess the regulatory expertise, or the wherewithal to develop an infrastructure suitable for bringing clinical gene therapy for lung cancer to fruition. Thus, ***it was my experience and expertise in the field that largely contributed to both the programmatic and structural development of the institutional expertise in gene and cell based therapies.***

Specifically, I was assigned on a task force that initially developed the cGMP gene and cell therapy facility within the JCCC. I advocated **against** establishing an institutional cGMP vector core, arguing that need be filled by the National Gene Vector Laboratories (NGVLs). I also participated with William Quan and Christine King to develop the regulatory compliance infrastructure at UCLA. I was asked to serve in that capacity by Drs Dubinett and Economou, who was head of the Gene Medicine Program at the time. *It bears noting that during this process, I was a mentor in this area to Dr Antoni Ribas, who was a much more junior colleague (or perhaps a clinical fellow at the time). Toni's career, however, subsequently followed an entirely different trajectory; based largely on differences in mentorship and the opportunities he was provided through his mentor, Dr Economou.*

For developing this institutional experience, and subsequently, for the development of the therapeutic protocols and surrogate safety/efficacy measures for the implementation of gene and cell based strategies for the experimental treatment of lung cancer, my expertise was provided freely, voluntarily, and voluminously. It was provided **with the direct understanding that I would spearhead the forthcoming clinical gene therapy trial in lung cancer**. At the time, taking a bench based biological therapy project into the clinic was **the holy grail of translational experimental medicine, and this was (and is) part of my expertise**.

Based on my own lab work, however, **it became clear that direct adenoviral mediated gene delivery was not likely to work for treating lung cancer**. Thus, I became a strong proponent for trying to get an ex-vivo gene therapy approach in place. Although I had significant issues with using dendritic cells as the ex-vivo transduction-targets for implementing this strategy, there was a pre-established institutional expertise (Drs Sharma, Dubinett and Roth) in these cells. I conceded, and we jointly decided to use Adenoviruses to transfer the IL-7 gene (and subsequently, the CCL-21 gene) into autologous dendritic cells in the cGMP lab, and then introduce these cells back into patients with lung cancer. This was (and had to be) a “team effort”, which included expertise in Dendritic Cell culture and immunobiology (Drs Dubinett and S Sharma) and the basic gene transfer and translational/regulatory aspects of viral gene therapy (Dr Batra). **But I was the primary architect of the overall translational and clinical plan, and the principal author of all of the intramural and extramural regulatory elements (NIH-Appendix M and institutional IND), on which the trial design and NIH-funding proposals were based.**

Thus, between 1999-2007, my **primary institutional assignment was to translate** gene/cell based therapy into a clinical trial, and to provide *ad hoc* support to post-docs and junior faculty as part of the general mission of the UCLA Lung Cancer Research Program. My publications over that interval support that I was a very good collaborator and the go-to mentor in this capacity. Acquiring funding for my “independent endeavors”, or working on trying to figure out better ways to apply Adenoviral gene delivery (my own expertise) was not consistent with that assignment, nor was it supported by the research infrastructure at the VA. In fact, I was often admonished that my highest priority work was generating and amassing the documents to meet the massive amount of prerequisite regulatory approvals, designing the experimental pre-clinical and clinical trials, and developing protocols to meet cGMP standards. **It was not until I actually ran out of NIH-funds/resources (in 2003/2004) that were in “my name” that I was told that my academic advancement was actually dependent on my “independent” ventures. I contend that since I was the only one in our group who possessed the incoming necessary translational and regulatory expertise for implementing a clinical gene therapy trial, my work already represented “independent” ventures.**

Since I was wholly isolated at the VA, and my “developmental support” was redirected towards developing others, only much later did I come to learn that the entirety of my work over this period was being promoted both within the institution, and in national forums, without reference to me. Dr Dubinett was usurping the credit, promoting the work as his own undertaking, and ascribing the developmental resources and credit elsewhere (i.e.: to Drs Karen Reckamp and/or Jay Moon Lee).

As indicated, the developmental and regulatory work was done with our clear understanding that I would spearhead the clinical trial. This was ***explicitly stated on two occasions***, and implied throughout our working relationship. ***First***, in early 2000, shortly after I received the NIH-RO1 for studying Adenoviral Gene Transfer in Lung Cancer, my two and a half year old son was diagnosed with Autism. I bring this issue up only because this event changed my life circumstances; it encumbered significant additional time and financial burdens. It prompted me to reconsider and question my institutionally assigned role, ***and I asked Dr Dubinett to be recused from my assignment to shepherd approvals for a clinical gene therapy trial in late 2000/ early 2001. He reinforced that my primary assignment was to translate gene/cell based therapy into a clinical trial, and that my success and individual promotion at UCLA was rooted in “team” success.*** He reassured me that I, by virtue of my skills, had a significant role to play in the development and implementation of processes that would lead to that team success.

The work itself was highly successful by all measures. We received NIH funding for continuing pre-clinical development of the immunogene therapy trial through a large UCLA Lung Cancer SPORE grant with Dr Dubinett as PI, which led to the birth of the UCLA Lung Cancer Research Program, with Dr Dubinett as program director.

However, although ***I*** was the one who had provided the background work and had developed the templates for how to translate the benchwork into the clinic for the SPORE, ***the continuation of that work was inexplicably reassigned to Dr Reckamp.*** It is unclear how this reassignment was cleared through the internal and external advisory boards governing the UCLA Lung Cancer SPORE, since Dr Reckamp had no background in gene therapy. This reassignment was also undertaken without my knowledge or assent, but by 2002, Dr Reckamp had assumed ALL of the benefits that arose from being part of the SPORE-program (national networking and team collaborations through the NCI-SPORE-program, and being designated the *de facto* clinical project leader for the immunogene therapy project). ***Although I had earned that position, and still was “in-training”, I was left to fend for myself at the VA.*** When I expressed concerns about the situation, I was cut off or ignored.

What this course of events began to illustrate is that the VA was in reality a parallel “training” program, designed to usher individuals towards a dead end. This switch in assignment of credit for work done was carried out quite easily because Dr Dubinett had assigned me (a “trainee”) in an environment that was largely devoid of cutting edge science, and that kept me ill-informed about the promotional role and responsibilities of an academic mentor. Moreover, the VA, which had awarded me the “career development award” did not provide any regulatory oversight to ensure that laboratory directors were living up to their training obligations. ***The institutional dichotomy allowed both UCLA and VA to plausibly claim deniability for research training oversight, and ironically, the only career that was developed was Dr Dubinett’s.***

As Dr Reckamp became the public face for the clinical gene therapy project, and was provided with a voice in the administrative aspects of the SPORE, ***this redirection of credit simultaneously stripped me of all established credibility in my own field of expertise.*** That credit came to be assigned to Dr Dubinett (the “translational” director) or to Dr Reckamp (the clinical trialist). Meanwhile, whenever issues arose with respect to the scientific or regulatory elements of the trial, Dr Reckamp was simply referred to me for explanations, clarifications and advice. Nevertheless, the promotional credit for that work in national forums, funding agencies, and within the Lung Cancer SPORE peer network went to Drs Dubinett and Reckamp.

Interestingly, by 2005/2006, it became clear that Dr Reckamp did not have the competence to complete the job to gain NIH-OBA and FDA regulatory approvals, and that she would be leaving the institution. I was again coerced to formulate the final documents that ultimately led to NIH-OBA and FDA approvals. Given personal circumstances, I requested and received a small stipend (\$15,000.00) to do this work, ***but the primary driver was the reassurance (for the second time) that I would lead the clinical trial.*** I was assured of that on a trip back from Washington DC where we had presented our proposal at a pre-IND hearing for the FDA.

To complete the regulatory framework, I was directed to work with Drs S Sharma and JM Lee to develop the trial. I was the key architect of the framework and design, and the principal author of the documents that led to the regulatory approval process. **However, unbeknownst to me at the time, Drs Sharma and Lee were jointly instructed to work with me to surreptitiously write up the funding mechanism for the trial; an application in which it was reportedly predetermined that I would not be listed as an investigator with effort. Dr Lee would be taking charge of the clinical implementation of the trial.**

In effect, my intellectual property and ideas were being directly usurped to develop a trial *that would fund others through the SPORE and/or R-21 mechanisms. This was not the first time such tactics were employed, but it was amongst the most blatant acts of exploitation and preplanned executive malfeasance.* Thus, *immediately after the documents for the FDA-IND were completed, it became clear that someone else (Dr JM Lee) would be heading the trial at UCLA.* Again, I was not consulted, nor did I assent to this action; it simply happened. My subsequent complaints were met with taciturn dismissals, or taunts, and I was forced to disengage from the lung cancer research program in 2007.

Harm Done:

The advent of UCLA Lung Cancer Research Program, and the NIH support it was able to garner was clearly influenced by its inclusion of a first ever gene modified cell-based biological therapy for lung cancer (**my work**). The FDA-approval for an immunogene therapy for lung cancer initiative at UCLA in 2007 further propelled Dr Dubinett to national acclaim and influence. The pre-IND and NIH-RAID interactions also afforded him the opportunity to sit on FDA councils for biological therapies (FDA-CBER). *I contend that this was a position which I rightfully deserved, given that it was largely my regulatory and translational work for which he had usurped credit.*

Moreover, by subsequently sharing the IND documents I had prepared with others in the field, Dr Dubinett also garnered influence and a network of support from other investigators in the biological therapy for cancer community. It gave him the opportunity to portray himself as a “generous and open collaborator”, and gain additional credibility off my work, while little of his own intellectual property or direct effort had been expended in generating the documents. He also gained considerable favor and positive influence with future reviewers of his grant applications; **that influence would later serve in being negatively directed towards me in peer-review panels.**

But even as the FDA-approval directly led to the clinical implementation of the first immunogene therapy trial for lung cancer in the world, and which likely played a key role in the deliberations to his being assigned the opportunity to head the institutional CTSI (and to advance to a UCLA-vice-chancellorship in “Translational Medicine”), *he indicated to me that the work I had done was “not of value” because it did not amount to “an academic advancement”.* This was exceptionally hurtful rhetoric, with the **most perverse irony being that the clinical “translation” of gene-based bench research for lung cancer is EXACTLY why I was recruited to UCLA in 1998.**

Particularly appalling for me (and a cause of significant concern) was that he was offered the leadership of the CTSI **after failing to secure the Lung Cancer SPORE renewal.** Whereas I had taken considerable pride in being part of developing that program, and bringing UCLA lung cancer research back on the map, not only had he wholly taken credit for all of the highly innovative work of that program, he had crashed it into a ditch. *And the institution seemed to be rewarding him for that lost opportunity.* I felt that Chancellor’s office for Research should have been populated not only by individuals with influence and networking skills, but who also possessed a strong knowledge base directing a cogent **scientific vision.** In my view, Dr Dubinett was not such a man.

