

EXTRAMURAL GRANTS

American Cancer Society

Total Grants In Effect On 7/1/2005

Total Number of Grants:

818

Total Amount of Grants:

\$411,678,147

GRANT LISTING IS ON NEXT PAGE

EXTRAMURAL GRANTS

American Cancer Society
Grants in effect on 7/1/2005

Page: 1

Grant No.	Investigator/Institution	Title	Term	Amount
Indiana				
RSG-05-202-01 (GMC)	Baker, Brian M., Ph.D. University of Notre Dame Department of Chemistry and Biochemistry 251 Nieuwland Science Hall Notre Dame, IN 46556	Structure and Thermodynamically Guided Design of Improved Cancer Vaccines	7/1/2005 through 6/30/2009	720,000
IRG-58-006-47 (IRG)	Borch, Richard F., M.D., Ph.D. Purdue University Purdue Cancer Center Hansen Life Sciences Research Building 201 South University Street West Lafayette, IN 47907-2064	Institutional Research Grant	1/1/2005 through 12/31/2007	120,000
RSGPB-04-089-01 (PBP)	Champion, Victoria L., DNS, RN, FAAN Indiana University, Indianapolis School of Nursing NU 338 1111 Middle Drive Indianapolis, IN 46202-5107	Quality of Life in Younger Breast Cancer Survivors	1/1/2004 through 12/31/2008	1,782,000
RSG-03-023-01 (CSM)	D'Souza-Schorey, Crislyn, Ph.D. University of Notre Dame Department of Biological Sciences Box 369 145 Galvin Life Sciences Building Notre Dame, IN 46556-0369	Role of Membrane Traffic in Epithelial Cell Migration	1/1/2003 through 12/31/2006	720,000
DSW-03-214-01 (SW)	Decker, Carol L., M.S.W. Indiana University, Indianapolis Research and Sponsored Programs Suite 110 , Room 618 Indianapolis, IN 46202-5167	Resilience and Quality of Life in Adolescents with Cancer	9/1/2003 through 8/31/2005	40,000

EXTRAMURAL GRANTS

American Cancer Society
Grants in effect on 7/1/2005

Page: 2

Grant No.	Investigator/Institution	Title	Term	Amount
Indiana				
DSCN-04-160-01 (SCN)	Elam, Julie L., M.S.N., R.N. Mentor: Carpenter, Janet S., Ph.D., R.N. Indiana University, Indianapolis School of Nursing NU340D 1111 Middle Drive Indianapolis, IN 46202-5107	Sleep Problems in Breast Cancer Survivors	8/1/2004 through 7/31/2006	30,000
IRG-84-002-19 (IRG)	Erickson, Leonard C., Ph.D. Indiana University, Indianapolis Department of Pharmacology and Toxicology R4-168 1044 W. Walnut Street Indianapolis, IN 46202	Institutional Research Grant	7/1/2002 through 12/31/2005 Funds ended 06/30/05	300,000
RSGPB-05-012-01 (CPPB)	Fife, Betsy L., Ph.D. Indiana University, Indianapolis School of Nursing NU 468 1111 Middle Drive Indianapolis, IN 46202	Adaptation of Families to Bone Marrow Transplant	1/1/2005 through 12/31/2008	907,000
RSG-03-236-01 (MGO)	Franklin, David S., Ph.D. Purdue University Department of Biological Sciences 201 South University Street West Lafayette, IN 47907-2064	Mechanisms of Tumorigenesis Using CDK Inhibitor-Deficient Mice	7/1/2003 through 6/30/2007	720,000
MRS-05-006-01 (CPPB)	Helft, Paul R., M.D. Indiana University, Indianapolis Department of Medicine 535 Barnhill Drive Indianapolis, IN 46202	Impact of Internet Information on Patients and Oncologists	1/1/2005 through 12/31/2009	717,000

EXTRAMURAL GRANTS

American Cancer Society
Grants in effect on 7/1/2005

Page: 3

Grant No.	Investigator/Institution	Title	Term	Amount
Indiana				
RSG-03-011-01 (CCG)	Hinchcliffe, Edward H., Ph.D. University of Notre Dame Department of Biological Sciences Walther Cancer Institute 107 Galvin Life Science Center Notre Dame, IN 46556	Centrosomes and Cell Cycle Progression	1/1/2003 through 12/31/2006	756,000
MRS-05-119-01 (CCE)	Matei, Daniela E., M.D. Indiana University, Indianapolis Department of Medicine 535 Barnhill Drive, RT457 Indianapolis, IN 46202	The Functional Role of PDGFR Alpha in Ovarian Carcinoma	7/1/2005 through 6/30/2010	729,000
RSG-02-192-01 (TBE)	Nephew, Kenneth P., Ph.D. Indiana University, Indianapolis Department of Cellular and Integrative Physiology 302 Jordan Hall 1001 East Third Street Bloomington, IN 47405-4401	Regulation of Estrogen Receptor Function by Ubiquitin-Like NEDD8 Pathway	7/1/2002 through 6/30/2006	782,000
RSG-03-235-01 (MBC)	Sanders, David A., Ph.D. Purdue University Department of Biological Sciences 1392 Lilly Hall West Lafayette, IN 47907	Mechanism and Regulation of Leukemia Virus Entry into Cells	7/1/2003 through 6/30/2007	720,000
RSG-02-028-01 (CNE)	Smith, Martin L., Ph.D. Indiana University, Indianapolis Department of Microbiology and Immunology Room 155 1044 W. Walnut Indianapolis, IN 46202	Role for Wild-Type p53 in Cancer Chemoprevention by Selenium	1/1/2002 through 12/31/2005 Funds ended 12/31/04	377,000

