STAR METRICS

A Prototype Application to Describe Innoation: The Remicade/Enbrel Trace Study

Measuring the Impacts of Federal Investments in Research

April 19, 2011

Stefano Bertuzzi, Ph.D.
Office of Science Policy Analysis
Office of the Director
National Institutes of Health

The Public is Watching

… judging by the only criterion that matters to patients and taxpayers—not how many interesting discoveries have been made, but how many treatments for diseases the money has bought—the return on investment to the American taxpayer has been approximately as satisfying as the AIG bailout.

NATURE|Vol 465|10 June 2010

What science is really worth

Spending on science is one of the best ways to generate jobs and economic growth, say research advocates. But as **Colin Macilwain** reports, the evidence behind such claims is patchy.

An Interesting Approach

The NEW ENGLAND JOURNAL of MEDICINE

SPECIAL ARTICLE

The Role of Public-Sector Research in the Discovery of Drugs and Vaccines

Ashley J. Stevens, D.Phil., Jonathan J. Jensen, M.B.A., Katrine Wyller, M.B.E., Patrick C. Kilgore, B.S., Sabarni Chatterjee, M.B.A., Ph.D., and Mark L. Rohrbaugh, Ph.D., J.D.

ABSTRACT

BACKGROUND

Historically, public-sector researchers have performed the upstream, basic research that elucidated the underlying mechanisms of disease and identified promising points of intervention, whereas corporate researchers have performed the downstream, applied research resulting in the discovery of drugs for the treatment of diseases and have carried out development activities to bring them to market. However, the boundaries between the roles of the public and private sectors have shifted substantially since the dawn of the biotechnology era, and the public sector now has a much more direct role in the applied-research phase of drug discovery.

METHODS

We identified new drugs and vaccines approved by the Food and Drug Administration (FDA) that were discovered by public-sector research institutions (PSRIs) and classified them according to their therapeutic category and potential therapeutic effect.

RESULTS

We found that during the past 40 years, 153 new FDA-approved drugs, vaccines, or new indications for existing drugs were discovered through research carried out in PSRIs. These drugs included 93 small-molecule drugs, 36 biologic agents, 15 vaccines, 8 in vivo diagnostic materials, and 1 over-the-counter drug. More than half of these drugs have been used in the treatment or prevention of cancer or infectious diseases. PSRI-discovered drugs are expected to have a disproportionately large therapeutic effect.

CONCLUSIONS

Public-sector research has had a more immediate effect on improving public health than was previously realized.

From the Institute for Technology Entrepreneurship and Commercialization (A.J.S.) and Office of Technology Development (A.J.S., J.J.J.), Boston University School of Management, Boston; the Norwegian Radium Hospital Research Foundation, Oslo (K.W.); Boston University School of Law, Boston (P.C.K.); and the Office of Technology Transfer, National Institutes of Health, Bethesda, MD (S.C., M.L.R.). Address reprint requests to Dr. Stevens at Boston University School of Management, 53 Bay State Rd., Boston, MA 02215, or at astevens@bu.edu.

N Engl J Med 2011;364:535-41.

STAR METRICS: Building an Empirical Framework

Start with scientists as the unit of analysis

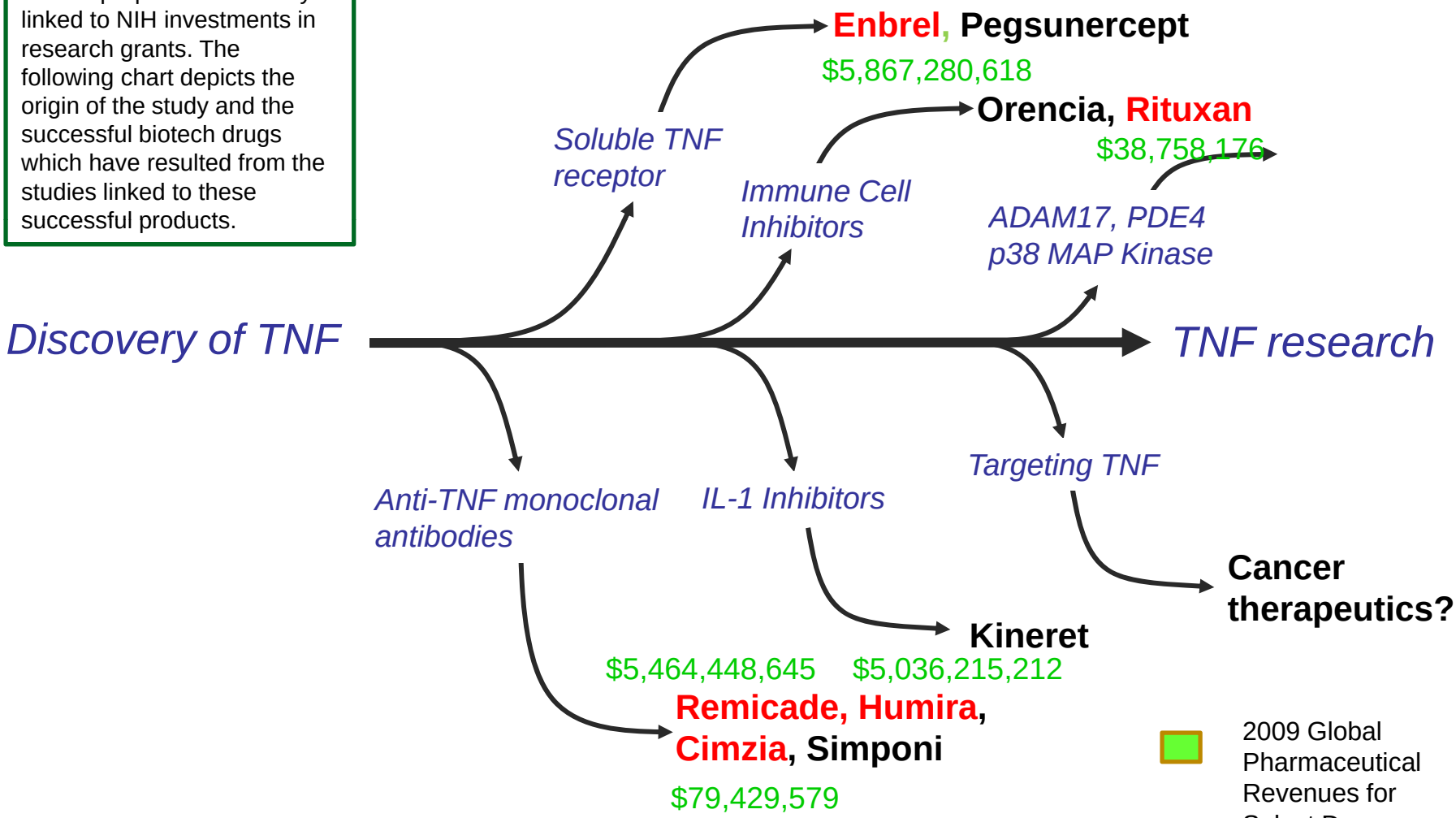
Include full description of input measures