In summary, between 1999-2007, when I most needed promotional support and scientific mentorship to enhance my academic career prospects, repeated promises of academic promotion or compensation for “team

effort" went out of the window when "team success" was garnered. My work was usurped or misappropriated, and I was stripped of my knowledge and expertise (the currency of academic medicine, as described to me much later by Dr Fogelman). ***Effectively, for me, a huge amount of work and effort over an 8 year time period amounted to an exercise in how to formulate and implement an institutional IND.*** I was deliberately and cruelly cut out, *with the justification that no compensation for the effort was warranted because my work was "not of academic value."*

B. I charge that Dr SM Dubinett failed to meet the responsibilities of instruction by a deliberate abdication of mentorship:

Whereas the lack of financial or promotional compensation for my highly specialized intellectual property hurt, ***the concomitant loss of mentorship was equally or more devastating.***

I contend that it was directly as a result of imposing an insurmountable workload (the various "team ventures" had taken place over several years, amounting to thousands of hours), coupled with an abdication of an avowed professional mentorship responsibility (see below), which allowed Dr Dubinett to promote the (unopposed) view at the University that "my work" had little value, since I was unsuccessful garnering independent national funding.

What actually happened is that by assigning a massive workload, by abdicating his mentorship role, and by instituting a blockade on effective collaborations with key personnel at the University, he imposed a severe handicap, which he has subsequently exploited as justification for denial of funding in a corrupted peer-review process in more recent years.

Specifically, we **never** sat down together to go over one of **my** proposals piece by piece in a constructive manner. He would instead ask for electronic copies, in which he would make cursory adjustments. It seemed he was simply looking to see where I was headed in my thinking, rather than actually helping.

Rationalizations for changes that were made to proposals were not provided, and the critiques did not help to reframe the ideas in the larger context (grantsmanship). Instead, I was told that since I had been funded on an RO1 in 1999, I was really an expert independent investigator, who did not need such mentoring. In hindsight, many of his comments and directions can be construed as deliberate mis-guidance. *Only after the proposals returned unfunded was I duly criticized, and told that I should have known better.*

Moreover, except for serving on the California American Lung Association Review process early in my career, as far as I can tell, my name was not put forth to participate in national governmental or foundation-based peer-review processes. Since Dr Dubinett was institutionally referred to serve on foundations, the VA and NIH study sections (e.g.: after Dr Economou's departure), this sort of institutional promotion is commonplace, and that it constitutes a very important component of "research training". He clearly referred others in his charge to peer-review panels. This did not occur in my case.

In addition to the lack of instruction on grantsmanship and referrals for peer-review, I also received no instruction *in the pragmatics of competing for funds, the process of scientific review, or the most applicable funding mechanisms.* Because administrative support staff at the VA-site was also not trained in putting together grants, the added administrative work was particularly odious. Access to crucial core resources (e.g.: bioinformatics and biostatistics) to procure research funding was hampered. Such collaborative expertise, since not available at the VA, needed a green light from Dr Dubinett to be activated, and this was not forthcoming.

Although I often asked, I was never referred to anyone within the larger UCLA community to solicit their guidance with respect to proposal development. Thus, by being at the VA in Dr Dubinett's Lung Cancer Research Program, Dr Dubinett was the sole liaison for promoting effective institutional (UCLA) collaborations. And he had deliberately disengaged from this role.

My characterization as a “*trainee*” versus “*an independent investigator*” was freely interchangeable throughout the time period of my association with the UCLA Lung Cancer Research Program. I was a “*trainee*” when it came time to assign credit for the work *in which I had the most expertise*, and I was an “*independent investigator*” when it came time to allocate resources. While Dr Reckamp (and later, Dr JM Lee) received the public promotion, scientific mentorship, training, and networking through the UCLA-SPORE program, I was concomitantly denied membership to those inner circles. Thus, I was denied access to the resources and peer review forums that I had “earned”.

Instead of mentorship towards my career enhancement, I was instead assigned to tasks which only advanced his career and resources, and not my own. For example, after Dr Dubinett assigned Dr Reckamp to “my NIH-supported translational project”, instead of helping me with advance with the development of my own basic science project on the cell and systems biology of Adenoviral gene transfer, *he instead assigned me to a couple of biopharmaceutical misadventures (Titan, NovaRx). What these projects had in common was that they were highly time-consuming, had little academic value, and in my judgment, no clinical application potential.* But I took them on because I was assigned to them, and he was the Director. ***Importantly, the funds that came in through these private contracts were in Dr Dubinett's name and control.*** One possible interpretation is that with the private contracts (which I was specifically asked to write up), he had much more freedom, and he did not have to abide by the stringent auditing and financial scrutiny that was applied to NIH proposals.

Importantly, Dr Dubinett also kept me off all NIH-grants, even when they arose from my ideas or direct intellectual property. In this way he administratively reallocated resources and academic credit elsewhere, with a façade of legitimacy. This can be confirmed by examining both UCLA-Lung cancer SPORE proposals submitted to the NIH. In the first, he kept me off the personnel pages by advertising that it was a way of limiting costs; ***on the second***, although I had spent several months (with years of prior experience as a backdrop) principally developing the project for immunogene therapy, ***I was kept off all together.*** Thus, for reasons only he can defend, my role was administratively minimized or removed from the funding applications, despite my playing significant roles in the development of projects to the NIH.

Accordingly, career-development, promotion, advancement, and the equitable distribution of resources were not based on merit. Work done and resources collected at the VA could be easily redistributed for self-advancement or for promoting others at UCLA. It maintained a very comfortable financial bottom line for the executive, who could then use both the resources and leverage authority to get rid of the individuals he called “trainees” whenever there was no longer a need or desire to support their work or career. Early on, when my “research mission” was to translate gene therapy into the clinical setting, I was desirable. After Dr Dubinett wholly took the promotional and academic credit and financial windfall for that mission, I became undesirable. I had to go!!

Not only was I assigned to work on academically worthless corporate misadventures, I was similarly assigned to write up highly time-consuming projects, which also ultimately had little academic value. For example, I was asked to write up a comprehensive global review on the Gene Therapy for Lung Cancer for an NIH series on the subject in 2002-2003. The project, by itself, took several months to complete. I trusted his judgment, and believed that these tasks were being assigned because they were in my interest, not realizing at the time that in terms of academia, this was a dead end.

This is evidence of a deliberate abdication of an avowed mentor-trainee relationship, and his fiduciary responsibility. It is exactly these sort of actions that erode trust in the academic endeavor, and demean our profession. From my retrospectoscope (as I did not have the understanding I do now at that time), his actions clearly highlight that Dr Dubinett's interest in my recruitment was purely selfish. I was recruited for a highly specialized talent and expertise, but not for further academic development. *There was no reciprocating research training instruction provided.*

These actions were in direct contrast to our dialogues during my recruitment, when I had made it very clear to him that I was committed to academic medicine (by God! I had already spent seven years learning about and becoming proficient in "Gene Therapy" before coming here), and had the openly stated goal of making a significant and long-lasting impact on the treatment of lung cancer.

In retrospect, it would have been vastly better if Dr Dubinett had informed me of his rationale for disengagement, and helped me secure another academic spot long ago. For example, Dr Boucher, my previous mentor, had effectively told me to find another job after 7 years and high research output at UNC. Although it hurt, the action was based on rational thinking (UNC was not interested at the time in developing a gene medicine program in lung cancer), and was ultimately forgivable. In this case, Dr Dubinett simply continued to lead me on, continued to extract IP and telling me that better times were ahead, but then took actions in direct detriment to my career.

An assignment to an environment devoid of research training-oversight responsibilities was a primary example. Although the NIH (and VA) views WLA-VA and UCLA as part of the same "institution", *practically, this is not the case.* Because there is no overarching regulatory policy or research oversight, a crafty operator, like Dr Dubinett, can usurp and/or misappropriate all research work done at the VA. Because most VA investigators are not academic senate members, they are effectively indentured servants. ***This gives a senior investigator a huge advantage towards building an efficient, "cost-effective" program.*** Educated and talented individuals can be brought in "to UCLA", and assigned to work in an environment that is ill-suited for them to perform the work that they are trained to do, while University associated investigators are arbitrarily assigned "leadership" to serve as the key liaisons to critical infrastructure. Thus, the VA-environment effectively functions as a research "sweat-shop", easily enabling the intent of a "revolving door policy".

It leaves both UCLA and the VA "off the hook." The UCLA Academic Mission does not really have to account for VA-investigators, because they are part of a different institution (VA). In turn, the VA, which does not care about the research or academic mission, doesn't really care whether their "trainees" progress in their academic careers. ***This may be great for the UCLA Academic mission and the program director exploiting these circumstances, but it is very bad for the deliberately misinformed trainee whose whole life has been spent in research training to try and make a real difference in his field of interest.***

Because there is no unifying administrative research oversight, the unscrupulous program director is given a free pass in terms of accounting for effective mentorship at the VA. Dr Dubinett recognized (or was coached) quite early that it only matters how "trainees" are being treated at the University. Program directors (mentors) are much more closely scrutinized for acting in accordance with the accepted ethical and academic values at the University. ***Trainees at UCLA are treated vastly differently than trainees at the VA.***

By contrast, at the VA, the program director can also wholly control the attribution of academic credit of work that is performed, while being in a secure position to block "academic advancement" at will. *Furthermore, the trainee is left bewildered in a working environment where there is no real sense of what should be expected of the mentor.* (For example, even though I was given the title of an "Assistant Professor in Residence" in 1999, I did not receive the "UCLA-CALL" until Dr Fogelman referred me to this policy manual in 2012). I contend that Dr Dubinett exploited the conditions quite effectively, with pre-calculation.