EXTRAMURAL GRANTS

American Cancer Society
Grants in effect on 7/1/2005

Page: 4

Grant No.	Investigator/Institution	Title	Term	Amount
Indiana				
PF-04-034-01 (DDC)	Spike, Caroline A., Ph.D. Mentor: Strome, Susan, Ph.D. Indiana University, Bloomington Department of Biology 1001 East 3rd Street Bloomington, IN 47405-3700	Elucidating the Assembly and Function of Germ Granules in <i>C. Elegans</i>	9/1/2004 through 8/31/2007	124,000
RSG-05-117-01 (CSM)	Vaughan, Kevin T., Ph.D. University of Notre Dame Department of Biological Sciences 057 Galvin Life Sciences Center Notre Dame, IN 46556	The Microtubule Binding Activity of Dynactin and Search-Capture	7/1/2005 through 6/30/2009	668,000
RSG-03-149-01 (CSM)	Walczak, Claire E., Ph.D. Indiana University, Bloomington Department of Medical Sciences Myers Hall 262 915 E. 3rd Street Bloomington, IN 47405	Mechanistic Analysis of the Microtubule Depolymerizing Kinesin, XKCM1	7/1/2003 through 6/30/2007	720,000

Totals for State

Number of Grants: 18
Total Amount of Grants: 10,932,000

Grant Profile Information

One role of the immune system is to distinguish between self and non-self, between the body's own cells and foreign invaders which are generally pathogens such as viruses and bacteria. However, the immune system can also recognize cancer as foreign because cancerous cells often make proteins not usually made by normal cells. The breakdown of these abnormal proteins results in the generation of tumor-associated antigens (TAA), which, when presented to the immune system can flag the cancerous cell as foreign. Despite the existence of TAAs, however, cancerous cells are often ignored by the immune system because most TAAs are ineffective at initiating an immune response. This is thought to result from poor presentation of the antigen to the immune system, which requires formation of a tight complex, or association, between the antigen and the protein molecule MHC. Since most TAAs are incapable of forming the necessary tight complex with the MHC, they are unable to initiate an immune response. Early studies, however, identified ways in which TAAs could be modified to enhance their affinity for the MHC, and once a response is initiated, the immune system is much more capable of reacting with TAAs, suggesting that modified TAAs can serve as therapeutic cancer vaccines.

One of the best examples of a TAA for which this has worked is the gp100-209 melanoma antigen. A simple modification to this antigen enhances its affinity for the MHC, and the modified antigen is much more effective at inducing antitumor immune responses in both laboratory and clinical settings. Our preliminary studies of the gp100-209 antigen and its modified form indicate how the substitution enhances affinity for the MHC, resulting in improved immunological activity.

Our goal is to develop more effective therapeutic cancer vaccines. Our preliminary data suggests ways in which the gp100-209 antigen can be modified to result in even more enhanced affinity for the MHC, perhaps leading to even greater immunological activity. Here, we propose to make and test these modifications. We will examine the effects of modifications on MHC affinity, the structure of the antigen/MHC complex, and immunological potency. We will examine the capacity for the immune system to distinguish between the modified and unmodified antigen. Finally, we will examine whether our strategy can be applied to other TAAs. Our studies have potential for the development of not only improved vaccines for melanoma, but for a myriad of other cancers for which TAAs have been identified.

Grant Profile Information

Research Promotion Information

Brian M. Baker, Ph.D.

Grant No. RSG-05-202-01-GMC

Hometown (currently reside): Granger, Indiana

Publications suggested by grantee to receive an ACS grant press release:

South Bend Tribune, South Bend, Indiana

Alamogordo Daily News, Alamogordo, New Mexico

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

William - Gilroy

Grant Profile Information

Richard F. Borch, M.D., Ph.D.

Institutional Research Grant

Purdue University
Purdue Cancer Center
Hansen Life Sciences Research Building
201 South University Street
West Lafayette, IN 47907-2064
(765) 494-9129 (phone)
(765) 494-9193 (fax)
mkhines@purdue.edu

Grant No. IRG-58-006-47-IRG
Division Great Lakes
Term of Grant: 1/1/2005 - 12/31/2007
Total Award: \$ 120,000
Total ACS Support: \$1,415,666

Priority Areas:

Special Categories:

Project Summary

The purpose of the IRG (Institutional Research Grant) is to provide "seed" money awards for the initiation of promising new projects by junior faculty members (or their equivalents), so they can obtain preliminary results that will enable them to compete successfully for national research grants. IRGs are intended to support independent, self-directed investigators early in their careers, for whom the institution must provide research facilities or space customary for an independent investigator.

Since an IRG is awarded to an institution as a whole, funds should be available to support proposals from all health sciences schools, colleges, and departments. Institutions are encouraged to partner with other institutions that are in the same geographical area, that would not on their own have a sufficiently large pool of junior faculty to apply for an IRG.

Grant Profile Information

Research Promotion Information

Richard F. Borch, M.D., Ph.D.

Grant No. IRG-58-006-47-IRG

Hometown (currently reside): Lafayette, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Lafayette Journal & Courier

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Marilyn K. Hines - (765) 494-9129

Grant Profile Information

Victoria L. Champion, DNS, RN, FAAN

Quality of Life in Younger Breast Cancer Survivors

Indiana University, Indianapolis
School of Nursing
NU 338
1111 Middle Drive
Indianapolis, IN 46202-5107
317-274-4187 (phone)
317-278-2021 (fax)
vchampion@iupui.edu

Grant No. RSGPB-04-089-01-PRP
Division Great Lakes
Term of Grant: 1/1/2004 - 12/31/2008
Total Award: \$1,782,000
Total ACS Support: \$1,782,000

Priority Areas:	Cancer Control Survivorship and Outcomes Research	100%
Organ Sites:	Breast	100%

Special Categories: Psychosocial and Behavioral, Health Policy or Health Services

Project Summary

About 9% of all breast cancer occurs in Caucasian women younger than 45. Breast cancer survivors have lingering problems as a result of treatment such as chemotherapy and radiation. Problems resulting from breast cancer and its treatment may have even more significant effects on younger women. Younger women are often in the middle of raising children or may want to have children. As a result of treatment, many younger women experience menopausal symptoms resulting in lack of fertility, hot flashes, and sexual problems. Partners of younger women may also experience significant problems as a result of their spouse's illness. Partners may have to take on additional responsibilities while maintaining support to their wife. We have little research that addresses the problems younger breast cancer survivors and their partners face.