Include full description of outputs and outcomes (economic, scientific and social)

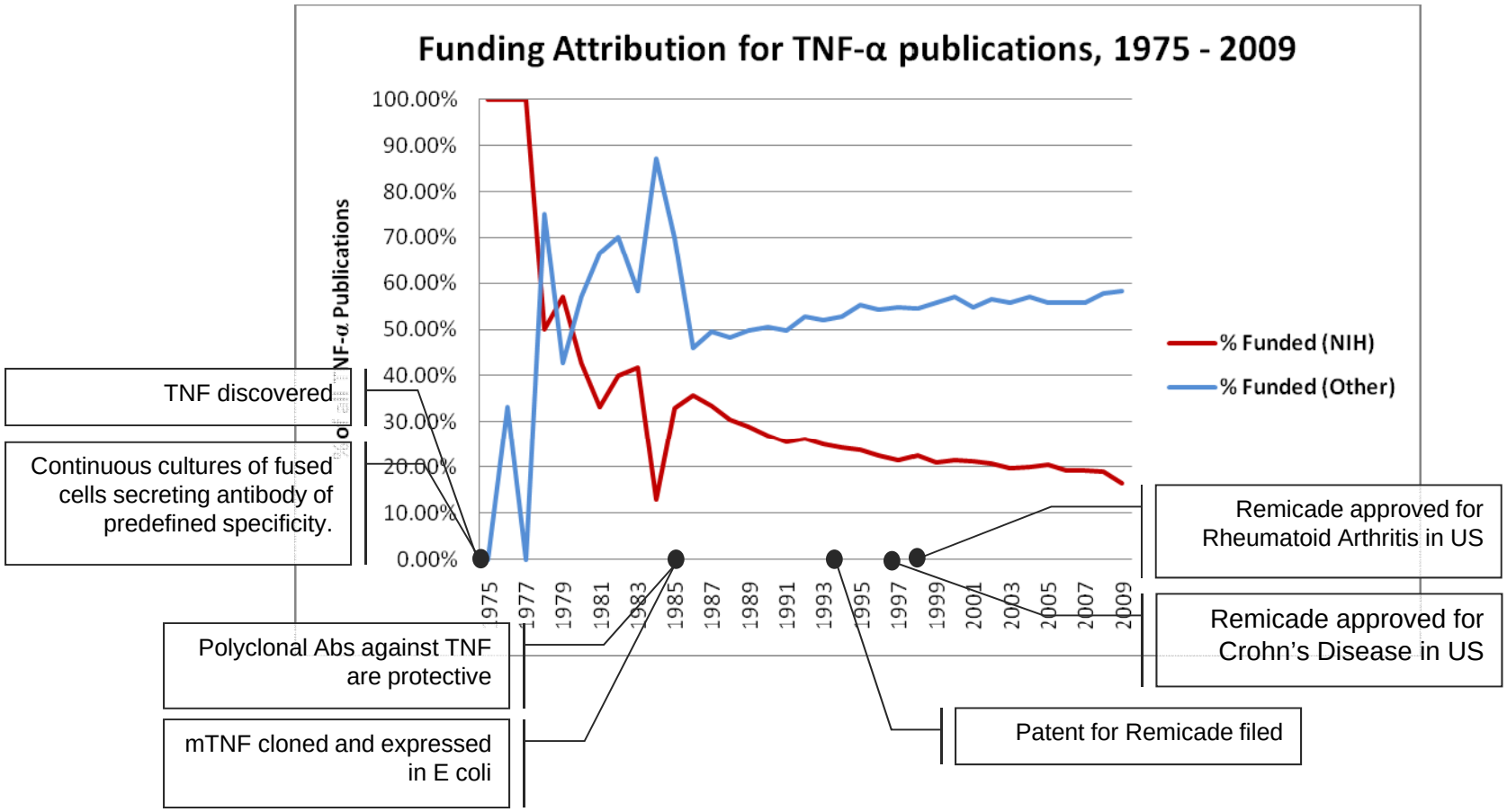
Link inputs, outputs and outcomes

Evolution of Disease Modifying Anti-Rheumatic Drugs (DMARD) TNF-a drugs

The discovery of TNF and its related properties is directly linked to NIH investments in research grants. The following chart depicts the origin of the study and the successful biotech drugs which have resulted from the studies linked to these successful products.



Funding trends in TNF-α publications



Events leading to the approval of Enbrel

1975 Discovery of TNFa

1975 Hybridoma technology for mAbs

1985 Passive immunization with polys against TNFa are protective against endotoxin

1984 Chimeric mAbs

1993 Characterization of a human-mouse TNFa chimeric mAb

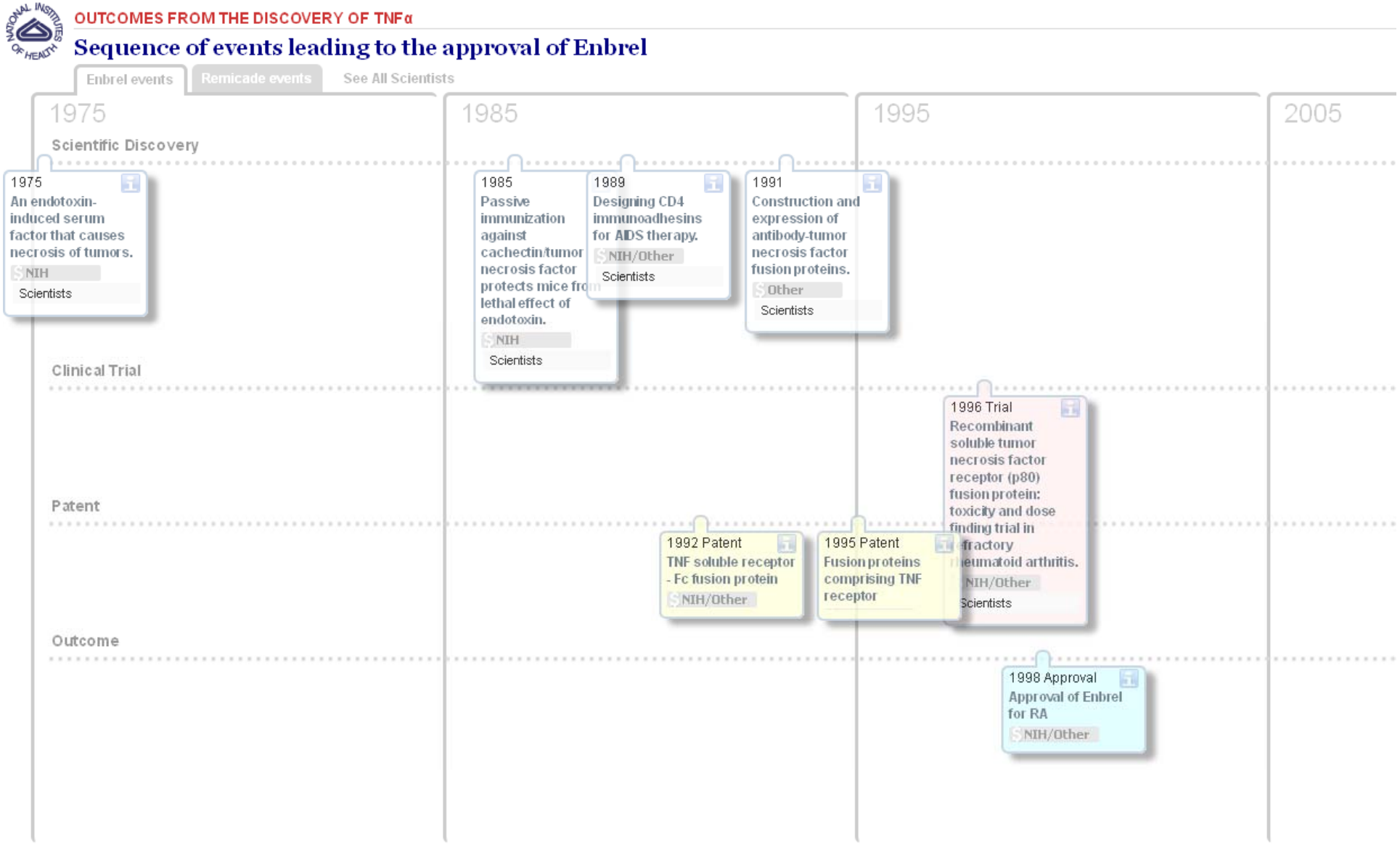
1997 Methods for treating Crohn's Disease using chimeric mAbs (Patent)

