ADDRESSES: The meeting will be held at the Crystal Gateway Marriott, 1700 Jefferson Davis Highway, Arlington, Virginia 22202.

FOR FURTHER INFORMATION CONTACT: Sharon Arnold, Ph.D., Executive Director, Competitive Pricing Advisory Committee, Health Care Financing Administration, 7500 Security Boulevard, C4–14–17, Baltimore, MD 21244–1850, (410) 786–6451.

SUPPLEMENTARY INFORMATION:

Section 4011 of the Balanced Budget Act of 1997 (BBA) (Public Law 105–33), requires the Secretary of the Department of Health and Human Services (the Secretary) to establish a demonstration project under which payments to Medicare+Choice organizations in designated areas are determined in accordance with a competitive pricing methodology. Section 4012(a) of the BBA requires the Secretary to appoint a Competitive Pricing Advisory Committee (the CPAC) to meet periodically and make recommendations to the Secretary concerning the designation of areas for inclusion in the project and appropriate research designs for implementing the project. The CPAC has previously met on May 7, 1998, June 24 and 25, 1998, September 23 and 24, 1998, October 28, 1998, January 6, 1999, May 13, 1999, and July 22, 1999.

The CPAC consists of 15 individuals who are independent actuaries, experts in competitive pricing and the administration of the Federal Employees Health Benefit Program, and representatives of health plans, insurers, employers, unions, and beneficiaries. The CPAC members are: James Cubbin, Executive Director, General Motors Health Care Initiative; Robert Berenson, M.D., Director, Center for Health Plans and Providers, Health Care Financing Administration; John Bertko, Actuary Principal, Reden & Anders, Ltd.; Dave Durenberger, Vice President, Public Policy Partners; Gary Goldstein, M.D., former CEO, The Oschner Clinic; Samuel Havens, Healthcare Consultant and Chairman of Health Scope/United; Margaret Jordan, President and CEO, The Margaret Jordan Group; Chip Kahn, President, The Health Insurance Association of America; Cleve Killingsworth, President, Health Alliance Plan; Nancy Kichak, Director, Office of Actuaries, Office of Personnel Management; Len Nichols, Principal Research Associate, The Urban Institute; Robert Reischauer, Senior Fellow, The Brookings Institution; John Rother, Director, Legislation and Public Policy, American Association of Retired Persons; Andrew Stern, President, Service Employees International Union, AFL–CIO; and Jay Wolfson, Director, Florida Health Information Center, University of South Florida. The chairperson of the CPAC is James Cubbin and the co-chairperson is Robert Berenson, M.D. In accordance with section 4012(a)(5) of the BBA, the CPAC will terminate on December 31, 2004.

The agenda for the September 16, 1999, meeting will include the following:

- A review of the competitive pricing demonstration design.
- A discussion on how to minimize disruption for beneficiaries in the demonstration sites.
- A discussion on the process for review of plan bids.
- A revision of the implementation timeline for the Kansas City, MO Metropolitan Area and Maricopa County, AZ demonstration sites.

Individuals or organizations that wish to make 5-minute oral presentations on the CPAC agenda issues should contact Sharon Arnold, CPAC Executive Director, by 12 noon, September 9, 1999, to be scheduled. The number of oral presentations may be limited by the time available. A written copy of the oral remarks should be submitted to the Executive Director no later than 12 noon, September 9, 1999. Anyone who is not scheduled to speak may submit written comments to the Executive Director, by 12 noon, September 13, 1999.

This meeting is open to the public, but attendance is limited to the space available.

(Sec. 4012 of the Balanced Budget Act of 1997, Pub. L. 105–33 (42 U.S.C. 1395w–23 note) and sec. 10(a) of Pub. L. 92–463 (5 U.S.C. App. 2, sec. 10(a))

(Catalog of Federal Domestic Assistance Program No. 93.773, Medicare—Hospital Insurance; and Program No. 93.774, Medicare—Supplementary Medical Insurance Program)

Dated: August 18, 1999.

Michael M. Hash,

Deputy Administrator, Health Care Financing Administration.

[FR Doc. 99–21859 Filed 8–23–99; 8:45 am]

BILLING CODE 4120–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by contacting Richard U. Rodriguez, M.B.A., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7056 ext. 287; fax: 301/402–0220; e-mail: rr154z@nih.gov. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Peptides and Their Utility in Modulation of Behavior of Cells Expressing $\alpha 3\beta 1$ Integrins

David D. Roberts, Henry C. Krutzsch (NCI)

DHHS Reference No. E–169–99/0 filed 15 Jul 1999

The present invention relates generally to peptides that bind to or are recognized by $\alpha 3\beta 1$ integrins and in particular, to pharmaceutical compositions containing and methods of using said peptides to inhibit or promote various functions of cells that express $\alpha 3\beta 1$ integrins.

Integrins are transmembrane α, $\beta 1$-heterodimer receptors expressed on a wide variety of cells which are involved in extracellular matrix (ECM) interactions. Experimental data has shown that the ECM can affect gene expression and that this altered gene expression can change the composition of the ECM. A bi-directional exchange of information between cells and their surrounding matrix is therefore taking place and because of this communication, integrins can control cell growth, motility, differentiation and survival. Defects in the regulation of these processes can result in many disease states, such as inheritable developmental disorders, defective wound repair, hemotological disorders, cardiovascular diseases, immunological disorders, neurodegenerative diseases and cancer initiation, invasion and metastasis. The disclosed peptides have been shown to inhibit angiogenesis, cell adhesion and proliferation and wound repair when administered in a soluble

form; however, when the same peptides are immobilized on a substratum, they promote adhesion and proliferation of endothelial cells. Thus by controlling the conditions, these peptides can be used to generate specific responses. Specific applications for the peptides include the treatment of angiogenesis-mediated diseases, production of vascular grafts and artificial blood vessels.