The purpose of this proposal is to compare younger and older breast cancer survivors' quality of life and to study problems partners may experience. We will use the Eastern Cooperative Oncology Group (ECOG) to identify both younger and older breast cancer survivors. We will study quality of life issues in younger breast cancer survivors and compare their quality of life with older breast cancer survivors. Additionally, acquaintances of the breast cancer survivors will be surveyed to determine if any differences exist between the younger breast cancer survivors and a group of women who have not been diagnosed with cancer. This study will provide information on how we can best help younger women and their families when they experience breast cancer.

Grant Profile Information

Research Promotion Information

Victoria L. Champion, DNS, RN, FAAN

Grant No. RSGPB-04-089-01-PBP

Hometown (currently reside): Avon, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Indianapolis Star News

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Mary Pat Poskon

Grant Profile Information

Crislyn D'Souza-Schorey, Ph.D.

Role of Membrane Traffic in Epithelial Cell Migration

University of Notre Dame
Department of Biological Sciences
Box 369
145 Galvin Life Sciences Building
Notre Dame, IN 46556-0369
(574)631-3735 (phone)
(574)631-7413 (fax)
cdsouzas@nd.edu

Grant No. RSG-03-023-01-CSM
Division Great Lakes
Term of Grant: 1/1/2003 - 12/31/2006
Total Award: \$ 720,000
Total ACS Support: \$ 720,000

Priority Areas:	Biology	100%
Organ Sites:	Breast	20%
	Colon - rectum	20%
	Kidney- renal cell	20%
	Ovary	20%
	Prostate	20%

Special Categories:

Project Summary

Many cancers develop from "epithelial" cells. These include cancers of the colon, kidney, breast, ovary and the prostate. For the onset and progression of malignancy, cells from the primary tumor must acquire the ability to become motile, i.e. to breakaway from associated cells and "migrate" through the surrounding tissue. This allows cancer cells to establish themselves at a secondary site in the body. despite its importance, we have little knowledge of the regulation of processes that can induce a cell to become motile. In this proposal we will investigate mechanisms by which epithelial cells become motile. Our hypothesis is that improper targeting of proteins reequred for cel-cell adhesion to their appropriate destinations can induce the acquisition of the motile phenotype. We will investigate the trasnport routes of adhesion molecules in cells and how these transport processes are regulated. We will also study altered regulation of such transport processes can confer on cells the ability to break away from its nieghboring cells and migrate, an event that correlates with a poor prognosis for cancer patients. A detailed understanding of cellular processes that regulate cell-cell adhesion willl aid in the designing of novel strategies to complement existing therapies that could effectively control cancers that arise from the disruption of these processes.

Grant Profile Information
Research Promotion Information
Crislyn D'Souza-Schorey, Ph.D.
Grant No. RSG-03-023-01-CSM

Hometown (currently reside): Granger, Indiana
Publications suggested by grantee to receive an ACS grant press release:

Grantee willing to discuss your projects with the media? N/A

Grantee willing to discuss to speak at ACS sponsored events? N/A

Grantee willing to serve as an expert or as a member of a speaker's bureau? N/A

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Grant Profile Information

Carol L. Decker, M.S.W.

Resilience and Quality of Life in Adolescents with Cancer

Indiana University, Indianapolis
Research and Sponsored Programs
Suite 110 , Room 618
Indianapolis, IN 46202-5167
317-278-7680 (phone)
317-278-4480 (fax)
cadecker@iupui.edu

Grant No. DSW-03-214-01-SW
Division Great Lakes
Term of Grant: 9/1/2003 - 8/31/2005
Total Award: \$ 40,000
Total ACS Support: \$ 40,000

Priority Areas: Cancer Control Survivorship and Outcomes Research 100%

Special Categories: Psychosocial and Behavioral, Health Policy or Health Services

Project Summary

Despite the numerous demands placed on adolescents who are diagnosed with cancer, many adapt well to experience. A better understanding of these resilient adolescents and related factors such as social support and family environment in this adjustment would assist social workers and other healthcare providers in their care of these adolescents. This knowledge would also provide direction for programs to assist adolescents in this adjustment process.

Grant Profile Information

Research Promotion Information

Carol L. Decker, M.S.W.

Grant No. DSW-03-214-01-SW

Hometown (currently reside):

Publications suggested by grantee to receive an ACS grant press release:

Indianapolis Star

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

i am interested in speaking to any community groups or on the topic of psychosocial adjustment to pediatric cancer or on my findings from this research project .

Institution Publicity Contact:

Mary Roberts Assistant to Dean Indiana Scholl of Social Work 317-274-8362

Grant Profile Information

Julie L. Elam, M.S.N., R.N.

Sleep Problems in Breast Cancer Survivors

Indiana University, Indianapolis
1944 William Glen Boulevard
Zionville, IN 46077
317-278-6095 (phone)
317-278-2021 (fax)
jlelam@iupui.edu

Grant No. DSCN-04-160-01-SCN
Division Great Lakes
Term of Grant: 8/1/2004 - 7/31/2006
Total Award: \$ 30,000
Total ACS Support: \$ 30,000

Priority Areas: Cancer Control Survivorship and Outcomes Research 100%

Organ Sites: Breast 100%

Special Categories: Psychosocial and Behavioral, Health Policy or Health Services

Project Summary

Sleep problems are a common complaint in women surviving breast cancer. Sleep problems can be due to inability to fall asleep, frequent events that wake a person up, inability to fall back asleep, or being uncomfortable during sleep. Sleep problems may be related to environmental factors such as poor companion sleep (e.g., partner with restless legs, partner who snores, unfamiliar environment, noise), physical symptoms (e.g., pain, hot flashes, restless legs), emotional distress (e.g., depression, anxiety, stress), and/or medication use (e.g., chemotherapy, steroids, anti-depressant use). Sleep problems are linked to fatigue, depression and negatively impact quality of life. Further study of this problem is needed to improve quality of life in breast cancer survivors.

