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For Grants and Contracts

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The NIH Guide announces scientific initiatives and provides policy and administrative information to individuals and organizations who need to be kept informed of opportunities, requirements, and changes in extramural programs administered by the National Institutes of Health.

Vol. 19, No. 22
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NOTICES

NOTICES OF AVAILABILITY (RFPs AND RFAs)

ONGOING PROGRAM ANNOUNCEMENTS

NOTICE OF MEETING - ELECTRONIC AVAILABILITY OF THE NIH GUIDE FOR GRANTS AND CONTRACTS

P.T. 16; K.W. 1004017

National Institutes of Health

A meeting will be held on September 7, 1990, in Bethesda, Maryland, area concerning the electronic transmission of the NIH Guide for Grants and Contracts. In response to the notice published in the NIH Guide for Grants and Contracts on April 20 (Vol. 19, No. 16), a number of people have expressed interest in a conference concerning the electronic Guide.

As plans are finalized, information and details will be announced in the Guide. Please feel free to contact:

Ms. Rebecca Duvall
Institutional Liaison Office
National Institutes of Health
Building 31, Room 5B31
Bethesda, MD 20892
Telephone: (301) 496-5366
BITNET: Q2C@NIHCU

NOTICES OF AVAILABILITY (RFPs AND RFAs)

OPERATIONS OFFICE

RFP AVAILABLE: RFP-NIH-NIAID-DAIDS-91-07

P.T. 34; K.W. 0755015, 0755018

National Institute of Allergy and Infectious Diseases

The National Institute of Allergy and Infectious Diseases (NIAID), National Institutes of Health, is soliciting proposals from small business organizations having the capabilities and facilities to provide the Division of Acquired Immunodeficiency Syndrome Treatment Research Program with scientific, technical, and operational support to facilitate and significantly reduce the time required to develop, implement, and bring clinical trial protocols to successful conclusion.

This critical contract support will be provided within a research environment and consortium of organizations called the AIDS Clinical Trials Group (ACTG). The ACTG brings together the AIDS Clinical Trials Unit clinical investigators and their institutions, the ACTG Statistical Data and Analysis Center, the Food and Drug Administration, the pharmaceutical industry, HIV-infected patients and their advocates, and Treatment Research Program scientific and administrative personnel to make key decisions about AIDS treatment research issues and conduct clinical trials.

Specifically, the selected Contractor must have the demonstrated scientific, technical, and operational capabilities necessary to play a critical role in the development and review/evaluation of proposed clinical trials protocols; the implementation and conduct of protocols; and the successful completion of protocols. Experience and expertise in the protocol development process, including scientific literature searches and evaluation of methodologies; the ability to monitor the progress of studies and to identify problems and recommend corrective actions and the ability to relate effectively with clinical trials investigators, pharmaceutical industry sponsors, and statistical contractors are all prerequisites for competing under this Request for Proposal (RFP). The nature and complexity of the work under this RFP has evolved substantially in scope and complexity since the establishment of the ACTG in December 1987. This is due in part to the sheer numbers of proposed and implemented protocols, field monitoring and data management tasks, regulatory issues, accountability to multiple organizations, and information flow requirements.

This NIAID-sponsored project will be for five years. A cost-plus-fixed-fee contract is anticipated.

This is an announcement for an anticipated RFP. RFP-NIH-NIAID-DAIDS-91-07 shall be issued on or about June 15, 1990, with a closing date tentatively set for August 15, 1990.

Requests for the proposal should be directed in writing to:

Nancy M. Hershey
Contract Management Branch
National Institutes of Allergy and Infectious Diseases
National Institutes of Health
Control Data Corporation Bldg., Room 222P
6003 Executive Boulevard
Bethesda, MD 20892
Telephone: (301) 496-0193

To receive a copy of the RFP, please provide this office with two (2) self-addressed mailing labels. (THIS IS A 100% SET-ASIDE FOR SMALL BUSINESS). All responsible small business sources may submit a proposal which shall be considered by NIAID. One award is anticipated. This acquisition applies to a 7379 size standard for computer related services, NEC.