However, the work that I was doing at the VA was NOT mediocre. For example, it can take 20 years or more to take an indigenously developed bench-based novel treatment strategy into the clinic; ***it took me eight years*** to formulate and submit an IND for the immune-genetic therapy of lung cancer. That time span could have been even shorter if the cGMP-viral vector for the clinical trial had been manufactured more quickly and efficiently by NIH-RAID mechanism. (The practical delay imposed by the NIH-RAID process may have been *fortuitous*; my translational and regulatory expertise was still needed when I came up for and was awarded “tenure” in 2005, which allows me to presently avail this Academic Senate process).

Although the VA is a great place to conduct healthcare services research and large cooperative clinical trials, basic and translational research is not well supported. *The VA is not a good place to do gene therapy or cell therapy research without tangible and effective University collaborations.* The VA process of grant review is also inadequate to gauge the novelty or ramifications of cutting edge projects (this is clearly evident by the process that discounted the novelty and significance of the projects we brought forth for their consideration between 2009-2012, even when it was clearly pointed out to the VA R&D leadership beforehand; this evidence can be provided if desired by the Senate).

As a result, although many on the clinical faculty at the WLA-VA are very highly educated and specialized, a minority survived, but only with supportive University ties. For many started off as great researchers, most went on to simply adapt to the clinical mission. Over time, most realized that the infrastructure is ill equipped, the internal regulatory mandates stifling, and workplace incompetence and apathy can hinder progress for serious biomedical research investigators.

Whereas the typical VA investigator is actually University Staff *with a joint appointment*, VAGLAHS (WLA-VA) is unique in being a “*free standing*” institution (for some investigators, anyway). Unfortunately, what this effectively means is that an unscrupulous university senior researcher who is assigned to serve on VA review panels by VA administration *has no vested stake in the advancement of investigators from his own institution*. ***A “trainee” is easily converted into a competitor (because the senior researcher derives no “indirect cost” benefits from having the VA investigator at his own institution funded), without the trainee knowing that he was now a competitor.*** I contend that Dr Dubinett made that calculation in his mind when he started to negatively bias the review panels at the VA. I was not informed; however, circumstances suggest that this is exactly what happened.

These problems with the UC/VA consortium are correctable with funding and resource support, ***but the lack of a uniform research understanding between UCLA and the VA makes that difficult.*** It may be difficult for some at the University to acknowledge (or perhaps not), but there is a clear stigma associated with VA-investigators when it comes to their requests for intramural support from the University, or for extramural resources at the State or National funding mechanisms. Personally, it seems that many investigators at the University assume that the reason an individual is assigned to the VA is because in effect, they are “second string”. The coach’s (program director’s) motives for assigning an individual to the second string environment is never questioned because it is ASSUMED that the program director is acting in the best interest of his program (but as mentioned, the VA-indirects go elsewhere). Personal motives underlying a negative rhetoric are generally not considered by review panels.

Thus, my initial assignment to the VA had placed me in a uniquely handicapped research environment. It may be hard for the UCLA-academic to fully comprehend this, but these are critical factors.

In addition, at the VAGLAHS, it is also impossible for the research ***trainee*** to internalize what their mission really is, or who is their real “supervisor” or “boss.” ***Whereas Dr Dubinett was the de facto “program director” in charge of my “academic career”, he washed his hands of all resource allocation that related to “training”.*** He informed me that this was the VA’s responsibility. The local VA leadership,

meanwhile, which has little comprehension of what it takes to succeed academically in translational (bench to bedside) medicine, is not equipped to direct and mold an academic career.

The day-to-day function and the mission priorities of the VA are very different from that of an investigator at the University. The VA's primary charge is to clinically take care of Veterans. It consumes the day. To perform this mission, the leadership constantly confronts a large gap between the personnel and funding resources with which they are provided, and the unfunded mandates with which they are forced to comply. Thus, the VA executive leadership is perpetually put in a state of juggling priorities and putting out fires. As such, there is a non-existent integrated basic or translational science infrastructure in this environment, and the ways and means to address this deficit never reach high priority. ***The university affiliate is supposedly contractually obligated to provide this environment, but as indicated, open access is blocked.***

It remains unclear why I, an avowed and committed translational physician-scientist upon my recruitment, was assigned to a position at the VA. In fact, I inquired about a shared appointment with UCLA (such as the one Dr Dubinett had) earlier, but when I raised the possibility of having part of my FTE be allocated at UCLA in the early 2000s, this was highly discouraged. "The departmental taxes" and "time commitment" would be too burdensome, and would distract me from my "research mission". At the time, "my research mission" was to translate gene therapy into the clinical arena (something for which Dr Dubinett ultimately took full promotional credit).

That the local VA leadership has little interest in seriously supporting the research mission became clear to me as I sought their help in my academic dispute with Dr Dubinett. "*You are on your own, because we really don't have a dog in this fight*", I was told by my then Department Chair (William Lance George MD). And after several years of passive non-intervention and/or ineffective intervention, "*you are welcome to take this up with the UCLA Academic Senate*", I was ultimately told by my Chief of Staff (Dean Norman MD) in early 2011.

That the ***National VA R&D leadership also has little interest*** in seriously supporting an *independent* research mission has also become clear to me as I have sought their help in my academic dispute with Dr Dubinett. The "independent" Merit Review Investigator is rhetorically "independent", and the Merit Grant is not a "Grant" after all, I was informed in 2012 (evidence can be provided as support, if needed). Despite the high innovation and quality of work, the real message emerging from the VA-R&D program is that my "*independence*" would not be supported by VA funds. Apparently I have been here to effectively serve at the pleasure of Dr Dubinett, irrespective of the significance, novelty, or clinical applicability and impact of the work.

Harm Done:

The effects of the abdication of **mentorship** linger to this day. Although I possess the aptitude, the vision, the scientific knowledge and technical wherewithal to be very highly successful, *I still lack a pragmatic knowledge of "grantmanship" (the ability to perceive what the study-section reviewer is thinking based on current knowledge in the field). Similarly, I have a lack of knowledge about the review and funding process, and I also have been deprived of the promotional support for effective networking amongst peer reviewers. These were (and remain) orchestrated handicaps*; such basic support has been afforded to others under the University's umbrella; in fact, "others" have been supported using the resources I brought in (with Dubinett as PI).

Since 2009, I have been trying to remedy the deficits in "grantmanship" by sheer trial and error, at great personal expense, and with the persistent lament that with his mentorship and promotional support, I would have been fully funded the last few years. ***I believe that with his support at the outset of my new venture, the whole institution would be in a better fiscal and promotional state for our cutting edge work in lung cancer.*** I have no doubt that if he had supported my scientific endeavors, we would have amassed tremendous financial and scientific acclaim by now. ***We would have been the first in the world to develop molecular signatures associated with the aggressive "lung cancer stem cell phenotypes", and I have voiced these sentiments both to him and the JCCC leadership.***

C) I charge that Dr SM Dubinett, in his supervisory capacity, devalued my professional competence not only for an appropriate initial assignment, but fair compensation and subsequent promotion; and I charge that Dr SM Dubinett used his position and powers of influence to coerce others to cause serious harm for arbitrary or personal reasons.

After my own RO1 in Adenoviral gene transfer ended in 2003/2004 (during which the vast majority of my time had been spent developing the regulatory- and implementation- infrastructure for the UCLA-SPORE Immunogene therapy project), I was informed that my ongoing promotion and professional advancement at UCLA would not take into account the five years of highly specialized work that had been done on behalf of the “team.”

And since I was not afforded the time or mentorship to successfully reapply for a continuation (to understand **why** Adenoviral gene therapy was not going to work), I lost my RO1, and my University grant supported income.

However, since I had been and was still participating in SPORE associated projects, I approached Dr Dubinett to help cover some of the lost income through the resources I had helped accrue, but was rebuffed. **My delta was my problem, I was told.** In retrospect, perhaps this was a signal for me to leave, but I was both too dumb to pick up on it, and too laden with personal responsibility (can elaborate, if needed) to act on it.

Given the facts that I had contributed greatly to the acquisition of a \$13 million dollar UCLA-SPORE, the Phase 1 clinical trial for immunogene therapy for lung cancer, followed through on projects that brought private funds in his name, helped train and bring Dr Reckamp up to speed on clinical translation, and generally helped bring UCLA on the map with respect to the innovative study and treatment of lung cancer, I felt that this rebuff was highly *unethical and cruel, especially given his deep knowledge of the personal life circumstances I was facing.*

In the 6-7 years of working together, although I inherently knew that I possessed a deeper understanding **of the disease process** that we were both studying, I had also come to appreciate Dr Dubinett as a crafty, and “mercenary” (his term) administrator and team leader. However, till then, I always felt that we were on the “same team”. It was only then, for the first time, I realized that his gentle exterior was a cultivated external façade for the rest of the world (including me), and recognized that he who was not the team leader I believed him to be.

However, professionally, I also recognized that I had little option. Whereas all earlier “team ventures” had been undertaken under a functional “team revenue” paradigm (with funds frequently outgoing from my purse because Dr Dubinett had effective control of funds both at the VA and UCLA), Dr Dubinett had cut me off with a single VA-Merit Review in my name. I was left without mentorship at a time I desperately needed it, because I already knew that my original plans to take adenoviral gene therapy into the clinic were not likely to work, and “my” clinical project had already been reassigned to Dr Reckamp.

What these historical recollections indicate is that Dr Dubinett had carefully cultured a dependency on his thematic input, and on his mediation to develop effective collaborations at UCLA. So not only did I lose credibility in gene therapy by my projects being assigned elsewhere, my ability to get his critical input with respect to contemporary reviewer perspectives, or to get effective UCLA-collaborative input on independent basic science projects were curtailed. I was left completely stranded at a time when I *had to* come up with something entirely new, on which my research career now suddenly depended.