Grant Profile Information

Research Promotion Information

Julie L. Elam, M.S.N., R.N.

Grant No. DSCN-04-160-01-SCN

Hometown (currently reside): Noblesville, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Indianapolis Star

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Kelly Young - 317-274-7711

Grant Profile Information

Leonard C. Erickson, Ph.D.

Institutional Research Grant

Indiana University, Indianapolis
Department of Pharmacology and Toxicology
R4-168
1044 W. Walnut Street
Indianapolis, IN 46202
317-274-5202 (phone)
317-274-8046 (fax)
lcericks@IUPUI.edu

Grant No. IRG-84-002-19-IRG
Division Great Lakes
Term of Grant: 7/1/2002 - 12/31/2005
Total Award: \$ 300,000
Total ACS Support: \$1,539,000

Priority Areas:

Special Categories:

Project Summary

The purpose of the IRG (Institutional Research Grant) is to provide "seed" money awards for the initiation of promising new projects by junior faculty members (or their equivalents), so they can obtain preliminary results that will enable them to compete successfully for national research grants. IRGs are intended to support independent, self-directed investigators early in their careers, for whom the institution must provide research facilities or space customary for an independent investigator.

Since an IRG is awarded to an institution as a whole, funds should be available to support proposals from all health sciences schools, colleges, and departments. Institutions are encouraged to partner with other institutions that are in the same geographical area, that would not on their own have a sufficiently large pool of junior faculty to apply for an IRG.

Grant Profile Information

Research Promotion Information

Leonard C. Erickson, Ph.D.

Grant No. IRG-84-002-19-IRG

Hometown (currently reside): Zionsville, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Grantee willing to discuss your projects with the media?

N/A

Grantee willing to discuss to speak at ACS sponsored events?

N/A

Grantee willing to serve as an expert or as a member of a speaker's bureau?

N/A

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Grant Profile Information

Betsy L. Fife, Ph.D.

Adaptation of Families to Bone Marrow Transplant

Indiana University, Indianapolis
School of Nursing
NU 468
1111 Middle Drive
Indianapolis, IN 46202
(317) 274-8788 (phone)
(317) 278-2021 (fax)
slynch2@iupui.edu

Grant No. RSGPB-05-012-01-CPPB
Division Great Lakes
Term of Grant: 1/1/2005 - 12/31/2008
Total Award: \$ 907,000
Total ACS Support: \$ 907,000

Priority Areas:

Special Categories: Psychosocial and Behavioral, Health Policy or Health Services

Project Summary

Bone Marrow transplantation is commonly used to treat a variety of forms of cancer; yet, it is associated with a high degree of risk, recovery is prolonged, and it challenges the coping abilities of both the recipient and family members. Research has demonstrated that the psychological and social impact on the recipient include anxiety, depression which is sometimes severe, loss of a sense of personal control, and an inability to resume previous roles among other responses. However, research examining the impact of this treatment on the family, who provide care for the recipient throughout recovery has just begun. Furthermore, the care they are obliged to give often involves procedures that require considerable skill, which many family members find intimidating. If we are successful in treating the illness and in turn the family suffers irreversible damage, what have we accomplished?

Intervention is needed to prevent secondary psychological and social problems within the family, but we must first understand what the specific problems are, at what point in the treatment trajectory they occur, and be able to identify which family units and individual family members are most vulnerable to developing serious problems. The purpose of this study will be to address these issues. We will study 150 families of individuals receiving BMT over a period of one year. We will gather data at specific time points by questionnaire from each family member including all children 11 years of age and older, and we will use interviews to obtain data from children.

Grant Profile Information

Research Promotion Information

Betsy L. Fife, Ph.D.

Grant No. RSGPB-05-012-01-CPPB

Hometown (currently reside): Indianapolis, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Indianapolis Star

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Kelly Young - (317) 274-7711

Grant Profile Information

David S. Franklin, Ph.D.

Mechanisms of Tumorigenesis Using CDK Inhibitor-Deficient Mice

Purdue University
Department of Biological Sciences
201 South University Street
West Lafayette, IN 47907-2064
765-494-8185 (phone)
765-496-1496 (fax)
franklin@bilbo.bio.purdue.edu

Grant No. RSG-03-236-01-MGO
Division Great Lakes
Term of Grant: 7/1/2003 - 6/30/2007
Total Award: \$ 720,000
Total ACS Support: \$ 720,000

Priority Areas:	Biology	50%
	Scientific Model Systems	50%

Special Categories:

Project Summary - Next Page

Grant Profile Information

A common feature of cancer cells is the ability to maintain continual proliferation, when normal cells would either grow at a reduced rate or cease dividing. To regulate whether to proliferate or arrest growth, our cells have developed a series of decision making "check points" at which cells evaluate whether proliferation should continue or cease. Combined, the series of check points are termed the cell cycle machinery. The cell cycle check points are regulated by proteins known as cyclin-dependent kinases (CDKs). When active, CDKs allow the cells to progress from one check point to the next. In cells that should not divide, the CDKs must be inactive, a function regulated by a family of CDK inhibitors (CDKIs). Although much is known about how CDKIs regulate CDK activity, little is known about how CDKIs actually work, how they are regulated, how their inhibitory functions are normally received and transmitted to regulate the CDKs and the cell cycle, and what happens to the CDKIs in human cancers that facilitates cell cycle dysfunction. We are just beginning to appreciate the importance of understanding these aspects of CDKI function. To examine how CDKIs normally function, mice were generated that lack two important CDKIs, p18 and p27. These CDKIs are absent in every cell within the mouse. Significantly, these mice develop multiple endocrine tumors, paralleling the human cancer syndrome multiple endocrine neoplasia (MEN). In this proposal, we focus our efforts on characterizing how p18, p27, and the cell cycle are regulated by the tumor suppressor gene, MEN1 and its protein MENIN. How does MENIN regulate expression and function of p18 and p27 to regulate the cell cycle? Mutational alteration in MEN1 predispose humans to MEN tumors. How do these mutant variants of MENIN deregulate p18, p27, and the cell cycle to cause cancer? The goals of this proposal are to understand the regulation and function of p18 and p27 during normal growth and during tumor formation. First, does MENIN regulate p18 and p27? Second, how do p18 and p27 affect growth arrest signals from MENIN to the cell cycle machinery? Third, how do p18 and p27 become inactivated during tumor formation and can this be precipitated by mutant forms of MENIN? Finally, can we develop acceptable mouse models by which we can study the human MEN syndrome? These aims are focused on specific goals: to examine how MENIN and the CDKIs regulate growth, and to provide mechanisms by which tumors develop in human cancers.