This advertisement does not commit the Government to award a contract.

ASTHMA AND ALLERGIC DISEASE COOPERATIVE RESEARCH CENTERS

RFA AVAILABLE: AI-90-05

P.T. 04; K.W. 0715013, 0710070, 0715110, 0715026, 0785035, 0710030

National Institute of Allergy and Infectious Diseases

Letter of Intent Receipt Date: July 27, 1990
Application Receipt Date: October 24, 1990

BACKGROUND INFORMATION

The Asthma and Allergy Branch of the Division of Allergy, Immunology and Transplantation (DAIT) of the National Institute of Allergy and Infectious Diseases (NIAID) sponsors fundamental and clinical research concerned with asthma, allergic and immunologic diseases and with relevant mechanisms of hypersensitivity and inflammation. For this purpose, twelve Asthma and Allergic Disease Centers (AADC) are currently funded. Participation of DAIT staff would be a valuable resource in order to improve the ability of the awardees to design and implement the dissemination of information or technology transfer in the Demonstration and Education projects by coordinating multi-Center projects and would provide cooperative support in planning and data analysis (where needed). It would also improve communication and networking between awardees, foster sharing of information and reagents, and prevent duplication of effort. In order to achieve these objectives, the NIAID intends to phase in a network of Asthma and Allergic Disease Cooperative Research Centers (AADCRC) and phase out the AADC. The present request for applications (RFA) is designed to encourage and invite the development of proposals from interdisciplinary clinical investigative groups interested in integrated studies of asthmatic and allergic processes. New applicants, as well as holders of an AADC grant whose support is terminating, may submit applications for an AADCRC cooperative agreement. Other current holders of an AADC grant may not apply.

RESEARCH GOALS AND SCOPE

The fundamental objective of the NIAID's AADCRC program is to foster acceleration of the application of knowledge of the immune system emerging from relevant biomedical sciences to clinical hypersensitivity disorders. The scope of the AADCRC program involves research designed to foster collaborative approaches that will integrate basic concepts in allergy, immunology, pathophysiology, genetics, microbiology, biochemistry, biostatistics, bioinstrumentation, computer science and pharmacology into clinical investigations, which, in addition to the fields of allergy and clinical immunology, may include such areas as dermatology, rheumatology, infectious diseases, pulmonary medicine, hematology and otorhinolaryngology, when a high degree of relevance to immunology exists. Because the role of hypersensitivity and immune-related inflammatory mechanisms has become increasingly evident in disorders of the skin, immunodermatologic studies are especially encouraged within an AADCRC.

Since transfer of the results of basic and clinical science to the community is an important objective of the AADCRC, a required feature of the AADCRC will

be at least one Demonstration and Education research project, which will test the effectiveness of interventions to promote health or prevent disease in defined populations. Because of the alarming increase in asthma mortality and morbidity since 1979 in minority populations in urban environments, this research will focus on asthma in minority residents in the inner city. Education and demonstration research in this area will be developed as a consensus process in a Coordinating Committee. Demonstration and education research is the testing of the effectiveness of interventions to promote health or prevent disease in defined populations. The interventions selected for such testing should be those that have already been found to be efficacious in other studies and include, but are not limited to, education strategies and modifications in health care and health-related practices. The studies should be based on fields of biomedical, behavioral, and social sciences.

The NIH places special emphasis on the need for inclusion of minorities and women in studies of diseases which disproportionately affect them and also urges that applicants/offerors give added attention, where feasible and appropriate, to their inclusion in other clinical studies. For proposed population-based studies, access to and inclusion of appropriate minority urban populations will be a necessity in order to perform the requisite clinical research.

MECHANISMS OF SUPPORT

Award(s) will be made as a Cooperative Agreement. This is an assistance relationship with substantial involvement of NIAID staff. An AADCRC Cooperative Agreement is awarded to an institution on behalf of a program director for support of a broadly based, multi-disciplinary long-term research program which has a specific objective or basic theme of research on asthma or allergic diseases. An AADCRC generally will involve the organized efforts of groups of investigators who conduct research projects related to the overall program objective. The agreement can provide support for the projects and for certain basic resources shared by individuals where the sharing facilitates the total research effort. Overall, each component project should demonstrate an essential element of unity and interdependence. In FY 1991 the NIAID plans to award at least two AADCRC cooperative agreements and depending on availability of funds and scientific merit more than two. Budgetary requests should be limited to no more than $500,000 total direct costs.