1997 Methods for treating RA using chimeric mAbs (Patent)

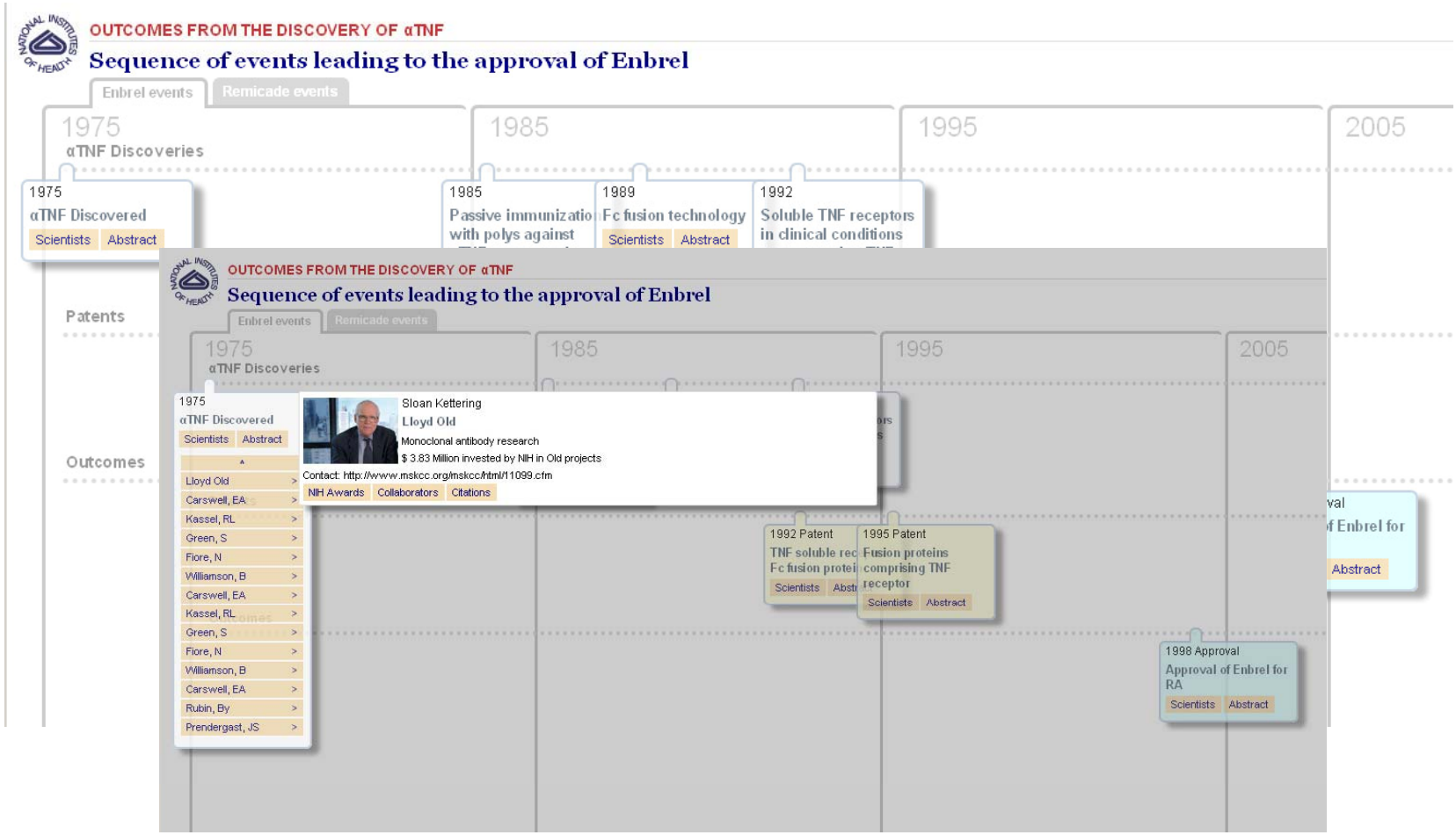
1998 Approval of Remicade for Crohn's Disease