Grant Profile Information

Research Promotion Information

David S. Franklin, Ph.D.

Grant No. RSG-03-236-01-MGO

Hometown (currently reside): West Lafayette, Indiana
Publications suggested by grantee to receive an ACS grant press release:

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Fund Raising Events, Relay for Life, etc...

Institution Publicity Contact:

Joseph Bennett

Grant Profile Information

Paul R. Helft, M.D.

Impact of Internet Information on Patients and Oncologists

Indiana University, Indianapolis
Department of Medicine
535 Barnhill Drive
Indianapolis, IN 46202
317-278-6942 (phone)
317-278-0079 (fax)
phelft@iupui.edu

Grant No. MRS-G-05-006-01-CPPB
Division Great Lakes
Term of Grant: 1/1/2005 - 12/31/2009
Total Award: \$ 717,000
Total ACS Support: \$ 717,000

Priority Areas: Cancer Control Survivorship and Outcomes Research 100%

Special Categories: Psychosocial and Behavioral, Health Policy or Health Services

Project Summary

Americans are rapidly increasing their use of the Internet to obtain health information. As the second leading cause of death in adult Americans, cancer has become the most common disease for which patients seek information on the Internet. Cancer patients now have unprecedented access to information. These changes, brought about by the Internet, represent a substantial opportunity to engage patients in their own medical care and decision making. Cancer patients' use of the Internet is probably already having profound effects on them and on their clinical encounters with oncologists. However, almost no empirical information is available concerning the impact on patients, providers and their interactions. The specific aims of this project are to: 1) gain a deeper understanding of the positive and negative effects of Internet use by cancer patients on satisfaction with information, providers, communication, decisions, and level of engagement 2) qualitatively evaluate the impact of Internet information on doctor-patient communications and patients' engagement in clinical decision making and 3) describe the associations between sociodemographic factors, information seeking preferences, use of the Internet to obtain cancer information, and discussion of Internet information in the clinical encounter, and satisfaction with decisions, providers, communication, and level of engagement. The methods to be used include focus groups, audiotaped patient consultations, and self-administered questionnaires. Subjects include indigent and non-indigent cancer patients recruited from a large, urban medical center and its affiliates. This project will lead to an improved understanding of the potential benefits and harms of using the Internet to obtain cancer information. This understanding will provide a rational basis for designing Web-based interventions targeting specific subgroups of cancer patients. These interventions will aim to improve patients' engagement in their own care, satisfaction with decisions, and doctor-patient communications through more effective use of information resources. It will also serve as a foundation for designing future interventions aimed at overcoming barriers to Internet access and use faced by many underserved, vulnerable cancer patients.

Grant Profile Information

Research Promotion Information

Paul R. Helft, M.D.

Grant No. MRSB-05-006-01-CPPB

Hometown (currently reside): Indianapolis, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Indiana University Cancer Center Newsletter

Indianapolis Star (newspaper)

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Grant Profile Information

Daniela E. Matei, M.D.

The Functional Role of PDGFR Alpha in Ovarian Carcinoma

Indiana University, Indianapolis
Department of Medicine
535 Barnhill Drive, RT457
Indianapolis, IN 46202
317 278 8844 (phone)
317 278 0074 (fax)
dmatei@iupui.edu

Grant No. MRSB-05-119-01-CCF
Division Great Lakes
Term of Grant: 7/1/2005 - 6/30/2010
Total Award: \$ 729,000
Total ACS Support: \$ 729,000

Priority Areas:

Organ Sites: Ovary 100%

Special Categories:

Project Summary - Next Page

Grant Profile Information

Background: Ovarian cancer is the leading cause of mortality among gynecological malignancies. With current treatment, the majority of patients succumb to their disease. Advancement in ovarian cancer therapy requires an in-depth understanding of the biology of the disease. We utilized state of the art microarray analysis and we identified that the platelet derived growth factor receptor (PDGFR) alpha is present at high levels in ovarian cancer cells and in ovarian tumors. This growth factor receptor is a protein present in the cancer cell membrane. It can be activated within the tumor bed and subsequently can drive and sustain the growth and the expansion of ovarian tumors. The functional role of the PDGF-PDGFR growth pathway has not been previously studied in ovarian cancer. Our preliminary data suggest that this mechanism is important for regulating the growth of ovarian tumors. We showed that blockade of this receptor with an anti-cancer agent (Gleevec) can stop the growth of ovarian cancer cells which harbor PDGFR. This may offer an opportunity for more specific anti-cancer therapy for women with ovarian cancer.

Hypothesis: Based on our preliminary data, we hypothesized that engagement of the PDGF-PDGFR signaling pathway is very important for ovarian tumor growth and survival.

We will test this hypothesis by pursuing the following three objectives:

Aim 1: Investigate mechanisms of activation of the PDGFR pathway in human ovarian tumors. For this objective, we will analyze ovarian tumors for the presence of the PDGF receptor and for the presence of proteins, which can be activated by this receptor. We will also study whether the presence of this protein influences prognosis and survival of women with ovarian cancer.