METHOD OF APPLYING

Before preparing an application, the prospective applicant should request a copy of the NIAID Information Brochure on Program Projects, Centers and Cooperative Agreements from:

Dr. Kamal Mittal
Executive Secretary
Allergy, Immunology and Transplantation Research Committee
National Institute of Allergy and Infectious Diseases
National Institutes of Health
Westwood Building, Room 3A06
Bethesda, MD 20892
Telephone: (301) 496-3528

STAFF CONTACT

The complete RFA may be obtained from:

Lawrence J. Prograis, Jr., M.D.
Chief, Asthma and Allergy Branch
Division of Allergy, Immunology and Transplantation
National Institute of Allergy and Infectious Diseases
Westwood Building, Room 752
National Institutes of Health
Bethesda, MD 20892
Telephone: (301) 496-8973
Telefax: (301) 402-0175

THE RFA LABEL AVAILABLE IN THE 10/88 REVISION OF APPLICATION FORM 398 MUST BE AFFIXED TO THE BOTTOM OF THE FACE PAGE. FAILURE TO USE THIS LABEL COULD RESULT IN DELAYED PROCESSING OF YOUR APPLICATION SUCH THAT IT MAY NOT REACH THE REVIEW COMMITTEE IN TIME FOR REVIEW.

IMMUNOLOGIC DISEASE COOPERATIVE RESEARCH CENTERS

RFA AVAILABLE: AI-90-06

P.T. 04; K.W. 0710070, 0755018, 0715013, 0403004, 0404000, 0755030, 0765033

National Institute of Allergy and Infectious diseases

Letter of Intent Receipt Date: July 27, 1990
Application Receipt Date: October 24, 1990

BACKGROUND INFORMATION

The Clinical Immunology Branch of the Division of Allergy, Immunology and Transplantation (DAIT) of the National Institute of Allergy and Infectious Diseases (NIAID) supports research on cellular and molecular mechanisms of immunologic diseases and the application of this knowledge to clinical problems. To support these objectives, 6 Centers for Interdisciplinary Research in Immunologic Diseases (CIRID) are currently funded. The NIAID intends to phase in a network of Immunologic Disease Cooperative Research Centers (IDCRCs) and phase out the CIRIDs for the purpose of having programmatic involvement by DAIT staff in order to improve the ability of the awardees to design and implement the dissemination of information or technology transfer in the Demonstration and Education projects by coordinating multi-Center projects and would provide cooperative support in planning and data analysis (where needed). It would also improve communication and networking between awardees, foster sharing of information and reagents, and prevent duplication of effort. This request for applications (RFA) is intended to encourage the development of applications from collaborative basic science and clinical investigative groups concerned with the study of immunologic diseases. New applicants, as well as holders of a CIRID grant whose support is terminating, may submit applications for an IDCRC cooperative agreement. Other current holders of a CIRID grant may not apply.

RESEARCH GOALS AND SCOPE

The fundamental objective of the IDCRC program is to foster acceleration of the application of knowledge of the immune system generated by research in the relevant biomedical sciences to clinical investigations concerned with immunologic disorders, including asthma and allergic diseases. The goals of the IDCRCs are increased knowledge of the etiology and pathogenetic mechanisms of immunologic diseases and improvements in diagnosis, treatment and prevention of these disorders.

The scope of the IDCRCs includes basic and clinical studies of primary immunologic diseases and other diseases in which a role for the immune system can be identified. Some research areas of interest are inherited and acquired immunodeficiency diseases, autoimmune disorders, interactions of the immune and endocrine systems (e.g. type 1 diabetes), interactions of the immune and nervous systems (e.g. multiple sclerosis), acute and chronic inflammation, immunopathologic aspects of host defense and phagocytes, leukocyte and complement system dysfunctions, asthma and other allergic diseases, and drug reactions. Study of a spectrum of allergic diseases, including asthma should be recognized as one necessary component of a Cooperative Center's program in immunologic diseases.