After I was forced out of the UCLA Lung Cancer Research Program in 2007, I came to learn that Dr Dubinett was propagating the narrative that my forced removal from the UCLA Lung Cancer SPORE program was because *I was a miscreant*, and that he “does not like me.” He impugned my competence and/or ability to function as a team player. That he was promulgating such a narrative was first inferred from conversations with Drs Gasson and Herschman at the JCCC in 2008, and later reaffirmed through dialogues with professional colleagues at professional society meetings (ATS) and more recently on my lecturing circuit. There are many possible explanations for his rationalization of past events, which only arose after he observed that I had begun pursuing an independent course of study, and I was starting to have some success at it. He had utilized a similar misinformation campaign in getting rid of another long standing and very hard working junior colleague whom he never promoted (Dr Mariam Dohadwala; after 10 years, he decided that he “could not count on her work”).

Thus, to provide cover and rationalization for his actions, he deliberately and maliciously damaged my academic credibility by an unopposed, fabricated narrative that I am a miscreant, who is incapable of providing value to the academic mission of the University

But this narrative is a lie. Even people in his own lab with whom I have directly interacted can attest to that. In fact, ***his academic*** record will indicate my critical participation in the UCLA Lung Cancer Research Program when it was at its finest. I enhanced its output and scientific credibility, and I was (and am) well liked by those who have directly worked with me. I was and remain a prototypical team player, and most members of *his* lab (which was “our” lab at the time) turned to me for advice with the writing and framing of their science in manuscripts. Those manuscripts were presented to Dr Dubinett when ready for submission. In addition, my free exchange of ideas and intellectual contributions in team meetings were often promoted for independent project development (but always under *his* direction). This is evidenced by the fact that ideas I proposed were used to procure funding and to advance the career for others (most notably Min Huang). Dr S Sharma, who has been a long standing colleague to both Dr Dubinett and me, can independently attest to this assertion.

Moreover, because Dr Dubinett was the primary beneficiary of my work, it imposed an inherent conflict of interest in his appraising my competency and expertise. And because he was (and still remains) in a position to have his narrative heard, this orchestration of events had a severe adverse effect on my institutional and national credibility.

Because my contributions were devalued, I was denied fair compensation for a very *highly specialized training* that culminated in a huge fiscal, scientific, and promotional windfall for him, and the University. Because work was usurped or misappropriated, my academic promotion was also suppressed. It is noteworthy that individuals (e.g.: Dr Ribas) who started off at a lower rank and who greatly benefitted from my tutelage and free exchange of ideas and expertise have since risen to positions of leadership within the cancer center and the institution. Their academic rank, credentials, and name-recognition now far surpass mine. ***I assert that the key difference has been “mentorship”, and a nurturing environment more laden with promotional opportunity.*** In this regard, the initial reassurance that an academic position at the VA was equivalent to an academic position at UCLA was blatant misdirection; an assignment at the VA simply allowed a shrewd and unprincipled administrative officer at UCLA to usurp work done off-site without due credit, and then to apply the “revolving door policy” to create a hostile environment in hopes that I leave, so that he is able to cover his trail.

By usurping and/or redistributing academic credit, Dr Dubinett deliberately suppressed my promotion and name recognition. Reviewers of grant applications and publications came to associate the academic credit with Drs Dubinett, Reckamp or Lee. After having spent 8 years in the background, my proposals came to be viewed with incredulity not because of work proposed, but because I was never promoted as the original architect of that work. This sense of incredulity became clear much later in dialogs with NIH-program officers, who had no knowledge of my prior contributions to the UCLA lung cancer research program.

Not being funded despite the quality and innovation of our work, I appealed an earlier biased VA-Merit Review funding decision in 2009. Then (and now), the review and grant scoring process had been orchestrated to give an outward appearance of legitimacy, but with prejudice against my applications. For example, my proposals have been “scored” arbitrarily low for the last seven, and have been systematically triaged the last four VA Merit cycles (although I can unabashedly claim that I am an expert on the cell and molecular biology of the molecule I had proposed for study during the last four cycles; refer to Dr A Jewett in UCLA-Dentistry, who is a collaborator on projects being developed presently).

The reasons underlying denials of funding are unclear, and are masked by the *VA-process of review*. This is a contrived process, in which members on the review panel (many of whom have been referred there by Dr Dubinett) can “select” my grant for review. For the last seven cycles, the contents of the reviews consistently belie a fair process, and fail to acknowledge that I know more about the functional ramifications of the protein I have proposed for my studies (the cell adenoviral adherence receptor or CAR) than nearly anyone else in the world. By denying me due credibility, and by repeatedly triaging my proposals, the reviewers also bypass sympathetic members on the committee who may rescue them. *Any grant can be criticized, scored arbitrarily low, be unintentionally misinterpreted, or be intentionally misrepresented. Any or all of these possibilities seem be applicable in the corruption of the process.*

That Dr Dubinett has great sway with the Review panels AND the Central VA office of research and development administrators who oversee the reviews is a matter of fact, not conjecture. As a complainant who has brought to the attention of the VA-Office of Research and Development that Dr Dubinett may have a conflict of interest, I have been dealt with very harshly. I have been shunned by the leadership, berated for questioning Dr Dubinett’s motives, and denied due process. *Ironically, Dr Dubinett has instead been asked to review and fix the concerns that I and others have raised regarding fair and equitable programmatic review.*

Dr Sherven Sharma participated in a recent review on the study section that reviews my proposals. He indicated to me that given the quality of the proposals that were getting funded, mine should have been funded long ago. He also acknowledged that it was my principled conflict with Dr Dubinett that was underlying the block. He confirmed Dr Dubinett’s unquestionable power and influence at the National VA panel. He is “revered” at the VA-ORD. On the peer review panel, many members had him as a paid consultant, or were collaborators. Dr Dubinett was (is?) a “permanent member” of the Oncology review panel, and over the years, he had developed and nurtured a close relationship with key other “permanent members” on the board. One of those individuals (e.g.: Dr A Lichy) was evidently responsible for killing my 6/2009 Merit Review application (see below).

Dr Dubinett also holds such sway because he has been in a position to provide a platform for the VA Research Leadership to highlight their research portfolio at National Academic Societies (e.g.: ATS), in an effort to raise VA research awareness. So his influence at the central VA research hierarchy is unquestionable, and his surreptitious negative influence there has dealt a grave blow to my continuation of independent ventures through the VA.

Dr S Sharma’s own situation is a direct testament to this assertion. Dr S Sharma, after 4-5 cycles of being non-funded, finally gained funding by adding Dr Dubinett as a “co-investigator” on his grant. This course of events has only served to heighten his anxiety for survival of livelihood on a Dubinett-dependency, and coincidentally, this now gives Dr Dubinett legitimacy for pillaging his VA-resources.

Early after being dispatched from the UCLA Lung Cancer Research Program (in 2007), I was still extremely bewildered and shocked at how my career had come down to where it was. I was very angry at myself for choosing academia because despite my knowledge base and high level of training, and despite following the rules of working hard and completing the assigned tasks, I felt swindled by a smooth talker and unethical

operator who was institutionally **promoted** by his orchestrated subterfuge. But by mid to late 2009, things were completely different. By then, I had spent some time as an elected member to the UCLA Department of Medicine Committee on Appointments and Promotions, and better understood how my downward spiral had likely occurred. It was not till I served on this committee that I came to appreciate the tremendous power of "**individual promotion**". *The framing of credentials by committee members was the key to the success of individual candidates.* I also came to see firsthand how that framing could be promoted, or disparaged by individuals who were in the committee and in positions of authority (both directly and indirectly). ***The actual work that candidates were doing, and the environment in which they accomplished their assigned tasks, was rarely considered by committee members. Moreover, issues related to individual opportunity or mentorship rarely arose as significant contributory variables.***

But by 2009, I now also had a different problem to contend with.

Extramural funding is the life blood of academic medicine, and there was an accruing body of evidence that **my funding was being actively (directly or indirectly) blocked by my former mentor**. Whereas some of my earlier proposals mostly suffered for lack of mentoring/grantsmanship, since 2007, I was coming to the realization that many of my efforts to procure resources for my work were being deliberately blocked. ***This block had become been particularly intensive since a highly impactful PLoS ONE publication in 2009*** (<http://dx.plos.org/10.1371/journal.pone.0005884>). This concern was prospectively raised with both the VA ACOS for Research and Chief of Staff (DR Dean Yamaguchi), and with Dr Gasson (Director, JCCC) shortly thereafter.

So what makes me think that Dr Dubinett was directly/indirectly orchestrating this block?

1) I am not the first individual to have believed that they had a friendly mentor/colleague in Dr Dubinett, only to be embroiled in career altering quarrels later. There may be others, but Drs Michael Roth (UCLA), Jenny Mao (now at University of New Mexico), Dr M Dohadwala (relocated to India, I believe), and Dr S Sharma (VAGLAHS) can be independently consulted about their experiences; I know of their cases peripherally.

The outcomes of these quarrels have been different, depending upon the institutional standing (power) of the individual engaged in a quarrel with Dr Dubinett. I think that Dr Roth fared best. Dr Donald Tashkin was able to mediate the dispute, and each investigator was handed over "control of an academic arena" (Dr Roth received scleroderma associated interstitial lung disease, and cannabinoid biology; Dr Dubinett received lung cancer). (I approached Dr Tashkin requesting his mediation in 2010, but he felt that Dr Dubinett was beyond his influence). Others were not so lucky as to have a senior professor mediate effectively on their behalf. Dr J Mao was dispatched to New Mexico. Yet others were unluckier still. As indicated, Dr Dohadwala was sent packing back to India after the loss of promotion, broken promises regarding visa status, and the creation of a very hostile work environment. I served Dr Dubinett faithfully till 2007, and then tried to disengage peacefully to pursue an independent course. But I had been made a pariah at my own institution.

But the unluckiest of all, I think, is Dr S Sharma. He has felt obligated to succumb to Dr Dubinett, and to spuriously turn against good friends (see below) in order to appease a boss who he knows has tried to get rid of him for several years, but on whom he presently feels wholly dependent for his funding and livelihood.

2) Through Dr S Sharma, in 2006, I was first informed that my quest to develop an independent program would be met harshly; an effort would be put forth to "bury me". In fact, I was told that Dr Dubinett was "having regrets about promoting my Merit Review Application for continued funding in 2005" (this also further corroborates Dr Dubinett's influence on VA funding). But by 2006, since I felt that I was already being treated as a competitor rather than a "team member" anyway, I forged ahead with building an independent program to study "lung cancer stem cells". Given Dr Sharma's warning, however, I became quite concerned, but felt that if

I developed and implemented a novel scientific program, I could win both his and UCLA-administration approval.