NIH Funded Studies data overlay –
pubs
grants
citations
Notable PI's

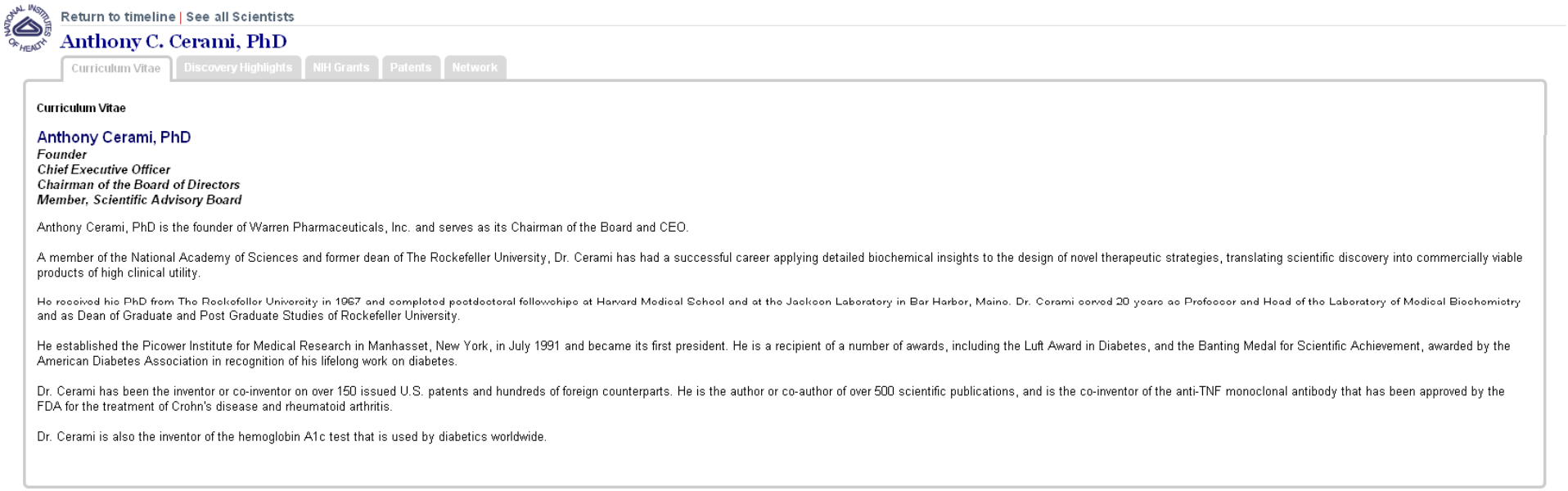
Milestone Events Interactive Timeline



The Network of Scientists that did it



Milestone Events Interactive Timeline



Milestone Events Interactive Timeline

Return to timeline | See all Scientists

Anthony C. Cerami, PhD

Curriculum Vitae | Discovery Highlights | NIH Grants | Patents | Network

Anthony Cerami: Career Discovery Highlights

Anthony Cerami has led research programs into genetic, metabolic and infectious diseases, with the goal of translating scientific discovery into clinically important products.

Among the successes of his approach are: glycated hemoglobin to monitor diabetic control , anti-tumor necrosis factor-alpha therapies to treat inflammatory diseases and, recently, tissue protective cytokines to safely treat devastating diseases and injuries.

He is the recipient the Luft Award in Diabetes and the Frederick Banting Medal for Scientific Achievement, awarded by the American Diabetes Association in recognition of his lifelong work on diabetes.

NIH INPUTS

Return to timeline | See all Scientists

Anthony C. Cerami, PhD

Curriculum Vitae | Discovery Highlights | NIH Grants | Patents | Network

Grant Number	Fiscal Year	Project Title	Grant_Funding
R01AI021359-14	1998	PARASITE INDUCED CATABOLISM	$309,173
R01AI021359-13	1997	PARASITE INDUCED CATABOLISM	$297,281
R01AI021359-12	1996	PARASITE INDUCED CATABOLISM	$323,947
R01DK019655-19	1996	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$289,696
R01AI021359-11	1995	PARASITE INDUCED CATABOLISM	$311,486
R01AI030660-06	1995	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$205,697
R01DK019655-18	1995	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$279,691
R37AI019428-14	1995	BIOCHEMISTRY OF TRYPANOSOMATIDS	$310,953
R01AI021359-10A2	1994	PARASITE INDUCED CATABOLISM	$271,995
R01AI030660-05	1994	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$216,722
R01DK019655-17	1994	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$270,069
R37AI019428-13	1994	BIOCHEMISTRY OF TRYPANOSOMATIDS	$288,461
S15AI037041-01	1994	SMALL INSTRUMENTATION GRANT	$16,473
R01AI030660-04	1993	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$250,322
R01DK019655-16	1993	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$237,705
R37AI019428-12	1993	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$272,257
S15AI035077-01	1993	SMALL INSTRUMENTATION GRANT	$14,528
R01AI021359-09	1992	PARASITE INDUCED CATABOLISM IN MAMMALS	$309,750
R01AI030660-03	1992	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$173,174
R01AI030660-03S1	1992	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$52,893
R01DK019655-15A1	1992	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$239,392
R37AI019428-11	1992	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$261,786
R01AI021359-07	1991	PARASITE INDUCED CATABOLISM IN MAMMALS	$45,081
R01AI021359-08	1991	PARASITE INDUCED CATABOLISM IN MAMMALS	$225,399
R01AI030660-01	1991	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$17,744
R01AI030660-02	1991	HEMOGLOBIN CATABOLISM BY PLASMODIUM FALCIPARUM	$157,371
R37AI019428-09	1991	BIOCHEMISTRY OF TRYPANOSOMATIDS - BASIS FOR DRUG DESIGN	$167,014
R37AI019428-10	1991	BIOCHEMISTRY OF TRYPANOSOMATIDS-BASIS FOR DRUG DESIGN	$83,884
R01AI021359-06	1990	PARASITE INDUCED CATABOLISM IN MAMMALS	$280,835
R01DK019655-14	1990	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$231,905
R37AI019428-08	1990	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$199,574
R01AI021359-05	1989	PARASITE INDUCED CATABOLISM IN MAMMALS	$281,770
R01DK019655-13	1989	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$222,946
R37AI019428-07	1989	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$192,210
R01AI021359-04	1988	PARASITE INDUCED CATABOLISM IN MAMMALS	$276,802
R01DK019655-12	1988	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$209,186
R37AI019428-06	1988	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$187,078
R01AI021359-02S1	1987	PARASITE-INDUCED CATABOLISM IN MAMMALS	$67,181
R01AI021359-03	1987	PARASITE INDUCED CATABOLISM IN MAMMALS	$120,132
R01DK019655-11	1987	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$205,596
R37AI019428-05	1987	BIOCHEMISTRY OF TRYPANOSOMATIDS - BASIS FOR DRUG DESIGN	$168,715
R01AI021359-02	1986	PARASITE INDUCED CATABOLISM IN MAMMALS	$99,348
R01DK019655-10	1986	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$220,687
R37AI019428-04	1986	BIOCHEMISTRY OF TRYPANOSOMATIDS - BASIS FOR DRUG DESIGN	$160,992
R01AI019428-03	1985	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$186,221
R01AI021359-01A1	1985	PARASITE INDUCED CATABOLISM IN MAMMALS	$102,560
R01AM019655-09	1985	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$121,530
R01AI019428-02	1984	BIOCHEMISTRY OF TRYPANOSOMATIDS--BASIS FOR DRUG DESIGN	$169,318
R01AM019655-07S1	1984	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$3,731
R01AM019655-08	1984	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	$125,461

Project Information

BACK TO QUERY FORM PRINT VERSION

DESCRIPTION | DETAILS | RESULTS | HISTORY | SUBPROJECTS

Project Number:	5R01DK019655-17	**Contact Principal Investigator:**	CERAMI, ANTHONY
Title:	BIOCHEMICAL BASIS OF THE COMPLICATIONS OF DIABETES	**Awardee Organization:**	PICOWER INSTITUTE FOR MEDICAL RESEARCH