The transfer of the results of basic and clinical science to the community is an important objective of the IDCRC. Therefore, a required feature of the IDCRC program is the inclusion of at least one Demonstration and Education Research Project. Due to the alarming increase in asthma mortality and morbidity since 1979, especially in urban environments, this research will focus on asthma in minority residents in the inner city. Demonstration and education research in this area will be developed as a consensus process in a Coordinating Committee. Demonstration and education research is the testing of the effectiveness of interventions to promote health or prevent disease in defined populations. The interventions selected for such testing should be those that have already been found to be efficacious in other studies and include, but are not limited to, education strategies and modifications in health care and health-related practices. The studies should be based on fields of biomedical, behavioral, and social sciences.

The NIH places special emphasis on the need for inclusion of minorities and women in studies of diseases which disproportionately affect them and also urges that applicants/offerors give added attention, where feasible and appropriate, to their inclusion in other clinical studies. For proposed population-based studies, access to and inclusion of appropriate minority

urban populations will be a necessity in order to perform the requisite clinical research.

MECHANISM OF SUPPORT

Award(s) will be made as a Cooperative Agreement. This is an assistance relationship with substantial involvement of NIAID staff. An IDCRC Cooperative Agreement is awarded to an institution on behalf of a program director for support of a broadly based, multidisciplinary or interdisciplinary, long-term research program which has a specific major objective or basic theme. An IDCRC generally involves the organized efforts of groups of investigators, members of which conduct research projects related to the overall program objective. The agreement can provide support for the projects and for certain core resources shared by individuals in a program where the sharing facilitates the total research effort. Overall, the projects should demonstrate an essential element of unity and interdependence.

In FY 1991, the NIAID plans to award at least one IDCRC cooperative agreement and depending on availability of funds and scientific merit more than one. budgetary requests should be limited to no more than $500,000 total direct costs.

METHOD OF APPLYING

Before preparing an application, the prospective applicant should request a copy of the NIAID Information Brochure on Program Projects, Centers and Cooperative Agreements from:

Dr. Kamal Mittal
Executive Secretary
Allergy, Immunology and Transplantation Research Committee
National Institute of Allergy and Infectious Diseases
National Institutes of Health
Westwood Building, Room 3A06
Bethesda, MD 20892
Telephone: (301) 496-3528

STAFF CONTACT

A more detailed RFA may be obtained from:

Howard B. Dickler, M.D.
Chief, Clinical Immunology Branch
Division of Allergy, Immunology and Transplantation
National Institute of Allergy and Infectious Diseases
Westwood Building, Room 755
Bethesda, MD 20892
Telephone: (301) 496-7104
Telefax: (301) 480-3780

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ONGOING PROGRAM ANNOUNCEMENTS

BIOLOGY OF AGING SKIN

PA: PA-90-16

P.T. 34; K.W. 0710010, 0715185, 1002004, 0760020, 0755030

National Institute on Aging
National Institute of Arthritis and Musculoskeletal and Skin Diseases

INTRODUCTION

Intrinsic Aging. Skin provides an excellent model to study the fundamental molecular mechanisms of intrinsic biological aging. Several physiological, structural and biochemical changes occur in aging human skin. Cutaneous cell replacement, structural integrity of the epidermis and dermis, immune response, wound repair, and vascular responsiveness are compromised in aged skin. Skin is a highly accessible organ where biopsy specimens can provide tissue and/or cells for in vivo and in vitro studies. In addition, longitudinal data on intrinsic aging changes can be readily obtained. The

three distinct skin compartments, epidermis, dermis, and subcutis, contain a variety of differentiated cell types. Well defined, age-dependent changes have been documented in each of the three compartments.