Redox-Stable, Non-Phosphorylated Cyclic Peptide Inhibitors of SH2 Domain Binding to Target Protein, Conjugates Thereof, Compositions and Methods of Synthesis and Use Serial No. 60/137,187 filed 02 Jun 1999

Peter Roller, Ya-Qiu Long, Feng-Di Lung, Charles R. King (NCI)

The present invention is predicated on the surprising and unexpected discovery of non-phoshorylated cyclic peptide inhibitors of binding SH2 domains in proteins comprising SH2 domains to target proteins which not only are redox-stable *in vivo* but have unprecedented specific binding affinities.

Src homology–2 (SH2) domains selectively bind to phosphotyrosyl (pTyr)-containing regions of target proteins. SH2 binding can modulate: c-src activity; substrate specificity for c-abl proto-oncoproteins; and the transduction of signals initiated by growth factor receptors and cellular attach systems. The SH2 domain of growth factor receptor-bound protein (Grb2) is a specific example which contains one SH2 domain and two src homology–3 (SH3) domains. The prevention of Grb2-mediated multi-protein assemblies is considered a promising therapeutic target for the development of antiproliferative agents directed to cells that over-express growth factor receptors. Previously identified SH2-inhibitors have detectable activity, but their binding affinities are substantially lower than natural ligands. A need therefore exists for more efficient and stable inhibitors, and the technology herein disclosed provides for such inhibitors. Additionally, the technology offers the possibility of conjugates comprising a compound (SH2 inhibitor) and a carrier agent, i.e., signal peptides, antennapedia peptides, or lipofectin. Suitable targets would preferably include, but not necessarily be limited to: growth factor receptors, such as EGFR; morphology determining proxies, such as FAK; a cellular attachment protein; a proto-oncoprotein; an oncoprotein, such as BCR-abl; or a mitogen-activated protein (MAP). In one application of this method, inhibition of the binding of a target protein by an SH2 domain in a protein comprising an SH2 domain prevents cancer, in particular, breast cancer. Administration of the SH2-inhibitor/SH2-conjugate can be accompanied by an anti-cancer agent such as a chemotherapeutic agent, a cytotoxic agent or its prodrug, radiation and/or a radioactive isotope.

Phenylalanine Derivatives

T. Burke *et al.* (NCI)
Serial No. 60/126,047 filed 23 Mar 1999

The present invention relates to novel phenylalanine derivatives, compositions and methods of using said derivatives to inhibit SH2 domain binding with a phosphoprotein. Additionally, the invention provides precursors suitable for preparing these phenylalanine derivatives.

The therapy and prophylaxis of proliferative diseases such as cancer, autoimmune disorders and hyperproliferative skin disorders can involve signal transduction. These signal-pathways are critical to normal cellular homeostasis and are necessary processes for relaying extracellular messages from various sources, e.g., growth factors, hormones or neurotransmitters, via receptors to the interior of the cell. Protein-tyrosine kinases are integral participants of many of these pathways, and they are responsible for the phosphorylation of specific tyrosine residues to form tyrosine phosphorylated residues. These pathways can involve complex networks which contain proteins with specific amino acid sequences called ''Src-homology 2'' (SH2) domains. Malfunctions in these protein-tyrosine phosphorylations through tyrosine kinase overexpression or deregulation, can manifest a variety of oncogenic and proliferative disorders. SH2 domain containing proteins that play roles in cellular signaling and transformation include, but are not limited to: Src, Lck, Ras GTPase-activating protein, Phospholipase C, PI–3 kinase, Grb2, BCR Abl and Tyk2. Central to the binding of SH2 domains with phosphotyrosine (pTyr)-containing ligands is the interaction of a doubly ionized pTyr phosphate with two highly conserved arginine residues. These interactions are critical, and binding is usually lost by removal of the phosphate group. While the pTyr-pharmacophore therefore plays a dominant role in SH2 domain-ligand interactions, pTyr residues are not suitable components of inhibitors intended for *in vivo* application, due to the enzymatic lability of the phosphate ester bond and the poor cellular penetration of the doubly ionized phosphate species. Therefore, a need exists for non phosphate containing compounds that can mimic the structural interactions of phosphotyrosyl residues within SH2 domain pTyr-binding sites, and in so doing disrupt the interactions between SH2 domains of proteins, e.g., Grb2, and proteins with phosphorylated moieties. The disclosed invention provides viable candidates for these compounds and could provide for the development of therapeutic agents for the treatment of proliferative diseases or conditions as well as relevant diagnostic or testing procedures.

Dated: August 17, 1999.

Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 99–21889 Filed 8–23–99; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the President's Cancer Panel.

The meeting will be open to the public, with attendance limited to space available. Individuals who plan to attend and meet special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

Name of Committee: President's Cancer Panel.

Date: September 22, 1999.

Time: 9:00 AM to 4:00 PM.

Agenda: The Changing Face of Public Health—Implications For The National Cancer Program Now And In The Future.

Place: National Institutes of Health, Building 31, C Wing, Conference Room 10, 9000 Rockville Pike, Bethesda, MD 20892.

Contact Person: Maureen O. Wilson, PhD, Executive Secretary, National Cancer Institute, National Institutes of Health, 31 Center Drive, Building 31, Room 4A48, Bethesda, MD 20892.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399,