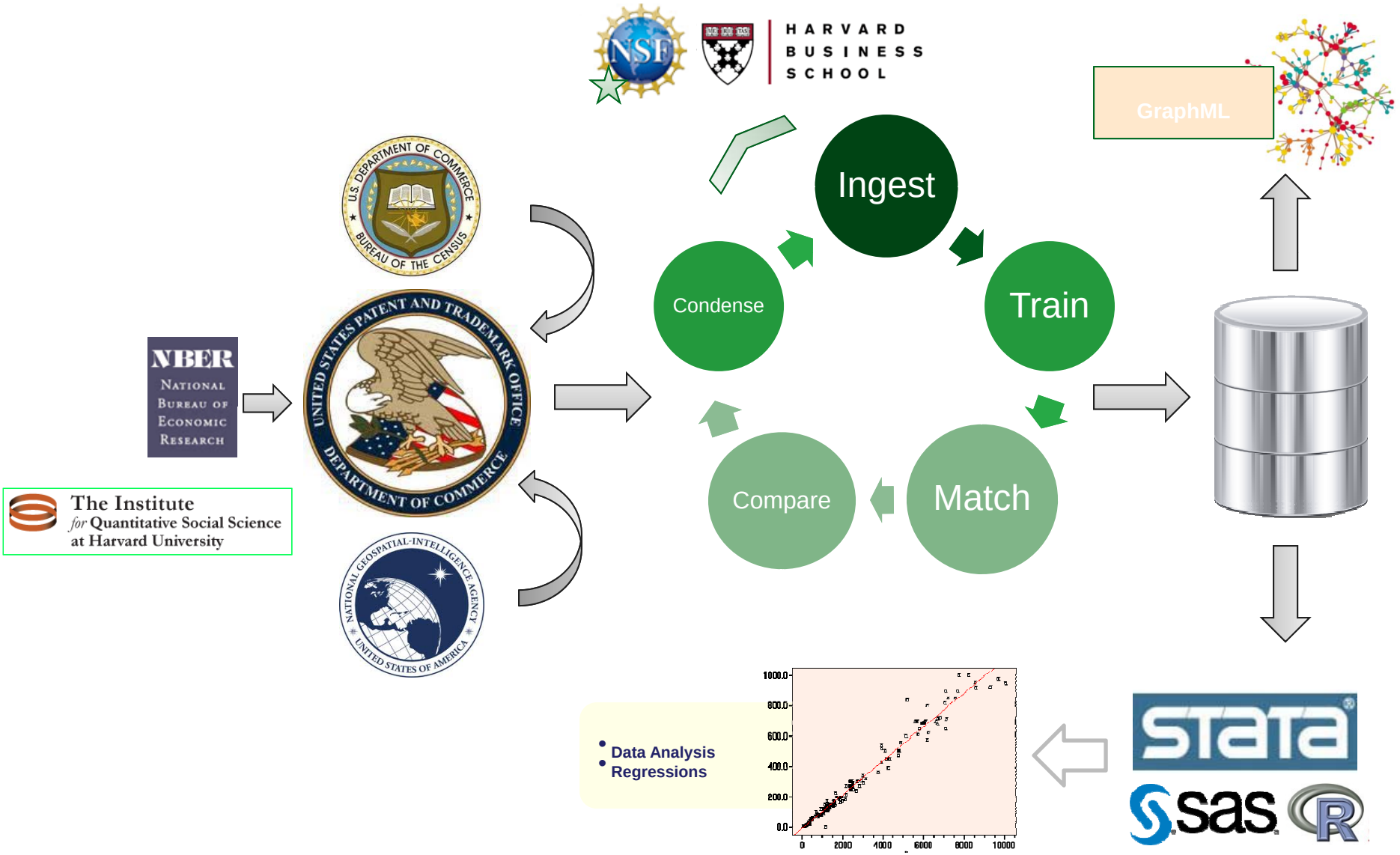
Abstract Text:

Diabetics slowly fall prey to a variety of serious, even lethal health complications which are apparently unrelated to insulin levels but have been clearly associated with hyperglycemia. We have studied the chemical molecular and cellular mechanisms which underly these diabetic sequelae for the past twenty years, and developed the central hypothesis that diabetic complications are caused in part by the covalent addition of reducing sugars, particularly glucose, to proteins by non-enzymatic reactions. Non- enzymatic glycation begins with the Amadori rearrangement of sugar-protein condensation products, and proceeds through a complex series of chemical rearrangements to generate a wide variety of Advanced Glycosylation Endproducts (AGEs) which are, as a class, permanent, fluorescent, cross- linking adducts that accumulate on cells, soluble proteins and tissue components exposed to glucose either in vitro or in vivo. Despite our prior success in identifying several AGE-related entities, the chemistry and structure of AGE adducts are known at only a rudimentary level. We now propose to extend certain novel approaches we have applied to this structural work, emphasizing the use of specially synthesized starting materials and AGE-trapping reagents to further elucidate the chemistry of AGEs. We believe, and are now demonstrating, that these chemical insights will lead to therapeutic innovations against diabetic complications. We also propose new experimental approaches to better understand the mechanism of action of one such potential therapeutic agent, aminoguanidine, discovered under the support of our last grant. Another long-standing priority has been to develop quantitative assay systems for AGEs on plasma or tissue components. We propose to refine our current assay technologies for AGEs in biological samples, primarily by developing an AGE receptor-based solid-phase competitive assay capitalizing on our recent success with a competitive whole-cell assay based on the same receptor.

Project Terms:

adduct; aminoguanidine; antibody formation; carbohydrate receptor; chemical addition; chemical synthesis; crosslink; diabetes mellitus; diabetes mellitus therapy; diabetic nephropathy; enzyme linked immunosorbent assay; glucose metabolism; glycosylation; human subject; human tissue; hyperglycemia; laboratory mouse; laboratory rabbit; laboratory rat; macrophage; medical complication; molecular pathology; nuclear magnetic resonance spectroscopy; radioimmunoassay

Capturing Outputs and Outcomes: Harvard/NBER Disambiguated Patent Database



OUTPUTS (all patents)

Return to timeline | See all Scientists

Anthony C. Cerami, PhD

Curriculum Vitae | Discovery Highlights | NIH Grants | Patents | Network

Filter:

Patent Number	App Year	Grant Year	Assignee	Technology Class	Patent Abstract	Patent Title
7645733	2004	2010	WARREN PHARMACEUTICALS INC	514/530		
7022721	2004	2006	None	548/514	In one embodiment, the present invention relates to compounds and compositions including pharmaceutical compositions containing the compounds and associated methods that uncouple sugar-mediated coupling of proteins, lipids, nucleic acids, and other biomaterials, and any combination thereof. In another embodiment, the compositions and associated methods have utility in vivo to reduce the deleterious effects of sugar-mediated coupling processes in an organism, when the organism is exposed to the compound or composition internally, by ingestion, transdermal application, or other means. In yet another embodiment, the compositions and associated methods are useful for the ex-vivo treatment of organs, cells and tissues and external treatment of hair, nails and skin to rejuvenate them by changing deformability and increase the tissue diffusion coefficient. In a further embodiment, the present invention relates to novel compounds and pharmaceutical compositions.	Method and composition for rejuvenating cells, tissues, organs, hair and nails
7718363	2003	2010	THE KENNETH S WARREN INSTITUTE INC	435		
6906076	2003	2005	CYTOKINE PHARMASCIENCES INC	514/436	The present invention concerns alkyl aryl carbonyl compounds that possess anti-infective activity. The compounds of the invention can be used to target specific nuclear localization signal, thereby blocking importation of specific proteins or molecular complex into the nucleus of a cell. The invention encompasses methods of use of such compounds for treatment or prevention of infectious diseases, such as parasitic and viral diseases, including, for example, malaria and acquired immunodeficiency syndrome. The use of the compounds to detect certain specific protein structures which are present in nuclear localization sequences is also taught.	Compounds and methods of use to treat infectious diseases
7517523	2003	2009	CYTOKINE PHARMASCIENCES INC	424	The present invention relates to compositions and methods for inhibiting the release and/or biological activity of migration inhibitory factor (MIF). In particular, the invention relates to the uses of such compositions and methods for the treatment of various conditions involving cytokine-mediated toxicity, which include, but are not limited to shock, inflammation, graft versus host disease, and/or autoimmune diseases.	Anti-MIF antibodies
7022719	2003	2006	ALTEON INC	514/548	The present invention relates to compositions and methods for inhibiting and reversing nonenzymatic cross-linking (protein aging). Accordingly, compositions are disclosed which comprise an agent capable of inhibiting the formation of advanced glycosylation endproducts of target proteins, and which additionally reverse pre-formed crosslinks in the advanced glycosylation endproducts by cleaving alpha-dicarbonyl-based protein crosslinks present in the advanced glycosylation endproducts. Certain useful agents are thiazolium salts. The method comprises contacting the target protein with the composition. Both industrial and therapeutic applications for the invention are envisioned, as food spoilage and animal protein aging can be treated. A novel immunoassay for detection of the reversal of the nonenzymatic crosslinking is also disclosed.	Preventing and reversing the formation of advance glycosylation endproducts
7090861	2003	2006	FARRINGTON PHARMACEUTICALS LLC	424	The present invention relates to devices that allow for linear, sustained-release of solutes with adjustable initial-release kinetics. In particular, the present invention relates to devices for delivering substances to the body of an animal. The present invention also relates to methods for delivering solutes in a constant, sustained-release fashion using the devices of the invention.	Sustained release delivery systems for solutes
7384625	2003	2008	FARINGTON PHARMACEUTICALS LLC	424/514/548	A method and composition for the treatment of hair, nail, ex-vivo organ, ex-vivo cell or ex-vivo tissue to improve the biomechanical and diffusional characteristics comprising an effective amount of a compound selected from the group consisting of compounds of the formula (I):	Method and composition for rejuvenating hair, nails, tissues, cells and organs by ex-vivo or immersive treatment
6790859	2003	2004	ALTEON INCORPORATED	514		Reversing advanced glycosylation cross-links using heterocyclic-substituted thiazolium compounds
6689777	2002	2004	KENNETH S WARREN INSTITUTE	514/544		Anti-malarial compounds, compositions and methods
6777557	2002	2004	None	514/548		Method and composition for rejuvenating cells, tissues organs, hair and nails
6713050	2002	2004	FARRINGTON PHARMACEUTICALS INC	424/514		Method and composition for rejuvenating hairs, nails, tissues, cells and organs by ex-vivo or immersive treatment
6649797	2001	2003	THE PICOWER INSTITUTE FOR MEDICAL RESEARCH	564/436		Compounds and methods of use to treat infectious diseases
6569152	2001	2003	FARRINGTON PHARMACEUTICALS LLC	604/424		Sustained release delivery systems for solutes
6531121	2000	2003	THE KENNETH S WARREN INSTITUTE INC	424/435/514		Protection and enhancement of erythropoietin-responsive cells, tissues and organs
7309687	2000	2007	THE KENNETH S WARREN INSTITUTE INC	514	Methods and compositions are provided for protecting or enhancing excitable tissue function in mammals by	Methods for treatment and prevention of