Epidermal keratinocytes produce the stratum corneum which serves as the major chemical and mechanical barrier of the body. Keratinocyte turnover rate and transit from basal compartment to epidermal surface in aged epidermis is significantly depressed compared to that in young adult tissue. Flattening of the dermo-epidermal junction with effacement of epidermal rete pegs and dermal papillae is a consistent age-dependent change. Epidermal melanocytes (neural crest origin) synthesize protective melanin pigment granules. An age-dependent decrease in enzymatically active melanocytes is characteristic in human skin. Bone marrow-derived Langerhans cells function in antigen presentation and processing. The marked reduction in these cells as a function of age may account, in part, for the age-associated loss of cutaneous immune responsiveness.

The dermis is a complex matrix composed of fibrous proteins and glycosaminoglycans synthesized by dermal fibroblasts. The dermis of aged human skin is relatively acellular and avascular compared to young adult dermis, having fewer fibroblasts, endothelial cells and mast cells. Signs of matrix atrophy are also evident as dermal thinning and structural deterioration. Age-dependent changes in elastic fibers, ranging from small cysts and lacunae to disorganization and fragmentation, are well documented. Age-dependent changes in collagen are also notable. The vasculature embedded in the dermal matrix regulates core body temperature via vasoconstriction and vasodilation. Loss of the microvasculature, especially the vertical capillary loops present in the dermal papillae of young adult skin, is a characteristic age-dependent change.

The subcutis (subcutaneous fat), which provides insulation and mechanical support, is populated by adipocytes. Regional loss of subcutaneous fat is characteristic of aged skin.

Photoaging. Photoaging denotes the degenerative cutaneous changes (actinic damage) that result from chronic ultraviolet radiation (UVR). Photoaged skin is characterized by a telangietactic, leathery, dry surface with blotchy discolorations, deep wrinkles, accentuated furrows, sags, and bags. A variety of benign, premalignant, and malignant lesions are also apparent in photoaged skin. Both UVA (320-400 nm) and UVB (290-320 nm) contribute to photoaging of skin. UVB is responsible for UV-induced sunburn and is the causative agent for premalignant actinic keratosis, squamous cell carcinoma, basal cell carcinoma, and probably malignant melanoma. UVA (either from sunlight or artificial tanning light sources) which is capable of deeper skin penetration than UVB, makes a significant contribution to the characteristic dermal damage in photoaged skin.

Although the photoaging process is often described as "premature aging" or accelerated normal aging, skin changes due to intrinsic aging and photoaging are in fact quite distinct and most likely develop via distinct mechanisms. Marked morphological and structural changes in the epidermis and dermis underlie the observed deterioration in photoaged skin. UVR-induced changes in the epidermis include epidermal thickening, hyperkeratosis, parakeratosis, and acanthosis. UVR-damaged basal keratinocytes show altered morphology and daughter cells show evidence of a defective terminal differentiation program, with consequent development of irregular (scaly) stratum corneum and multiple actinic keratoses. Suprabasal keratinocytes have nuclear inclusions and excessive melanosome accumulation above the nucleus. Dyskeratotic keratinocytes (sunburn cells) also appear in the superficial layers of the epidermis. Melanocytes are increased in number, enlarged and found at higher levels in actinically damaged epidermis. Chronic UVR also induces freckles (melanocyte hyperplasia and depletion of injured cells), solar lentigenes (increased number of highly pigmented melanocytes), and lentigo maligna, a well established precursor to invasive malignant melanoma. UVB alters the immunologic function of Langerhans cells by reduction of antigen-presenting capability, interruption of the normal effector pathway, and activation of the T cell suppressor networks leading to an inappropriate immune response.

Chronic UVR causes unique dermal deterioration including altered cellular function, dermal matrix architecture and composition, and vascular structure and function. Hyperactive fibroblasts, increased deposition of glycosaminoglycans, elevated type III:I collagen ratios, and increased amounts of elastotic material (solar elastosis) are evident in repair zones of photoaged skin. Dermal vessels become dilated and leaky, and show excessive accumulation of basement membrane-like material. Partially degranulated mast cells (chronic inflammation) accumulate around damaged vessels.