Aim 2: Characterize the biological effects of modulation of PDGFR expression for the survival and growth of ovarian cancer cells and tumors. To accomplish this goal, we will use standard methods of PDGFR over-expression (increased levels of PDGFR) and knock-down (decreased levels of PDGFR) to study how these alterations modify the growth and the survival of ovarian cancer cells.

Aim 3: Determine the significance of signaling mechanisms, downstream of the PDGFR, to the growth and survival of ovarian cancer cells and tumors. To accomplish this goal, we will study the mechanisms used by PDGFR to control the growth of ovarian cancer cells.

In summary, this is a hypothesis-driven research proposal aiming to characterize the PDGF-PDGFR growth pathway in ovarian tumors. Our hypothesis

Grant Profile Information

Research Promotion Information

Daniela E. Matei, M.D.

Grant No. MRS-05-119-01-CCE

Hometown (currently reside): Indianapolis, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Indiana University Cancer Center Newsletter

Indiana University School of Medicine Newsletter

Indianapolis Star

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Grant Profile Information

Kenneth P. Nephew, Ph.D.

Regulation of Estrogen Receptor Function by Ubiquitin-Like NEDD8 Pathway

Indiana University, Indianapolis
Department of Cellular and Integrative
Physiology
302 Jordan Hall
1001 East Third Street
Bloomington, IN 47405-4401
812-855-9445 (phone)
812-855-4436 (fax)
knephew@indiana.edu

Grant No. RSG-02-192-01-TBF
Division Great Lakes
Term of Grant: 7/1/2002 - 6/30/2006
Total Award: \$ 782,000
Total ACS Support: \$ 782,000

Priority Areas:	Biology	80%
	Etiology	20%
Organ Sites:	Breast	50%
	Endometrium	20%
	Uterus	30%

Special Categories:

Project Summary

The association between breast cancer and estrogen is well known. Breast cancer cells often contain steroid receptors, and the estrogen receptor (ER) represents a crucial point of control of estrogen action. The ER content of individual tumors is a valuable predictor of whether an individual patient will respond to endocrine therapy, such as treatment with with antiestrogens like tamoxifen. Tamoxifen therapy is effective in the treatment of breast cancer, but the response of tumors to tamoxifen can be variable. Because most breast tumors eventually fail to respond to treatment with the antiestrogen, tamoxifen resistance is also major limitation. Furthermore, the use of tamoxifen is complicated by its estrogen-like side effects, particularly the well-documented increase in endometrial cancer in women taking the drug for treatment and prevention of breast cancer. Because many breast cancer therapies target ER, the development of ideal therapies for breast cancer will require a full understanding of the receptor and how it is regulated. We have identified a novel protein called Uba3. We have demonstrated that Uba3 functions as an ER inhibitor. Uba3 is part of a recently discovered ubiquitin-like pathway called NEDD8 (that is, neddylation) that regulates cellular functions. A role for the neddylation pathway in ER regulation represents what may be an important new insight into ER function. The proposed studies on mechanisms that regulate ER levels and activity are highly relevant to the development and progression of hormone dependent cancers, such as breast cancer, including tumor progression to states of hormone independence and antiestrogen resistance. These studies will also examine the mechanism of tamoxifen action in the uterus, and may shed light on why the drug can increase the risk of endometrial cancer.

Grant Profile Information

Research Promotion Information

Kenneth P. Nephew, Ph.D.

Grant No. RSG-02-192-01-TBE

Hometown (currently reside): Bloomington, Illinois

Publications suggested by grantee to receive an ACS grant press release:

Herald Times; Indianapolis Star; The IU Medical School SCOPE, IU Cancer Center Newsletter

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Pamela Perry, Medical School Relations

Grant Profile Information

David A. Sanders, Ph.D.

Mechanism and Regulation of Leukemia Virus Entry into Cells

Purdue University
Department of Biological Sciences
1392 Lilly Hall
West Lafayette, IN 47907
765-494-6453 (phone)
765-496-1189 (fax)
retrovir@bragg.bio.purdue.edu

Grant No. RSG-03-235-01-MBC
Division Great Lakes
Term of Grant: 7/1/2003 - 6/30/2007
Total Award: \$ 720,000
Total ACS Support: \$ 720,000

Priority Areas: Etiology 100%

Organ Sites: Leukemia 100%

Special Categories:

Project Summary

Infection with human T-cell leukemia virus is now a global epidemic, affecting up to 20 million people. This virus is an etiologic agent of adult T-cell leukemia/lymphoma (ATLL). In addition, coinfection with HTLV-I and HIV has been shown to accelerate the progression of acquired immune deficiency syndrome (AIDS) and the consequent susceptibility to associated cancers. We are investigating how a leukemia virus that is closely related to the human T-cell lymphotropic virus enters cells. Our results will certainly continue to have immediate applications towards understanding and preventing HTLV-1 entry into cells. In addition, the virus that we study is a common vehicle for retrovirus-mediated gene therapy including anticancer therapies. The results of our research will have important implications for the enhancement of retrovirus-mediated gene therapy including the construction of viruses with specificity for entry into particular cells such as tumor cells. Finally proteins closely related to those with which we work have been utilized recently as an antitumor agent themselves, and furthering our understanding of their mechanism of action will facilitate improvements in their capacities.

Grant Profile Information

Research Promotion Information

David A. Sanders, Ph.D.

Grant No. RSG-03-235-01-MBC

Hometown (currently reside): West Lafayette, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Lafayette Journal and Courier

Indianapolis Star

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Joseph Bennett

Grant Profile Information

Martin L. Smith, Ph.D.