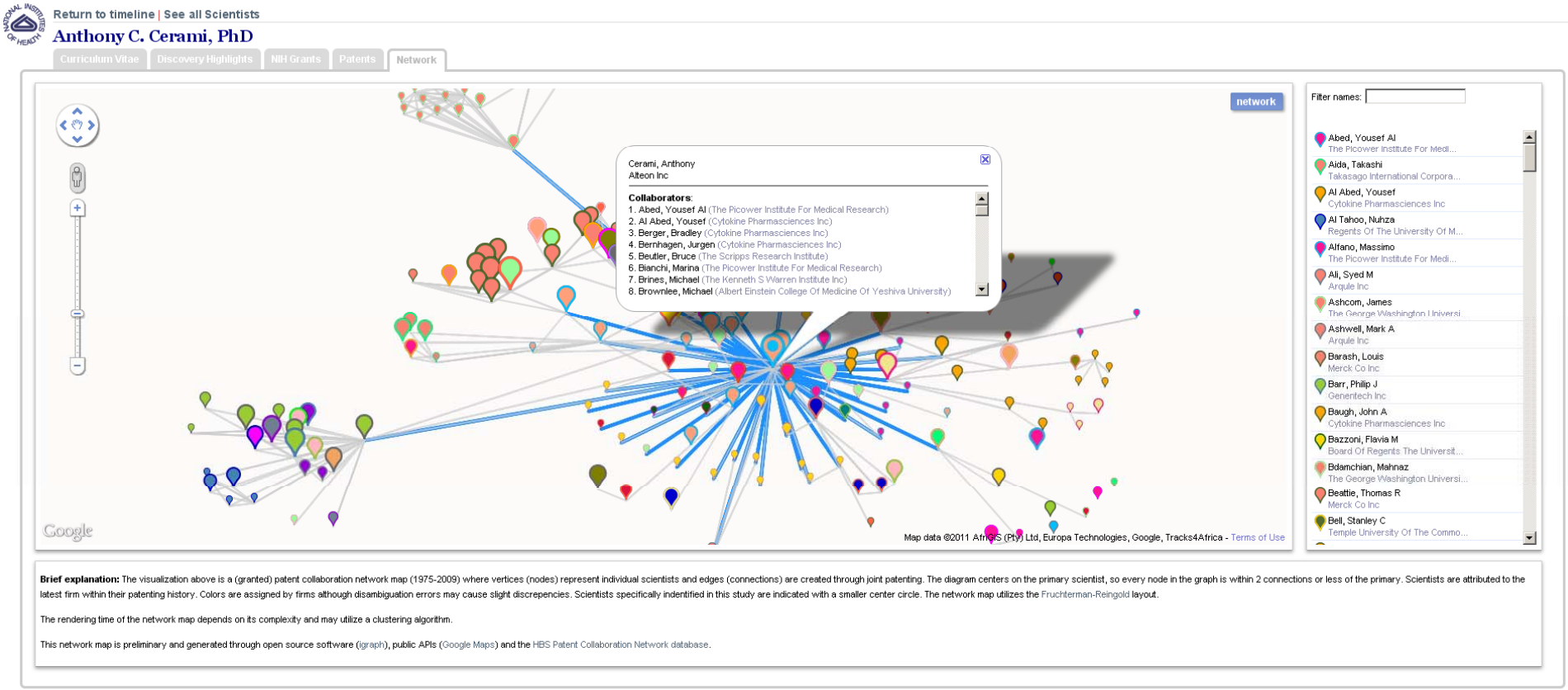
Filtered patents for TNF-a

Anthony C. Cerami, PhD

Filter: TNF

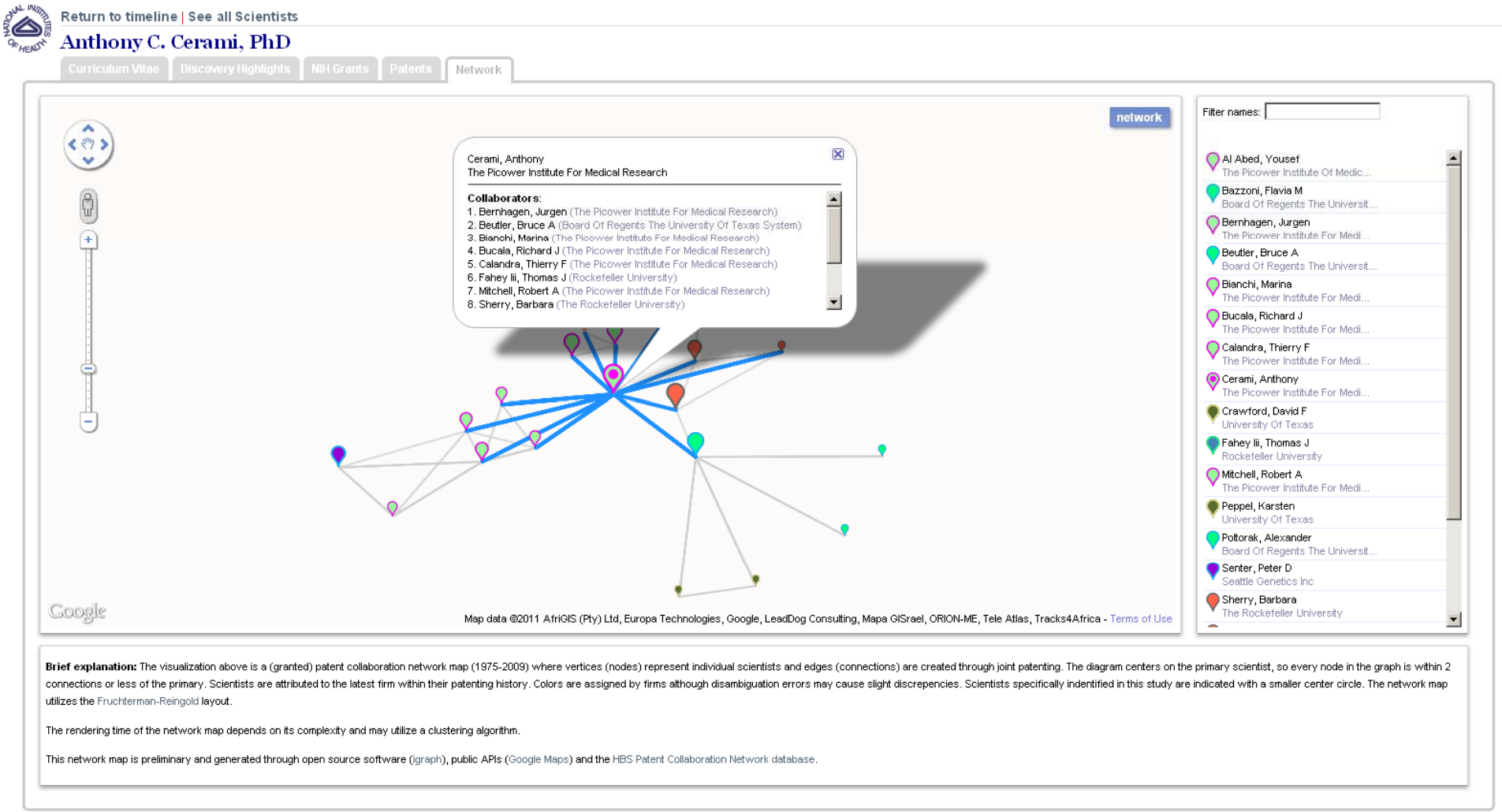
Patent Number	App Year	Grant Year	Assignee	Technology Class	Patent Abstract	Patent Title
5717074	1995	1998	THE ROCKEFELLER UNIVERSITY	530/435	An antibody to an inflammatory cytokine is disclosed. The inflammatory cytokine has been isolated from cells that have been incubated with a stimulator material. The inflammatory cytokine is capable of binding to heparin, inducing localized inflammation characterized by polymorphonuclear cell infiltration when administered subcutaneously and having potent in vitro chemotactic activity while inducing little or no in vitro chemokinesis in polymorphonuclear cells, while lacking the ability to suppress the activity of the anabolic enzyme lipoprotein lipase, cause the cytotoxicity of cachectin/TNF-sensitive cells, stimulate the blastogenesis of endotoxin-resistant C3H/HeJ thymocytes, or induce the production of cachectin/TNF by primary thioglycbllate-elicited mouse macrophage cells. A particular inflammatory cytokine has been isolated and its cDNA has been sequenced. The sequence predicts a cDNA of 74 amino acids in length and a molecular weight of 7,908. Diagnostic and therapeutic utilities are proposed, and testing procedures, materials in kit form, recombinant materials and procedures, and pharmaceutical compositions comprising an antibody to the cytokine are likewise set forth.	Macrophage-derived inflammatory mediator (MIP-2)
5741484	1995	1998	THE ROCKEFELLER UNIVERSITY	424/514	An inflammatory cytokine is disclosed which has been isolated from cells that have been incubated with a stimulator material. The inflammatory cytokine comprises a protein that is capable of binding to heparin, inducing localized inflammation characterized by polymorphonuclear cell infiltration when administered subcutaneously and inducing in vitro polymorphonuclear cell chemokinesis, while lacking the ability to suppress the activity of the anabolic enzyme lipoprotein lipase, cause the cytotoxicity of cachectin/TNF-sensitive cells, stimulate the blastogenesis of endotoxin-resistant C3H/HeJ thymocytes, or induce the production of cachectin/TNF by primary thioglycollate-elicited mouse macrophage cells. A particular inflammatory cytokine MIP-1 has been isolated and has been found to comprise a peptide doublet of similar molecular weights of about 8,000 daltons, and to show a pI of about 4.6. The doublet has been resolved into its component peptides, MIP-1.alpha. and MIP-1.beta. for which distinct cDNA's have been cloned and sequenced. Diagnostic and therapeutic utilities are proposed, and testing procedures, materials in kit form and pharmaceutical compositions are likewise set forth.	Macrophage-derived inflammatory mediator (MIP-1.alpha. and MIP-1.beta.)
5760186	1995	1998	THE ROCKEFELLER UNIVERSITY	530	Antibodies to an inflammatory cytokine are disclosed. The inflammatory cytokine has been isolated from cells that have been incubated with a stimulator material and comprises a protein that is capable of binding to heparin, inducing localized inflammation characterized by polymorphonuclear cell infiltration when administered subcutaneously and inducing in vitro polymorphonuclear cell chemokinesis, while lacking the ability to suppress the activity of the anabolic enzyme lipoprotein lipase, cause the cytotoxicity of cachectin/TNF-sensitive cells, stimulate the blastogenesis of endotoxin-resistant C3H/HeJ thymocytes, or induce the production of cachectin/TNF by primary thioglycollate-elicited mouse macrophage cells. A particular inflammatory cytokine MIP-1 has been isolated and has been found to comprise a peptide doublet of similar molecular weights of about 8,000 daltons, and to show a pI of about 4.6. The doublet has been resolved into its component peptides, MIP-1.alpha. and MIP-1.beta. for which distinct cDNA's have been cloned and sequenced. Diagnostic and therapeutic utilities are proposed, and testing procedures, materials in kit form and pharmaceutical compositions are likewise set forth.	Antibody to macrophage-derived inflammatory mediator (MIP-1.alpha. and
5817763	1995	1998	THE ROCKEFELLER UNIVERSITY	530/424	An inflammatory cytokine is disclosed which has been isolated from cells that have been incubated with a stimulator material. The inflammatory cytokine comprises a protein that is capable of binding to heparin, inducing localized inflammation characterized by polymorphonuclear cell infiltration when administered subcutaneously and inducing in vitro polymorphonuclear cell chemokinesis, while lacking the ability to suppress the activity of the anabolic enzyme lipoprotein lipase, cause the cytotoxicity of cachectin/TNF-sensitive cells, stimulate the blastogenesis of endotoxin-resistant C3H/HeJ thymocytes, or induce the production of cachectin/TNF by primary thioglycollate-elicited mouse macrophage cells. A particular inflammatory cytokine MIP-1 has been isolated and has been found to comprise a peptide doublet of similar molecular weights of about 8,000 daltons, and to show a pI of about 4.6. The doublet has been resolved into its component peptides, MIP-1.alpha. and MIP-1.beta. for which distinct cDNA's have been cloned and sequenced. Diagnostic and therapeutic utilities are proposed, and testing procedures, materials in kit form and pharmaceutical compositions are likewise set forth.	Macrophage-derived inflammatory mediator (MIP-1.alpha. and MIP-1.beta.)
5849873	1995	1998	THE ROCKEFELLER UNIVERSITY	530/424	An inflammatory cytokine is disclosed which has been isolated from cells that have been	Macrophage-derived inflammatory