A significant reduction in photoaging can be achieved with the use of chemical and physical sunscreening agents and/or limited exposure to sunlight and artificial UV light sources. Combination UVA/UVB sunscreens are most effective for prevention of both epidermal and dermal damage. New damage to photoaged skin can be prevented with sunscreening agents and limited exposure to UVR.

SPECIFIC OBJECTIVES

The age-dependent biochemical and structural changes in normal, sun-protected skin are very distinct from those in photoaged skin. These differences suggest that intrinsic aging and photoaging are governed by distinct mechanisms. The molecular mechanisms which underlie intrinsic aging and photoaging are not known. The National Institute on Aging (NIA) wishes to stimulate research to define the molecular basis of intrinsic aging in skin and to establish the effectiveness and mode of action of intervention strategies to retard and/or reverse age-dependent changes. It is anticipated that research in this area will yield important information on both tissue-specific and fundamental regulatory mechanisms involved in biological aging. The National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) wishes to encourage research to define the molecular mechanisms which underlie skin photoaging and to establish the effectiveness and mode of action of agents which may stimulate repair of actinic damage.

Molecular Mechanisms of Intrinsic Skin Aging

Changes in epidermal and dermal cellularity are well documented age-dependent changes in sun-protected human skin.

- Are age-dependent changes in the control of cell proliferation involved in cellular loss?
- Are changes in the expression of cellular proto-oncogenes, tumor suppressor genes or other cell-cycle regulatory molecules responsible for the observed age-dependent changes?
- Are senescence factors produced by aged skin cells, and if so, by what mechanisms do these senescence factors regulate cell proliferation and/or terminal differentiation of skin cells?
- Are the concentrations of endogenous growth factors altered (stimulatory and inhibitory) in aged skin?
- Are growth factor signal transduction pathways and cellular responses altered in aged skin cells?

What are the underlying molecular mechanisms responsible for loss of epidermal rete pegs and dermal papillae leading to reduced dermo-epidermal adhesion?

Disorganization and degeneration of the dermal matrix, including disorganization of collagen fibrillar units, degradation of elastic fibers, and loss of matrix glycosaminoglycans is an important component of intrinsic aging in human skin.

- Are age-dependent changes in dermal fibroblast gene expression involved in dermal matrix degeneration?
- Are the observed structural changes related to altered expression of collagen I 1 and 2 genes, collagenase, elastin, elastase (serine and metalloprotease forms), and stromelysin genes?
- Are age-dependent changes in the expression of tissue-specific transcription factor genes involved in intrinsic dermal aging?

Disorganization and loss of the microvasculature are characteristic changes in aged skin.

- Do age-dependent changes in angiogenesis contribute to the observed loss?
- Are age-dependent changes in endothelial cell gene expression contributing factors?

Retinoic acid is an endogenous regulator of epidermal keratinocyte and dermal fibroblast gene expression.

- Are age-dependent changes in keratinocyte and fibroblast gene expression modulated by retinoids?
- Are intrinsic age-related changes in epidermis and/or dermis reversed by exogenous retinoids (topical retinoid treatment)?
- Are age-dependent changes in skin retinoid homeostasis (endogenous retinoid metabolite levels and retinoid metabolism) a factor in the intrinsic aging of skin?
- Are age-dependent changes in retinoic acid receptor gene expression (RAR, RARa, RARb) related to intrinsic skin aging?

Molecular Mechanisms of Skin Photoaging

Actinic damage induces keratinocyte, melanocyte, and dermal fibroblast hypertrophy in human skin.

- What are the molecular mechanisms involved in the UVR-induced changes in cell proliferation?
- Do these cellular alterations arise from acute or long-term changes in the control of cell proliferation?

Functionally altered fibroblasts appear to be responsible for dermal matrix degeneration in photoaged skin.

- What is the molecular basis of chronic UVR-induced solar elastosis?
- By what mechanisms does chronic UVR alter collagen I, collagen III, and elastin gene expression in the dermal fibroblast?
- Does chronic UVR alter collagenase and elastase gene expression, and if so, by what mechanisms?
- By what mechanisms does chronic UVR affect glycosaminoglycan metabolism and deposition in the dermal matrix?