I was also concerned that the resources I had at my disposal may not allow me to fully develop the plan I had in mind, ***but because the concept was totally separable from any prior work affiliated with Dr Dubinett, I felt I had to develop it.*** As things turned out, however, my developing a novel approach simply hardened his self-preservation stance, and it seems that the institution was more concerned with preserving the hierarchical status quo rather than developing a promising new strategy to effectively deal with lung cancer.

So what made me consider developing something new, which had nothing to do with my prior work?

3) In 2007, when we were just getting started on our “lung cancer stem cell” project, I submitted a proposal entitled “Molecular markers of TZD-chemoprevention effect in lungs” to the CA-TRDRP (administered by UCOP). Dr Dubinett was consulted about this submission, and had knowledge of the submitted proposal. The TRDRP-review panel for that proposal included Dr Y Miller from the University of Colorado, (a friend and likely referred panel member by Dr Dubinett). ***Whereas my proposal was not funded, an investigator (Dr Robert Keith) who was working with Dr Miller submitted a very similar proposal to the VA the very next cycle, and was funded through the VA-Clinical Merit Review Award.*** I found it difficult to believe that Dr Keith independently came up with the theme and similar trial design. Given the friendship between Dr Dubinett and Dr Miller, I also found it difficult that Dr Miller would have shared the proposal with Dr Keith without conferring with Dr Dubinett. (In 2012, I had an opportunity to ask Dr Miller about this co-incidence, but he said that he did not review my proposal to the TRDRP).

That proposal (funded by the VA) effectively launched Dr Keith’s career. He currently sits on a key foundation (LUNGevity) that reviews lung cancer proposals with Dr Dubinett, and is the current Associate Chief of Staff for Research at the Denver VAMC. ***This incident suggested to me that Dr Dubinett was willing to give away work and an idea that was not his to curry favor with lung cancer investigators at other institutions.*** It was an effective, but to me, a highly irregular way to build a friendly external review network around the country. It confirmed my growing suspicions that Dr Dubinett felt that he had nothing to gain by my being funded.

4) In 2003, an interesting article had appeared in PNAS. A group at the University of Michigan (UM) demonstrated that a subgroup of cells in malignant pleural effusion (MPE) from breast cancer biospecimens were more tumorigenic than other tumor cells in those effusions. ***I am an expert in malignant pleural effusions,*** and through extensive past work at UNC Chapel Hill before I came here, I also knew how to generate primary cultures from these advanced stage tumors. I had earlier recognized there was significant morphological and antigenic heterogeneity in the MPE-tumors that were derived from lung cancers, so what the UM investigators had discovered in breast cancer was quite possible for lung cancer as well. However, whereas the UM investigators believed that they had identified “breast cancer stem cells”, I wasn’t convinced that “cancer stem cells” existed.

Because of my life circumstances, I was intensively studying Autism as well. This study introduced me to the concept of “*endophenotypes*.” This concept predicts that one can use biological or behavioral biomarkers to segregate individuals with autism into discrete subsets. By doing so, it was believed that one could get closer to the genetic and epigenetic underpinnings of specific disease-associated behaviors/phenotypes.

I simply extended this *endophenotype*-concept to predict that the greatest utility of the approach really lay in the study of cancer. I proposed that given our unique ability to separate cancer cells of *individual patients* on the basis of reproducible and *quantifiable differences in behavior*, we may be able to efficiently derive the genetic and epigenetic *drivers of specific tumor cell behaviors*.

I feel strongly that this is a game changing idea. I decided that we would use cancer stem cell biomarkers to begin to isolate and validate the aggressive phenotypes in MPE lung cancer biospecimens. I went about getting IRB approvals for conducting the project in 2005-2006, and began collecting, processing and culturing MPE-biospecimens in 2006. Since this was a totally novel experimental paradigm, there were no pre-established rules to follow, and lots of troubleshooting to satisfactorily establish primary cultures in a reliable manner. But as indicated, I had much familiarity with the MPE-model system, since this was the clinical target for a lot of my earlier gene therapy work at UNC-Chapel Hill.

This endeavor was based ENTIRELY on my own past and prospective work. We chose an efficient experimental paradigm to derive *key proof-of-concept goals* with less than \$500,000.00 dollars between 2005-2009. Given the initial plaudits we received after our feasibility publication was accepted, *we fully expected VA-funding for our work to continue*. But we were denied.

Our A1 VA-Merit Review Application was submitted in 6/2009. Shortly after submitting the proposal, we also submitted our accepted PLoS ONE manuscript for disbursement to the reviewers, and received confirmation from the program officer. Thus, we were quite puzzled when after having received plaudits for our novel work by the head of VA R&D, and confirmation from the program official that our accepted manuscript would be considered during the review process, we were notified that the proposal was not funded. *We were further dismayed to learn that the chief criticism that arose in discussion was that this PI had not published in the arena.*

It appeared that despite an a priori knowledge of published work that was to be distributed to reviewers, the VA program official did not distribute the work to reviewers, and also did not correct the factual error that the manuscript was in progress during the panel discussion.

A few weeks later, I received an anonymous phone call. The caller informed me that he had learned from incredulous committee members exactly what had happened. The summary score that I had received (297) did not reveal the real story. In fact, the primary and secondary reviewers had scored the application in the 150 to 180 range. It was a tertiary reviewer who arbitrarily gave a score of 450, even while acknowledging that *his earlier concerns were met, and that the score he was giving now was worse than the score he had given the original application* (before revision). This tertiary reviewer (Dr A Lichy), a “permanent member” on the VA Oncology study section, also successfully argued others to downgrade the proposal.

Importantly, I was also informed that this tertiary reviewer was a close friend and contact of Dr Dubinett. Thus, an anonymous source suggested that it was my former mentor who was directly, or indirectly (through Dr Matthew Rettig, who chaired that review panel) blocking our efforts at getting funding. We filed an appeal with the VA medical research program, but it was summarily denied. The subsequent A2 application scored better, but remained unfunded. .

5) After having failed three times to garner funding for our innovative work, we were advised by the VA program officer that we had to submit a proposal on an entirely different theme. In 2010, we began our odyssey to revive our funding through a proposal on the coxsackie-adenoviral receptor (CAR)-biology. I feel strongly that I know and understand CAR biology in lung epithelial/cancer cells better than any other investigator in the world. Again, despite answering all scientific critiques, we remain unfunded after four unsuccessful cycles. More remarkably, the recent review process (last four cycles) has been corrupted to make sure that none of the proposals even make it to the floor for discussion. As described earlier, the last four proposals have all been triaged, to ensure that committee members sympathetic to my cause are not allowed to sway the vote. The reviewers take special care to repeatedly disparage the PI, and to point the critiques in disparate directions to make a response statement impossible.

6) Over 2009-2010, Dr S Sharma's case provided an interesting comparison. For several cycles, he too was going through inexplicable denials of funding, with a series of reviews that were directing him in opposite directions, and chastising him that he was not "collaborating with known lung cancer experts" locally. He informed me that only after he added Dr Dubinett on as a collaborator did his proposals move forward to funding. Interestingly, although the content of the reviews remained negative, the score was fundable.

While waiting to hear whether he would get funded, Dr Sharma became ill with worry, and lost a lot of weight over the last couple of years. He received a "pink slip" through Dr Yamaguchi (ACOS Research VAGLAHS), and I suspect that in the "terminal phase of his employment" in 2010, it was my residual funds from the 2005-2009 Merit Review that covered his salary support.

The last point illustrates that Dr Dubinett did not intervene to save Dr Sharma's career after 15 years of service, and did not deliver the bad news that his career was being terminated. It confirmed to me that in his mind, both Sherven and I were expendable, indentured servants. The episode also illustrates how the VA administrative leadership may arbitrarily *shift grant funds around without PI consent*. Although I would have willingly allowed my money to be used for Sherven's salary support, the money was simply absconded (and even though Dr Sharma is now funded, it is not clear whether the monies taken from me will ever be administratively restored).

7) I have continued to apply for funds continuously, but the efforts have been futile, without scientific justification. For example, over the last year, I have had two LOIs rejected (without invitation for full submission) by the LUNGevity foundation, three LOIs rejected by CIRM, an American Thoracic Society A2-grant resubmission was rejected without scientific basis, 3 different Merit review and Applications were rejected, and in 5-2011, an A2 application to the California Tobacco Related Disease Program (TRDRP) was not funded.

The fact that the TRDRP-application was not funded came as a big and unpleasant surprise; ***the same application was earlier linked with a summary score in the "exceptional" range on the Altum Proposal Central website***. Since the TRDRP-program is administered by the University of California Office of the President, and since Dr Dubinett is now Vice Chancellor for Translational Medicine at UCLA, we have a lingering concern that influence-peddling through the Chancellor's or DGSoM Dean's office played a role.

Moreover, since Dr Dubinett also sits on the scientific advisory board of ***all of the named foundations that fund preliminary work on lung cancer***, he has considerable influence in the administration of those funds, and towards recommending panel members for the review process. We have previously cited concerns about *quid pro quos* and influence-peddling as exemplified by the Dr R Keith case, so we are reasonably drawing a conclusion that based on the animosity he has displayed towards me and my program locally, he is similarly doing so at the level of the peer review process. .

Harm Done: Collectively, these circumstances indicate that Dr Dubinett has been at key central chokepoints and in influential positions to block funds, and he has done exactly that for the last three years. In light of past circumstances, perhaps it was with the *a priori* knowledge that he could effectively control the central access point that Dr Dubinett assigned me into a VA position at the outset. This gave him an insurmountable advantage in controlling the direction of my career, and when he decided to kill it, the process was streamlined to make it easy.

The harm done by instituting this block cannot be overstated. Briefly, I have had to suffer not only the loss of key personnel whom I trained and developed between 2006-2010, but we are fast losing the competitive edge on our work. The block to funding has prohibited us in our ability to collect additional key preliminary data for more substantive finding towards clinical translation, and huge fiscal benefit to UCLA. And from a personal

standpoint, the block has been instrumental in having the executive administration justify its actions in not providing an appropriate TNS.