Role for Wild-Type p53 in Cancer Chemoprevention by Selenium

Indiana University, Indianapolis
Department of Microbiology and Immunology
Room 155
1044 W. Walnut
Indianapolis, IN 46202
317-278-0225 (phone)
312-274-7592 (fax)
marlsmit@iupui.edu

Grant No. RSG-02-028-01-CNE
Division Great Lakes
Term of Grant: 1/1/2002 - 12/31/2005
Total Award: \$ 377,000
Total ACS Support: \$ 377,000

Priority Areas:	Biology	33%
	Prevention	67%
Organ Sites:	Breast	100%

Special Categories:

Project Summary

Even though much progress has been made in treating certain cancers, others, such as breast cancer, remain a difficult challenge. Because a single cause is not known, perhaps more emphasis should be placed on prevention. Since cancer is a multistep process, intervention at points along known molecular pathways could theoretically block cancer progression. The cancer chemoprevention agent selenium can activate the p53 tumor suppressor pathway and enhance DNA repair. Dietary selenium supplementation, in addition to augmentation of DNA repair in healthy individuals, may also promote more robust levels of DNA repair in high-risk individuals. Because selenium has been shown to dramatically reduce the incidence of mammary cancers in rodent models, we want to explore the mechanism of selenium activity, involving the p53 tumor suppressor and DNA repair. Low-level DNA damage occurs frequently within an organism on a daily basis, and it is likely that the capacity to repair this damage is important in cancer prevention.

Grant Profile Information

Research Promotion Information

Martin L. Smith, Ph.D.

Grant No. RSG-02-028-01-CNE

Hometown (currently reside): Indianapolis, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Indianapolis News

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Ms. Pamela Perry

Grant Profile Information

Caroline A. Spike, Ph.D.

Elucidating the Assembly and Function of Germ Granules in *C. Elegans*

Indiana University, Bloomington
Department of Biology
Jordan Hall 142
1001 E. Third Street
Bloomington, IN 47405-3700
812-855-7271 (phone)
812-855-6705 (fax)
cspike@bio.indiana.edu

Grant No. PF-04-034-01-DDC
Division Great Lakes
Term of Grant: 9/1/2004 - 8/31/2007
Total Award: \$ 124,000
Total ACS Support: \$ 124,000

Mentor: Susan Strome, Ph.D.

Priority Areas:	Biology	95%
	Scientific Model Systems	5%

Special Categories:

Project Summary

Germ cells and cancer cells share at least two important traits. First, it has been well documented that cancer cells are in some sense de-differentiated since they lack the distinctive appearance and behavior of normal differentiated cells. Second, cancer cells proliferate in an uncontrolled fashion, while normal differentiated cells divide rarely, if at all. Primordial germ cells, the precursors of the specialized cells required for reproduction (sperm and egg), are also an undifferentiated cell type that is capable of rapid proliferation. Primordial germ cells are set apart early in embryogenesis from somatic cell precursors, and retain their capacity for proliferation throughout much of development. In many organisms, germ cells are marked as different from normal somatic cells (at least in part) by subcellular structures known as germ granules. This proposal aims to use the model organism *Caenorhabditis elegans*, an excellent system for genetic analysis of germ cell development, to identify new factors that associate with germ granules and are required for germ cell proliferation and normal germ cell development. These studies should be relevant to other organisms since at least one component of germ granules (GLH-1/VASA) is important for germ cell development in a variety of species. Ultimately, studies of germ granules will help us understand how all types of cells remain in an undifferentiated and proliferative state.

Grant Profile Information

Research Promotion Information

Caroline A. Spike, Ph.D.

Grant No. PF-04-034-01-DDC

Hometown (currently reside): Bloomington, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

George Vlahakis

Grant Profile Information

All cells of the body arise from the division of a pre-existing mother cell - each division yields two daughter cells. Thus, we develop from a single fertilized egg into a multicellular organism through many cell divisions. Our cells "know" where they are in the body, and they know when to proliferate (divide many times), and when to stop dividing. This is why we grow to a defined shape and size, and then our growth stops.

The problem arises when cells inherit certain growth characteristics that cause them to lose their place in the plan. These cells grow uncontrollably, and move to different places within the body. These cancer cells cannot respond to the normal cues that tell them when and where to go, when to divide, when to grow or when to die. A major research focus of cellular biology is understanding how cells acquire these lethal growth characteristics.

One answer to this question may be during the cell division process itself. The most critical facet of cell division is the equal segregation of DNA to each daughter cell. Chromosomes are analogous to volumes of an encyclopedia: the DNA represents the entire instruction manual for making our cells, and each chromosome is one volume of that manual, containing a specific set of instructions. The cell makes an exact copy of its DNA (so it has two copies). When the mother cell divides, each daughter cell gets exactly one complete copy of chromosomes. However, if a mistake is made, and one daughter gets too few or too many copies of a particular volume, they cell can become cancerous. Thus, we are very interested in understanding how is it that cells divide their DNA, and how do they ensure that there are no mistakes in the process.

My work focuses on a part of the cell called the centrosome. The centrosome was discovered over one hundred years ago. Just like DNA, there is one centrosome in the mother cell, and then the centrosome reproduces or duplicates to form two daughter centrosomes - one will go to each daughter cell. It is the job of the centrosome to help build the cellular scaffold that will separate the chromosomes to the daughter cells. Thus, the centrosome plays a crucial role in ensuring that each daughter cell gets the right amount of DNA. But we do not know what that role is.

To figure out what the centrosome is doing in the cell, we remove it, and then watch what happens to cells when they divide without centrosomes. This tells us which activities in cell division require the centrosome, and which do not. The hope is that we will uncover new aspects of the cell division process. By knowing more about how the process works, we can provide input into the design of anti-cancer therapies.

Grant Profile Information

Research Promotion Information

Edward H. Hinchcliffe, Ph.D.

Grant No. RSG-03-011-01-CCG

Hometown (currently reside): South Bend, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Soth Bend Tribune

Grantee willing to dicuss your projects with the media? Yes

Grantee willing to dicuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Loretta Wasmusth

Grant Profile Information

Kevin T. Vaughan, Ph.D.

