EGRP Research Highlights

Epidemiology and Genetics Research Program

Web Site: epi.grants.cancer.gov

The Epidemiology and Genetics Research Program (EGRP) supports about 450 grants and cooperative agreements annually. Investigators from throughout the United States and internationally are funded to conduct population-based research to increase our understanding of cancer etiology and prevention. Some of their recent research findings are highlighted in the following pages. Also, visit EGRP's Web site to view a special section with highlights from many other studies: *epi.grants.cancer.gov*.

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Epidemiology and Genetics Research Program
Division of Cancer Control and Population Sciences
6130 Executive Boulevard, Room 5113, MSC 7395, Bethesda, MD 20892-7395
Telephone: 301-496-9600; Fax: 301-435-6609

National Institutes of Health
U.S. Department of Health and Human Services

Fall 2005

Physical Activity, Obesity, and Weight Gain Associated With Breast Cancer Survival

Michelle Holmes, M.D., Dr.P.H.

Two recent studies provide new information on physical activity, obesity, and weight gain in relation to breast cancer survival. Michelle Holmes, M.D., Dr.P.H., of Brigham and Women's Hospital and Harvard Medical School, and colleagues analyzed data from nearly 3,000 women participating in the Nurses' Health Study (NHS) who were diagnosed with stages I, II, and III breast cancer. Women with breast cancer who engaged in physical activity equivalent to walking 1 or more hours per week had better survival than those who exercised less than that or not at all. The benefit was particularly apparent for women with hormone-responsive tumors. Physical activity has been linked to lower levels of circulating ovarian hormones, which may explain the relationship between physical activity and breast cancer. The researchers concluded that women who follow the U.S. government's recommendations to exercise at moderate intensity for 30 or more minutes per day for 5 or more days per week may survive longer.

In other NHS research, Candyce Kroenke, Sc.D., also of Brigham and Women's Hospital and Harvard Medical School, and colleagues found that women who are overweight prior to breast cancer diagnosis, or who are lean but gain weight following diagnosis, are more likely to have their disease return or to die from it. This effect was particularly pronounced among women who had never smoked. This is the first study of obesity and breast cancer to separate smokers from nonsmokers. Maintaining a healthy weight is important to reduce the risk of breast cancer recurrence and death, reported the researchers. The study was based on data on 5,204 breast cancer patients collected over 24 years.

Candyce Kroenke, Sc.D.

The two studies were supported by EGRP grants to Graham Colditz, M.D., Dr.P.H. EGRP has supported the NHS since its establishment in 1973.

Holmes MD, Chen WY, Feskanich D, Kroenke CH, Colditz GA. Physical activity and survival after breast cancer diagnosis. JAMA 2005 May 25;293(20):2479-86.

Kroenke CH, Chen WY, Rosner B, Holmes MD. Weight, weight gain, and survival after breast cancer diagnosis. J Clin Oncol 2005 Mar 1;23(7):1370-8. Epub 2005 Jan 31.

DNA Repair Deficiency Associated With Breast Cancer Risk in Sister Pairs

David Kennedy, Ph.D.

Deficient DNA repair capacity may influence risk for breast cancer and may be a valuable *in vitro* biomarker to identify high-risk individuals, especially in breast cancer families, according to a study by David Kennedy, Ph.D., of Columbia University, and colleagues. The researchers analyzed DNA repair capacity in lymphoblastoid cells from sister pairs, comparing women diagnosed with breast cancer to their unaffected sisters. The cells were treated with a DNA-damaging carcinogen (benzo[a]pyrene diolepoxide), and those cells of sisters with breast cancer were 8.6 percent less effective than their sisters' cells in responding to the assault. Women who had the lowest levels of DNA repair capability had double the risk for breast cancer compared with women who had the highest capability. The largest differences were found between patients and controls younger than age 40. In addition, the relative risk of breast cancer was nearly 3 times greater between the groups with the most and the least DNA repair capabilities. The study population (158 case patients and 154 controls) was from the EGRP-funded Metropolitan New York Registry of Breast Cancer Families, for which Rubie Senie, Ph.D., is principal investigator.

Kennedy DO, Agrawal M, Shen J, Terry MB, Zhang FF, Senie RT, Motykiewicz G, Santella RM. DNA repair capacity of lymphoblastoid cell lines from sisters discordant for breast cancer. J Natl Cancer Inst 2005 Jan 19;97(2):127-32.

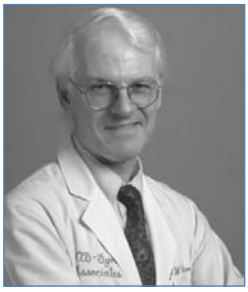
Sex Hormone-Binding Globulin Gene Variant Associated With Decreased Risk of Breast Cancer

Young Cui, M.D.