Since 2004, I have repeatedly approached the VA (and UCLA) executive leadership to live up to the terms of my hire. ***Throughout this time period, I had been told that my request was not meritorious because I had not been able to secure funds through competitive peer review.*** However, even when I have pointed out to them (since 2009) that the process was corrupted, this mantra has persisted. ***In effect, the executive leadership has orchestrated and implemented a “futile cycle”.*** I am being repeatedly asked to address reviewer concerns (that point in different directions cycle after cycle) with no resources, all the while negatively influencing a pliable peer-review process to not fund our work, and then using that to deny both academic promotion and livelihood.

D. I charge that Dr SM Dubinett participated in intimidation and the orchestration of a severely hostile work environment.

By 2009, I had come to KNOW that the work I was developing was very important, but that Dubinett was obstructing our efforts. By 2009-2010, Dr Gasson (JCCC Director) had also openly recognized that I was on to something different, and important. However, I did not have the open collaborative access available to help me refine the presentations of my proposals. Upon my request, Dr Gasson first referred me to Dr Lange in the Department of Genetics, who also seemed impressed with the approach I had developed, but that initial enthusiasm and impression seemed to inexplicably wane. To date, the “collaborations” (***excepting Dr M. Pellegrini***) have largely been rhetorically enthusiastic and supportive, rather than intellectually or materially helpful.

In June or July of 2010, I received a surprise phone call from Dr Strieter (the former pulmonary Division Chief at UCLA) while I was in Chicago. He indicated that he was familiar with my work, and advised me to go and speak with Dr Dubinett. He suggested that if “I ever wanted to get funded in lung cancer”, I would *have* to go through him. He also suggested that I take a subservient tone in my conversation. He also informed me that Dr Dubinett was the institutional designate for the CTSA application, and that given the institutional knowledge gained from past failures and the support the was receiving towards securing that grant, he was likely to get it.

Upon returning to LA, I did as prescribed. At that meeting, Dr Dubinett acknowledged that what I was working on was very important and competitive. He compared my quest to that of Dr J Folkman, (a man whom I idolized, and with whom I had directly spoken about his past experiences on two occasions, one at the VAGLAHS by coincidence). Thus, I knew that while he was not getting NIH funding for several years, Dr Folkman was being heavily supported by the local infrastructure at Harvard.

The problem with this false analogy was that in this case, the institution (Dr Dubinett, by default *the lung cancer expert*) seemed to be blocking tangible support, while jointly indicating that he thought the idea important and worthy. ***His denial to support our emerging work really confused me.*** Even I knew that the program I was/am developing would place the institution *at the forefront of research towards developing a better understanding and treatment of lung cancer*. However, the single most recognized institutional authority on lung cancer was unwilling to provide support, while recognizing that the work was innovative and important.

Why would he do it? Earlier, I would have thought that it was simply finances. ***It was clear that my Merit or RO1-funding had little to no impact on the take of direct and indirect costs for his Division at UCLA***, so it was no skin off his nose to kill those endeavors (the “VA-indirects” simply go to the CEO at the VA). However, since big NIH-money was likely around the corner, and he was working toward the CTSA and Vice-chancellorship, his denial of support made no sense.

Thus, after the July 2010 meeting with Dr Dubinett, for the first time in over a decade of working with him, *I openly vented my concerns to outsiders (Dr J Gasson)*. After a series of e-mails, she responded by referring me to Dr F Fawzy (see attached communiqués). ***I specifically wanted to iron out our differences before the institutional clinical translational award that Dr Dubinett had applied for as PI was funded, and would become a corrupting influence on an equitable mediation between us.*** Ironically, now many months later, that is exactly the compromised position in which I currently find myself.

I met with Dr Fawzy in mid-December of 2010, exchanged my concerns and perspectives over a broad range of topics. I thought that the meeting had been substantive, though emotionally draining. I was there to seek his administrative help in mediating a long-standing unresolved conflict that I felt was making my former mentor act irrationally. I confided to him much of what is presented in this letter. There were issues of redress; and there were issues from which relief was desired. ***Chief amongst my concerns was the perceived block to funding mediated by Dr Dubinett*** (and that perception was even stronger by now). To negate that block, I asked Dr Fawzy to set up mediation to determine whether Dr Dubinett would consent to support my emerging program with \$100,000.00 in seed funds. The rationale was that if Dr Dubinett invested his own money into the program, he would also support it extramurally. The funds were not for my salary support, but to keep a nascent but important program afloat. The meeting ended with Dr Fawzy setting up a date for us to meet in early January 2011. The goal of the intervention was to try and hammer out a prospective agreement that would result in a “win-win” situation for both the complainant and the University

Again, the key reason I asked for these funds was to tangibly invest Dr Dubinett in my program. He had already conceded the work was important, and he knew that I had the wherewithal to accomplish it from my past work. By having him tangibly invested, ***I wanted foremost to negate the negative influence I believed he was exercising in the equitable review of our work***, and hopefully, persuade him to back the program.

The meeting was subsequently canceled by Dr Dubinett. ***Instead, in early January, with the coerced help of my long-time friend and colleague, Dr S Sharma, he filed a spurious criminal “harassment” complaint with the VA police.*** This abuse of legal process went nowhere: no impartial investigating body would move forward with such a groundless allegation.

Importantly, a day or so before I was called in to the VA-Chief of Staff (Dr D Norman) office regarding the complaint, I had a chance encounter with Dr Dubinett and Dr S Sharma in the VA-lab. I remember it because this was the first time in years that I had seen Dr Dubinett at the VA lab. I would later come to learn from Dr S Sharma that his appearance was to direct a plan for how to present the fabricated charges against me to the VA-associate chief of staff for research (ACOS research, Dr D Yamaguchi), and to the VA police. Thus, by the power he wielded, Dr Dubinett coerced a long standing friend and colleague (Dr S Sharma) to fabricate a criminal charge against me.

This act hardened me. It strengthened my belief that I was dealing with a highly irrational and highly immoral psychopath. Whereas the supposed reason for filing this charge against me was “fear of harm”, it was actually Dr Dubinett who was now displaying overt animosity and aggression. A couple of weeks after filing the harassment charge, Dr Dubinett also tried to have me forcibly evicted out of my VA-research lab space. Not having dealt with this sort of situation before, it took me a few weeks (and much emotional turmoil) to realize that perhaps, all this was an act of intimidation and aggression. But this strengthened my resolve even more to fight it (him).

Since then, I have spoken out about my scientific differences and mutual mistrust regarding Dr Dubinett with several university leaders (Dr O Witte, Dr A Fogelman, Dr Robinson, Dr Rome, Dr Gordon, Dr Hiatt and the Ombudsman) in order to affect a resolution, and to bypass his severe irrational block to our (and the University’s) opportunity.

Harm Done:

There persists an ongoing block to renew the development of a World leading Lung Cancer Research Program at UCLA. But the creation of a severely harsh (beyond the “institutional pariah” or “lone wolf” characterizations of earlier acrimony) had left me hardened to pursue justice and fair play. These events have provoked SEVERE personal and marital stress (requiring counseling), in addition to loss of livelihood. Over the years, the executive shenanigans and orchestrated “futile cycles for gaining extramural funding” (which have been used as justifications for denial of TNS support) have not only left me in a huge personal debt crises, but I have actively blocked from correcting those deficits based on my own work.

E. What is the future of my research vision at UCLA or elsewhere?

Over the last year, there are several indications that Dr Dubinett does not favor the ongoing development of my program.

Given my long association with him, I have come to recognize his mode of action. In an effort to gain an aire of “plausible deniability”, Dr Dubinett sends messages through intermediaries/emissaries (e.g.: Dr J Kaunitz, Dr M Zeidler, Dr S Santiago). In 2011, Dr Kaunitz and I spoke shortly after the fabricated harassment charge against me was formally dismissed by the Los Angles DA’s office. He informed me that it would be in my best interest to stop my research, and to stop seeking mediation because “they will find a way to get rid of you”. I indicated to him that I was not working not out of malice towards anyone, ***but to provide a direly needed livelihood for my family, and to fulfill a calling.*** Another emissary (Dr M Zeidler) posed the same question after my recent Merit Review was not funded. Perhaps, if I change my field of research away from lung cancer, it will be supported.

Concerned that my vision may have no future at UCLA, I attended our annual professional society meeting (American Thoracic Society, 5/2011) in Denver to test the waters elsewhere. ***At that meeting, I was surprised to learn through professional colleagues that although Dr Dubinett had privately acknowledged the importance of our work, he was actively and publically disparaging the idea.*** This was disconcerting, only because his opinions seem to matter in the field.

In April or May 2011, I sought help from Dr Fogelman, who was the first individual after several years of solicitation to provide some intramural support to our program. Dr Fogelman posed an important question in our dialogs. ***Why would Dr Dubinett block opportunity and request that I stop my innovative work when he had recently garnered a large CTSA?*** Why was there a persistent block to my Merit Review funding (in 6/2011 and early 2012)?

There are many possible explanations. A possible answer is that Dr Dubinett had pre-decided long ago that he had extracted all the info and work he could from me, and now he finds it difficult to admit an error in judgment regarding my “academic potential or scientific acumen”. Perhaps, it is simpler to continue to try to kill my program rather than deal with issues of redress for past misdeeds. Perhaps, the real reason he wants me to stop what I am doing is because over the years of dismissing my approach, ***he has been publically promoting and materially supported an alternative but fundamentally flawed experimental paradigm by another investigator at Boston University.***

On April 28, 2011, I attended an educational Medical Lecture at CHS. Dr Dubinett introduced the speaker with high aplomb, and showered praise upon him as an established star in the field. This established stardom was based on recent publications in high quality journals, but mostly on the fact that the speaker (Dr A Spira) had recently received a big \$15 million dollar DoD-grant. The speaker then got up and shared his mutual affection for Dr Dubinett with the audience. He reminisced about how he met Dr Dubinett, at a Scandinavian hotel, where they had shared science and bad food together. He profusely thanked Dr Dubinett and Dr Minna (a

leading long-standing authority in the lung cancer field) for help with getting the DoD grant together, and he thanked Dr Dubinett's staff members for their help in making last minute changes in the proposal.

Why would Dr Dubinett not only promote, but provide mentorship along with material and resource support to an investigator at another institution?