Collaboration & Innovation Network Maps
The ripple effect of NIH funding



Collaboration & Innovation Network Maps
The ripple effect of NIH funding

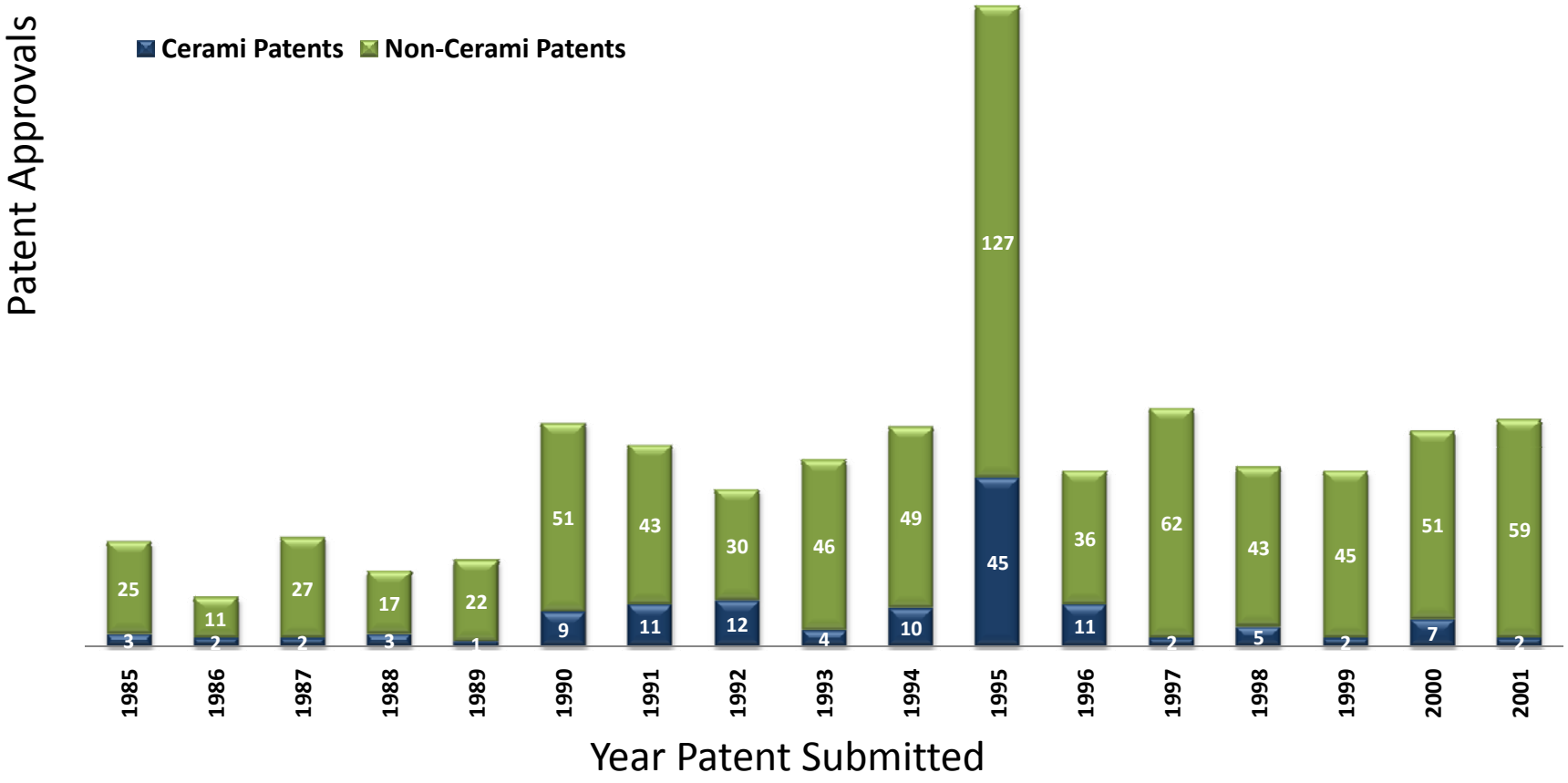
The network of *Enbrel/Remicade-relevant patents* authored by Dr. Anthony Cerami and his immediate collaborators:



Summary Statistics:

The ripple effect of NIH funding

All Patents in Dr. Cerami's Collaborator Network



Summary Statistics:

The ripple effect of NIH funding

ALL PATENTS SUBMITTED 1985-2001

	Anthony Cerami's Direct Collaborators (patent co-authors)	2 ° Collaborators (patent co-authors with Cerami's direct collaborators)
Number of Collaborators	50	123
Total Patents	451	1522
Number of Institutions	8	108

More Information: https://www.starmetrics.nih.gov/