The Microtubule Binding Activity of Dynactin and Search-Capture

University of Notre Dame
Department of Biological Sciences
057 Galvin Life Sciences Center
Notre Dame, IN 46556
(574)631-3733 (phone)
(574)631-7413 (fax)
vaughan.4@nd.edu

Grant No. RSG-05-117-01-CSM
Division Great Lakes
Term of Grant: 7/1/2005 - 6/30/2009
Total Award: \$ 668,000
Total ACS Support: \$ 668,000

Priority Areas:

Organ Sites:	Colon - rectum	50%
	Prostate	50%

Special Categories:

Project Summary - Next Page

Grant Profile Information

In cancer, cells of our bodies change from being a good citizen to becoming selfish. Mutations in genetic material (DNA) are recognized as the major cause of these transitions in cancer. Although there are many mechanisms, mistakes during the process of cell division are thought to cause most mutations. During the aging process, these mistakes accumulate until a threshold is reached that we cannot tolerate. This proposal is focused on the processes which mediate segregation of DNA during cell division. Many aspects of this process are poorly understood, making it difficult to define the exact mistakes being made, and what drugs can be most effective. During the cell division process, DNA structures called chromosomes interact with filamentous structures called microtubules which function as roadways for transport into the new daughter cells. Mistakes in these interactions with microtubules are now thought to cause many of the defects we see in cancer cells. For example, loss of chromosomes during this process results in the loss of "tumor suppressor" genes whose job it is to protect cells from progression to cancer. The experiments in this proposal will advance our understanding of how the interactions between chromosomes and microtubules occur, how they are controlled and how mistakes can be corrected with anti-cancer therapies. This project is focused on a protein (dynactin) which functions as the "trailer-hitch" for one of the molecular motors responsible for chromosome transport during cell division. In addition to functioning as a "hitch", our work reveals a new and unexpected function. Our goals are to determine the significance of this new function, and to determine if this is a mutagenic target during cancer initiation. We are particularly intrigued because this protein localizes to structures in the cell undergoing initial contact with these mitotic motors. Several hypotheses have been proposed to explain the significance of this coincidence, but we have evidence which suggests that this protein actually mediates the contact. This identifies our candidate protein as an important component of this process, and a likely target for mistakes and mutations. One significant challenge in the analysis of this process is that these events are hard to observe because of technical limitations. The Vaughan laboratory has developed new model systems which overcome many of the technical limitations associated with this imaging, allowing our laboratory to make substantial progress on understanding this process. Our goals in this project are to determine the function of proteins involved in initiating contact with chromosomes during mitosis, starting in this project with one promising candidate. We will determine what functions this protein carries out, identify mechanisms that lead to cancer in humans, and identify targets for drug and gene therapy.

Grant Profile Information

Research Promotion Information

Kevin T. Vaughan, Ph.D.

Grant No. RSG-05-117-01-CSM

Hometown (currently reside): Granger, Indiana

Publications suggested by grantee to receive an ACS grant press release:

South Bend Tribune

Notre Dame Magazine

Renaissance (ND College of Science Publication)

Pathways (Departmental Publication)

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Dennis Brown - 574-631-4313

Grant Profile Information

Claire E. Walczak, Ph.D.

Mechanistic Analysis of the Microtubule Depolymerizing Kinesin, XKCM1

Indiana University, Bloomington
Department of Medical Sciences
Myers Hall 262
915 E. 3rd Street
Bloomington, IN 47405
812-855-5919 (phone)
812-855-6082 (fax)
cwalczak@indiana.edu

Grant No. RSG-03-149-01-CSM
Division Great Lakes
Term of Grant: 7/1/2003 - 6/30/2007
Total Award: \$ 720,000
Total ACS Support: \$ 815,000

Priority Areas:	Biology	60%
	Scientific Model Systems	40%
Organ Sites:	Breast	60%
	Leukemia	25%
	Lung	5%
	Non-Hodgkin's Lymphoma	5%
	Ovary	5%

Special Categories:

Project Summary - Next Page

Grant Profile Information

Our lab studies the process by which cells divide (mitosis) so that we can understand what goes wrong in the uncontrollable divisions of cancerous cells. We focus our studies on understanding how the molecular machinery that is used to segregate chromosomes to two daughter cells functions. The cellular machine that segregates chromosomes is known as the mitotic spindle and is comprised of a protein called tubulin as well as hundreds of accessory proteins. Tubulin is a component of a fibrous polymer known as a microtubule. A growing class of antimetabolic drugs, including taxol and the vinca alkaloids vinblastine and vincristine are targeted to microtubules and interfere with their function. Initially, it was thought that these antimetabolic drugs interfered with mitosis by causing dramatic changes in the microtubule polymer content of the spindle. More recent studies suggest that these drugs act by suppressing only one aspect of microtubule function and thus halt cell division. After prolonged periods of mitotic arrest, the cells eventually undergo apoptosis and die. The effectiveness of the antimetabolic drugs varies with the type of cancer. For instance, taxol is routinely used for treatment of breast and ovarian cancers but has had little effect on leukemias. Vinblastine and vincristine on the other hand are part of the regimen often used to treat leukemia and lymphoma. The differences in the efficacy of treatment are not yet understood but illustrate the importance of having a diversity of drugs targeted at microtubules of the mitotic spindle. It is therefore reasonable to speculate that development of new drugs that target microtubules would make viable therapeutic agents. We propose to study other proteins that regulate the mitotic spindle and to compare their function to the action of drugs such as taxol or vinblastine. This proposal focuses on the study of one such molecule, XKCM1, whose biochemical function is to regulate microtubule function in cells. Thus, XKCM1 may provide a novel therapeutic target for the treatment of many types of cancers.

Grant Profile Information

Research Promotion Information

Claire E. Walczak, Ph.D.

Grant No. RSG-03-149-01-CSM

Hometown (currently reside): Bloomington, Indiana

Publications suggested by grantee to receive an ACS grant press release:

Hoosier Times

Indiana University Cancer Center Newsletter

Grantee willing to discuss your projects with the media? Yes

Grantee willing to discuss to speak at ACS sponsored events? Yes

Grantee willing to serve as an expert or as a member of a speaker's bureau? Yes

Other ways grantee would like to assist ACS:

Institution Publicity Contact:

Pamela Perry