Perhaps, the real reason is that now that Dr Dubinett has established himself as a translational research vice-chancellor, he wants to further solidify his base by reaching for the Chair of the Department of Medicine (I had learned that Dr Fogelman was contemplating stepping down in 2011). He may want to leave a Divisional legacy as the replacement Chair, and I suspect that Dr Spira is that preordained candidate for the Chair of Pulmonary and Critical Care Medicine. Certainly, he has a 15 million dollar grant to his name; a grant that he has acquired through Dr Dubinett's mentorship and influence. Certainly, that grant is likely to garner matching support in "recruitment." But there are a couple of problems.

First, ***the idea is fundamentally flawed.*** Defining biomarkers of progression through blind (airway or nasal biopsies) screening for genetic mutations and expression profiles is highly unlikely to tell us anything about progression, no matter how sophisticated the biostatistics are in analyzing the data. ***Our idea is much better.***

Second, if that is the agenda, then where is the academic redemption or societal benefit in this subterfuge? Is the academic promotion of a known faulty idea acceptable if the Vice-chancellor deems it so? Is the promotion of diversity in the upper eschelon of the University leadership hollow rhetoric? ***Why would one artificially drown out an internal candidate who has developed the most innovative approach to the study of lung cancer to date?***

From another personal standpoint, although I am very grateful for Dr Fogelman's intervention for transient financial support with the Dean's office, an important idea that first took root at UCLA (VAGLAHS) continues to languish at UCLA, even as competitors half-way around the world move towards making and exploiting those key discoveries. Thus, the restoration of our program, and acquisition of key resources needed to bring it to full matriculation remains our top priority.

In summary, I believe that Dr Dubinett is the anti-thesis of what constitutes a "Professor of Medicine", and an "upstanding academic leader". I believe that such labels and titles have meaning and value, and think that he does not deserve them. He is simply an opportunist who is intellectually inferior, but who has been promoted to a high station through institutional cronyism. Unfortunately, he does not seem to have the emotional security or the moral fortitude for that post; I think that UCLA deserves a better person who is ethically equipped to support the *sanctity* of the University Academic Mission, and to provide benefit to the institution and the public at large.

SUMMARY of GRIEVANCE: (Previous iterations provided to Senate leadership in 7/2011 and 3/2012).

I contend that I was wrongfully taken advantage of by my supposed mentor. My work and expertise was usurped or misappropriated, and mentorship responsibilities were abdicated from the time of my recruitment in 1998, till 2007, when I was forced to withdraw from the UCLA lung cancer research program. Further, since then, my recent efforts towards academic independence have been blocked by my former mentor. In effect, it seems that my academic career has been targeted for elimination by the very person who earlier benefitted most from my work and expertise. With great reluctance, I make this formal complaint to seek relief and to restore my academic credibility. I take this action with great trepidation, because it is unclear to me whether Dr Dubinett as an individual is actually separable from Dr Dubinett, the institutional steward of translational medical research for UCLA. Moreover, to get to this position, he has been backed and promoted by many institutional titans. Dr Dubinett has been sequentially “mentored” by Drs S Korenman and G Levey over the years, and counts many members of the former (and present) Dean’s staff as personal friends and collaborators (e.g.: Drs Rome and Robinson). The Vice Chancellor for Research at UCLA (Dr J Economou) and the directing leadership of the Jonsson Comprehensive Cancer Center (Drs Gasson and Herschman) have been strong collaborators, promoters and backers for over a decade.

Thus, the truth of the matter is that I have been pre-warned that given Dr Dubinett’s current status, he is “untouchable”, and that my going forward with this academic senate process is not likely to be useful because irrespective of what the senate decides, the final adjudicator of his actions is the office in which he presently resides. I hope this is not the case, and that a fair judicial perspective is brought to bear, because I strongly believe that the institutional legacy is at stake.

The course of events over the last several years has left me quite despondent, and I strongly believe that it is important to take this step. It is necessary for us to resolve this conflict in order for us to move forward in a concerted manner to implement an innovative approach for the study and treatment of lung cancer. I remain hopeful that I will be able to do this within the Geffen School of Medicine at UCLA. However, the implementation of my comprehensive translational vision (from the bedside to the bench and back to bedside with experimental rational combination therapy in 4-5 years) will require ***broad based institutional and extramural support***. Thus, the sooner we are able to resolve and move beyond the conflict to develop this support, the better.

I have exhausted several avenues to air my grievances and seek effective mediation, over several years. In fact, I specifically requested the matter to be administratively considered and resolved long ***before*** Dr Dubinett came to be the recipient of the institutional CTSA, and the vice chancellor for “translational research”. I did not want those to be complicating variables in a fair process, as they are now.

That Dr Dubinett is the Vice Chancellor for Translational Research and the PI for the Institutional CTSA are facts. But I will ask the Senate to consider the process and due diligence with which he came to be offered the opportunity to serve as the PI for this accomplishment, especially after his leadership of the UCLA Lung Cancer Research Program (which *I* spent countless hours developing without actually being given credit for it) bankrupted and dissolved that program. Whereas it is clear that UCLA now is the beneficiary of the large CTSA that Dr Dubinett currently administers, I will ask the Senate to consider what opportunities and scientific discoveries have been stalled (to institutional detriment), and what careers have been obstructed and/or lost along the way, and how to systemically remedy those problems.

It is also important for the academic senate to recognize that my tortuous path to their doorstep has been long, with deliberate misdirection and stonewalling. The process has been very significantly complicated by a nebulous inter-institutional relationship between UCLA and its research affiliate (VAGLAHS). These institutions don’t share a synchronized research vision, and my attempts for fair mediation have been ineffective. After direct dialog with my mentor failed (over 6-7 years), I sought consultation with many senior

staff and administrative personnel at both the VA and UCLA. Although empathy was provided, there has been no redress or relief. In fact, over the last year, my efforts for effective mediation have led to severe personal intimidation and retaliation. Moreover, sitting still and waiting for the storm to pass is also not an option. The proximal event precipitating this grievance is that for the seventh straight cycle (nearly four years), my Merit Review application has returned unfunded, and Dr Dubinett's negative influence over the review process is strongly suspected.

In 1996, after 4-5 years training in gene delivery technologies, I was informed by Dr R Boucher (Director of the Cystic Fibrosis Center and Chief, Pulmonary and Critical Care Medicine; UNC Chapel Hill) that the development of a research program in gene therapy for lung cancer was not in that institution's plans, and that I should seek that opportunity elsewhere. Although I was **very highly trained** as a physician-scientist, I did not know anything about "academic politics" when recruited to come to UCLA. After meeting with Dr Dubinett, his calm and gentle demeanor, as well as a common sense of shared purpose also reassured me that there was little need for me to get embroiled in administrative affairs. He recruited me and told me that he would be my advisor and mentor, and together we would make inroads into a better understanding and treatment of lung cancer. I had prepared myself well for that task long before I got here, and I took him at his word.

However, it was only after gaining an external perspective that allowed me to recognize that his "mentorship" (1998-2007) was really a prototypical implementation of what is commonly known as the "revolving door policy". My case, in its simplest form, may represent a revolving door policy that went awry in its implementation. ***Based on the nature of my appointment and the course of events following my recruitment,*** I suspect that I was specifically recruited into the institution with a highly specialized talent and expertise in order to bolster *his* credibility and funding, but then apparently overstayed my welcome. Between 1999-2007, my former mentor largely usurped or misappropriated my innovative work and expertise, and as a necessary correlate, devalued my contributions to the development and building of the UCLA lung cancer research program. Then, after extracting the labor needed for *his own* (institutional and national) academic advancement, I was dispatched to wither away with very little resources, no mentorship, and in scientific isolation at VAGLAHS. By deliberately abdicating tutelage over the entire time I was an assistant professor, and by abandoning my academic promotion in national peer-review circles and presentations, *the former mentor's actions undermined my competence and credibility that linger to this day. I seek redress for this period*, during which my status was freely interchangeable between "trainee" versus "independent investigator", depending upon which designation most benefitted Dr Dubinett's interests at the time.

In fact, recent revelations by Dr Fogelman, the Chairman of the Department of Medicine (regarding the terms of my University "appointment"; see appendix), and by Dr Leroy Frey, Chief of the VA Research and Development Program Review (regarding the terms of VA "grants"; see appendix) seem to suggest that my entire UCLA career to date may really be the byproduct of an orchestrated scam from the outset. The terms and conditions of both the appointment and the assignment were ideally designed for a crafty administrator to take novel intellectual property (which was/ and is this "trainees" research identity) with impunity, without regard to due compensation or promotion. That the scam was "orchestrated" is suggested by evidence that the "official documents" about the agreed upon terms of my appointment (which were signed by high ranking administrative personnel at both institutions), actually had no "legal basis", and were subsequently "lost" by the Department of Medicine Administration at the VA. My "recruitment package" and my subsequent contract and grant funds were effectively under Dr Dubinett's direct/indirect control until I lost all funding in 2009. Because my salary was largely paid for by a "VA-Career Development Award", and "delta support" was initially provided through an RO1, both the VA and UCLA derived great benefit from exploiting an expertise that was not present in the environment, without having to leverage or gamble on anything (except for the leadership of the lung cancer research program).

As described, in 2007, after FDA approval for the immunogene therapy trial for lung cancer that *I* had largely developed was granted, I was forced out of the UCLA Lung Cancer Research Program. Subsequently, in my

exile and scientific isolation at GLAVAHHS, I developed a wholly new and highly innovative program to study lung cancer from a completely different perspective. With sparse resources, I developed a wholly independent scientific voice. But as I have found that voice, *the measures that are being brought to bear to curb my independence have become increasingly menacing and extreme, in order to cover up both earlier and more recent mistakes in both scientific judgment and personnel appraisal* (see detailed fact sheet and historical narrative).

Over the last two to three years, while going through the process to seek redress and relief, I have increasingly become aware that Dr Dubinett was (and is) able to easily take advantage of me and apply these menacing measures *because of the lack of an overarching research oversight or established memorandum of understanding between the primary academic institution (Geffen SoM at UCLA), and its VA-research affiliate (VAGLAHS)*, where I was assigned upon my recruitment. In retrospect, the process of recruitment and assignment to the VA was the first step in a deliberate and conspired effort to exploit a specific talent and expertise, in the absence of an appropriate or just inter-institutional administrative research oversight.