Dermal vessels display unique damage and deterioration in photoaged skin.

- What are the mechanisms whereby chronic UVR alters endothelial cell metabolism and function?
- What role do mast cells play in vessel damage? What are the underlying mechanisms of mast cell-mediated damage?

Chronic UVB exposure has a pronounced and prolonged systemic immunosuppressive effect due to altered Langerhans cell function and activation of suppressor T cells.

- What are the mechanisms responsible for the UVB-induced decreases in Langerhans cells in photoaged skin?
- What mechanisms are responsible for compromised Langerhans cell antigen presentation and processing in photoaged skin?
- Which skin cells are involved in the activation of suppressor T cells, and what are the molecular mechanisms reponsible for this inappropriate immune response?

Topical retinoids may enhance the repair of UVR-induced epidermal and dermal matrix damage in photoaged skin.

What are the cellular, biochemical, and structural changes induced by topical retinoids in photoaged skin?

- What are the effects of retinoids on gene expression in differentiated skin cells (i.e. keratinocytes, fibroblasts, endothelial cells)?
- What are the molecular mechanisms by which retinoids regulate gene expression in skin cells?
- Is retinoid homeostasis (levels of endogenous retinoid metabolites, expression of retinoid receptors) altered in photoaged skin?

APPLICATION AND REVIEW PROCEDURES

The primary mechanisms for NIA and NIAMS support of the Biology of Aging Skin program are:

- Research Project Grant (R01)
- Program Project Grant (P01)
- First Independent Research Support and Transition Award (R29)
- Fellowship Grants (F32, F33)

Applicants should use grant application form PHS 398 (revised 10/88) for R01, P01, and R29 applications and form PHS 416-1 (revised 7/88) for F32 and F33 fellowship applications. These forms are available at the applicant's institution or from:

Office of Grant Inquiries
Division of Research Grants
Westwood Building, Room 449
National Institutes of Health
Bethesda, MD 20205
Telephone: (301) 496-7441

To expedite the routing of proposals within NIH, please check "yes" in item 2 of the application face page and indicate that the proposal is in response to NIA/NIAMS: Biology of Aging Skin PA-90-16.

The completed application plus 6 copies should be sent to:

Division of Research Grants
National Institutes of Health
Westwood Building, Room 240
Bethesda, MD 20892**

Receipt dates for Research Project Grant (R01), Program Project Grant (P01), and FIRST Award (R29) applications are February 1, June 1 and October 1; Fellowship application receipt dates are January 10, May 10, and September 10.

All applications submitted in response to this announcement will be assigned according to standard referral guidelines to appropriate NIH study sections for initial scientific review and to the appropriate Institute of NIH for final review by its National Advisory Council or Board. It is anticipated that most applications will have dual Institute assignments. There are no set-aside funds for these proposals. Applications will compete for available funds based on scientific merit. Traditional NIH review criteria for scientific and technical merit will apply to all proposals submitted.

Applications from women and minority scientists are encouraged. Inclusion of minority groups and/or women in study populations, where feasible and appropriate, is also encouraged by the NIH.

Investigators who may be considering submitting proposals in response to the "Biology of Aging Skin" program announcement are encouraged to discuss their research goals and the range of grant mechanisms available with NIA or NIAMS program directors prior to formal submission of research proposals. The appropriate Institute Program Directors are:

Basic Mechanisms of Intrinsic Aging in Skin

Anna M. McCormick, Ph.D.
Director, Genetics Program
Molecular and Cell Biology Branch
Biomedical Research and Clinical Medicine Program
National Institute on Aging
Building 31, Room 5C21
Bethesda, MD 20892
Telephone: (301) 496-6402

Basic Mechanisms of Skin Photoaging

Alan N. Moshell, M.D.
Director, Skin Diseases Branch
National Institute of Arthritis and Musculoskeletal and Skin Diseases
Westwood Building, Room 407A
Bethesda, MD 20205
Telephone: (301) 496-7326

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5333 Westbard Avenue
Bethesda, Maryland 20816

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