And as indicated, the pathway to independence is also blocked. After being propelled to national acclaim and spheres of high influence based largely on our “combined” work, my former mentor has re-directed that fame and influence against my independent advancement. The key tool to stifle my academic advancement has been a **sustained and relentless blockade of opportunity in every aspect of academic life** (funding through “independent peer-review”, personnel and resources, time, unfettered access to effective collaborations, and openly exercising academic freedom). In part, the block was enabled by an orchestrated handicap, imposed by a lack of promotion and mentorship during the time I was assigned to build the Translational Program (1999-2007). Despite my broad based knowledge base, the imposed lack of resources and mentoring in “grantsmanship” (an appreciation of the process of contemporary reviewer perspectives, framing of ideas, and pragmatics of managing grant funds) led to a series of ineffective funding submissions and tremendous frustration. Moreover, even when I was finally able to get my highly innovative ideas across to peer reviewers, Dr Dubinett’s administrative influence quenched those opportunities. These measures have clearly impacted not only my academic career and the pursuit of livelihood for my family; they have also contributed to an overtly hostile work environment for several years.

Whereas a legitimate, *evidence-based* concern about a contaminated peer-review process for my grants applications dates to as far back as 2007, the block to funding opportunity has been particularly intensive since a high-impact 2009 publication. Recently (since late 2009), for example, in the VA Merit Review Process, my last four applications have been triaged, although I can unabashedly declare that I know more about the molecular, cellular, and translational biology of the epithelial adhesion molecule I had proposed to study than nearly anyone else in the world. Whereas the reviews repeatedly cite that earlier scientific issues are addressed, reviewers simply come up with newer, more absurd tangential concerns. Reviewers feign a lack of understanding of intent or aims. Different reviewers direct changes in the application in different directions, suggesting that the process is orchestrated to befuddle a logical response. Importantly, this sort of orchestration is easily made possible *by the process* by which grant reviews at the VA are conducted. But the block to funding is not just within the VA system; ALL mechanisms to fund our research are blocked. *In the detailed historical narrative below, I cite specific reasons that lead me to believe that Dr Dubinett has negatively influenced the fairness of the peer-review process that is utilized for allocation of public funds for medical research.* These actions are also consistent with his longstanding antipathy (underneath an obsequious/benevolent façade), and more recent overt aggression towards my independent programmatic efforts. Because this block stems directly from Dr Dubinett’s influence, I feel that I am left with no choice but to air my grievances.

In addition, the ramifications of my initial nebulous appointment with UCLA have now spilled over into issues related to prospective and future intellectual property rights. As indicated, since 2006, I have developed a wholly new way to study the clinically relevant pathogenesis of lung cancer. I have clearly envisioned and

articulated how my approach will lead to effective control (?cure?) of this disease. The processes and approach have been developed in the absence of support (*and likely blockade of support*) by UCLA, but nevertheless, we are now at the threshold of garnering key molecular signatures (and novel therapeutic targets) for this disease. But because it is not clear who or what entity owns the property and downstream licensing rights to this development, to protect these rights, I have provisionally secured them in my own name.

Perhaps, it is the imprecise nature of the initial appointment that prompted the UCLA Administration to impose the funding block on my work (*it seems highly illogical that a program that has been in development since 2006, and was appreciated by the Director of the Cancer Center as being highly important by early 2009, has received no tangible support until Dr Fogelman's mediation in June of 2011*). Because there is apparently no legal basis for my joint appointment with UCLA, and the funding for the ongoing development of my invention has been deliberately blocked and/or stalled, the intellectual property rights surrounding my new and highly innovative program to study lung cancer have been unnecessarily jeopardized for over two years [*see communications dating back to 2009, and more recently with the Federal Office of General Counsel (OCG) and the VA-OIP*]. Irrespective of the underlying motivations, the downstream ramifications of my initial appointment seem to have taken on added complexity. Once the senate is able to direct a resolution towards redressing past concerns, and advise regarding the terms and conditions in which the prospective and future work is undertaken, I am hopeful that these novel programmatic efforts will now also have a clear course to follow.

What are the possible personal motivations (masquerading as “institutional concerns”) that stirred my former mentor to block my promotion or advancement? Afterall, he is now the UCLA-PI for a large CTSA-grant, and vice chancellor for the institution’s translational endeavors. He is thoroughly secure, and infinitely busy. My nascent program should not be a concern.

There are many possible motivations; only Dr Dubinett knows the real reason(s). However, recent events allow me to make some inferences, based on several years of working closely with him. The practical need to suppress my academic advancement stems from my recent recognition that although he has personally compared (in the summer of 2010, during our last face-to-face meeting) my innovative quest to the study and treatment of lung cancer to an earlier (infamous) journey by Dr Judah Folkman (the “Father of anti-angiogenic therapy for Cancer”, and a personal hero of mine), **he has publicly disavowed my work**. While Dr Dubinett has been impervious to my repeated solicitations to tangibly support our innovative approach for over 5 years, **he has publicly promoted and materially supported an alternative but fundamentally flawed experimental paradigm of another investigator at Boston University**. That the alternative approach he has promoted is fundamentally flawed is not only my longstanding view, but it is now also an emerging general perspective amongst those who think about lung cancer a lot.

Why would Dr Dubinett publicly advocate for massively funding (through a \$15 million DoD proposal in 2010) a faulty approach, while ignoring and then blocking a UCLA-developed paradigm-shifting strategy that he has seen emerging over the last five years? This remains a puzzling conundrum, and is especially confusing because whereas the world recognizes UCLA as being a major hub for Stem Cell Biology, the administration has collectively ignored and/or suppressed world leading work for lung cancer in this arena (see detailed preface for the factual basis of this statement). Why would he allow that to happen to UCLA?

There are several possible explanations. Perhaps, there is a desire to bring the PI (Dr A Spira, Boston University) of the DoD grant that he supported to be his replacement as the Chief of Pulmonary and Critical Care Medicine. Certainly, this would make sense. Dr Spira, by virtue of a supportive long standing mentorship by Dr JS Brody, and more recently, a friend and mentor in Dr S Dubinett, has compiled an impressive CV with many publications and high funding record. It would certainly garner substantive matching support from the institution if he was to be successfully recruited to UCLA, and the CTSA that Dr Dubinett heads also has FTE that need to be filled. Without doubt, my funding record *on paper* is comparatively weak (but indexed to the

funding that actually came under *my name*, I will match my research accomplishments to those of any other investigator or physician-scientist). Perhaps, by continuing a block to my funding, he simply highlights the contrast (*on paper*) between a chosen successor versus a local innovator even more, and further limits local competition for critical resources.

And it is exactly this aspect of Academia (dependence on a “fair and equitable review, promotion and resource-allocation process”) that concerns me the most, especially in the current times of sparse resources. *It is not the threat of competing with an alternative idea that concerns me* (I actually relish and welcome that challenge); *it is this uneven administrative orchestration of a stacked deck (against my approach and idea) to acquire needed resources that brings about significant concern.*

If my suspicions are correct, then Dr Dubinett’s conflicts of interest have not only put the brakes on my work and career, but they may hurt the institution (and the medical science of our academic field) in the long run. If Dr Spira is indeed a preordained legacy candidate to replace Dr Dubinett as the Chief of Pulmonary Medicine at UCLA, then the mentorship provided to help him procure a large DoD grant is largely a waste, and any money spent in his recruitment is likely towards a scientific dead end. ***Simply put, my approach is not only more innovative and unique, it is better.***

In conclusion, the differences between Dr Dubinett and me are plain and obvious. I contend that he picked up the needed scientific rhetoric and materials for the translational endeavors he called his own from me, and was coached by other administrators about how to exploit those for his own (and institutional) benefit. The problem is that he did not really comprehend how to best apply the technology for which he proclaimed himself as the expert. Without question, he is a crafty administrator and a maven of process, who knows which buttons to push to squeeze money out of systems. The problem is that those buttons are pushed without regard to ethics or the advancement of a noble agenda or who gets hurt along the way. I view him as an incarnation of Salieri; unfortunately, he has tried to kill this golden goose after stealing a single egg.

On the other hand, until recently, I was driven solely by the desire to make a big difference in the disease process I had long studied to amass a substantive knowledge base. Until recently, I had not considered the business aspects of medical science. I believed the important ideas I was promoting were sufficient, but I was kept ignorant (and remain quite ignorant) *of the process of garnering the resources* needed to make that big impact. From a personal perspective, it appears that the institutional tutelage “in process and pragmatics” was afforded to a few pre-selected personnel. Not having been taught the pragmatics, and having been deprived of the needed professional influence, I remain at a disadvantage when it comes to the political rules of the game.

But, despite that, I have developed a *virtual program* which will wholly redefine lung cancer. Moreover, I have figured out why my earlier attempts at viral gene therapy for lung cancer failed (and how to overcome that failure). Further, with my wife (Dr Anshu Batra, a developmental pediatrician), we have already begun pilot implementation of a novel approach for the rehabilitation of children on the Autism spectrum. So the current question is whether these are programs and projects will be allowed to develop under the University’s umbrella.

Where do we go from here?

Whereas I discuss the timeline of this grievance, and issues related to redress of earlier grievances herein, ***the key issues moving forward are several-fold.***

First, despite a recent very kind gesture by Dr Fogelman, it remains unclear to me whether UCLA is large enough to promote two fundamentally different and competing visions when it comes to the study and treatment of lung cancer. I think it is possible, but if so, would it be fair for me to ask for the sort of mentorship, institutional promotion, and tangible access to resources that Dr Dubinett has provided to Dr Spira (without his

being here)? Afterall, I have already suggested that our emerging approach is not only more innovative, it is better, and that the differences between Dr Spira and I on paper (CV) are largely contrived.

Second, it is unclear to me whether UCLA is large enough to accommodate the two very different personalities that embody the differences between Dr Dubinett and me. If it is possible, then let's move onward with open cooperation and collaboration. But if that is not possible, then I ask this committee to simply consider my grievances for issues related to redress, and to exercise its influence in removing the current opportunity-block on our important work. By helping me redeem the ground that I have lost, by opening access to state and national funding, and by allowing unfettered access to collaborative resources without negative influence, I am confident that our important work will find another worthy and supportive home.