Sex hormone-binding globulin (SHBG) modulates the bioavailability of circulating sex hormones, regulating their effects on target tissues. The *SHBG* gene in breast cancer cells inhibits estradiol-induced cell proliferation and thus may influence breast cancer risk. Young Cui, M.D., of Vanderbilt University School of Medicine, and colleagues examined the association between a common functional polymorphism of the *SHBG* gene (*Asp327Asn*) and risk of breast cancer in a large population-based case-control study in Shanghai, China. They found the variant *Asn* allele associated with elevated plasma *SHBG* levels and decreased risk of breast cancer in postmenopausal women (27% reduction in risk). Furthermore, the allele's protective effect was much stronger among postmenopausal women with low adiposity (more than 50% reduction in risk). The inverse association between the allele and breast cancer risk also was stronger for estrogen receptor-positive cancer than for estrogen receptor-negative cancer. The study included 1,106 cases and 1,180 controls. It was funded by EGRP grants to Wei Zheng, Ph.D.

Cui Y, Shu XO, Cai Q, Jin F, Cheng JR, Cai H, Gao YT, Zheng W. Association of breast cancer risk with a common functional polymorphism (*Asp327Asn*) in the sex hormone-binding globulin gene. Cancer Epidemiol Biomarkers Prev 2005 May;14(5):1096-101.

Predictors of Natural Immunity to Ovarian Cancer Identified



Daniel Cramer, M.D., Sc.D.

MUC1 is a high molecular weight protein that is expressed in different forms by both healthy and cancerous cells. When expressed by cancerous cells, MUC1 stimulates the production of antibodies. In a case-control study of ovarian cancer, Daniel Cramer, M.D., Sc.D., of Brigham and Women's Hospital, and colleagues measured anti-MUC1 antibodies in 705 women who did not have ovarian cancer, identified events that predicted antibody production, and estimated risk for the cancer by comparing profiles of the events that generated antibodies in the women with similar events in 668 women with ovarian cancer. Factors that predicted antibody production included use of oral contraceptives and intrauterine devices, breast mastitis, bone fracture or osteoporosis, pelvic surgeries, nonuse of talc in genital hygiene, and being a current smoker. Women who had two factors leading to elevated MUC1 antibody levels were about 30 percent less likely to develop ovarian cancer than women with none or only one MUC1 antibody-promoting event. Women with five of the factors had about a 70 percent reduction in risk compared with women with none or one of them. Besides presenting a new model to explain risk factors for ovarian cancer, the researchers speculated that the findings could lead to development of preventive vaccines for ovarian and perhaps other cancers that express MUC1. The study was partly funded by an EGRP grant to Dr. Cramer.

Cramer DW, Titus-Ernstoff L, McKolanis JR, Welch WR, Vitonis AF, Berkowitz RS, Finn OJ. Conditions associated with antibodies against the tumor-associated antigen MUC1 and their relationship to risk for ovarian cancer. Cancer Epidemiol Biomarkers Prev 2005 May; 4(5):1125-31.

HPV Reactivated in HIV-Positive Women

For the first time, researchers have found strong evidence that the virus that causes cervical cancer, human papillomavirus (HPV), can be reactivated after being undetectable, which is especially likely in women with impaired immunity due to HIV infection as well as other causes. These findings have major implications for HIV-positive women and others with poor immune status, report Howard Strickler, Ph.D., of Albert Einstein College of Medicine of Yeshiva University, and colleagues. The study is based on data from the Women's Interagency HIV Study (WIHS), a prospective

Howard Strickler, Ph.D.

cohort of more than 2,500 HIV-positive and HIV-negative women. Their data suggest that undetectable HPV infections become active much more frequently in HIV-positive women, which may help explain the high rates of HPV infection in these women. For HIV-positive women, the researchers found that CD4+ cell count in combination with HIV RNA levels appeared to have a significant association with incident detection of HPV, some of the association possibly reflecting HPV reactivation in sexually inactive women.

Although weakened immune status due to HIV had a major effect on allowing HPV infections to develop, HIV had a relatively modest effect on HPV persistence, a necessity for cervical cancer to occur. This finding may help explain why cervical cancer rates have not reached more epidemic proportions in HIV-positive women. The research was partly funded by an EGRP grant to Dr. Strickler.

Strickler HD, Burk RD, Fazzari M, Anastos K, Minkoff H, Massad LS, Hall C, Bacon M, Levine AM, Watts DH, Silverberg MJ, Xue X, Schlecht NF, Melnick S, Palefsky JM. Natural history and possible reactivation of human papillomavirus in human immunodeficiency virus-positive women. J Natl Cancer Inst 2005 Apr 20;97(8):577-86.

Improved Method Demonstrated To Detect HNPCC Mutations

Graham Casey, Ph.D.

Several genes involved in DNA mismatch repair have been implicated in hereditary nonpolyposis colorectal cancer (HNPCC). Detection of mutations in these genes is crucial for recommending appropriate genetic counseling, screening, and surveillance. Graham Casey, Ph.D., of The Cleveland Clinic, and other investigators of the Colon Cancer Family Registry (CFR), compared the ability of conversion analysis with conventional DNA sequencing to detect heterogeneous germline mutations in mismatch repair genes *MHL1* and *MSH2* in HNPCC patients. Using conventional sequencing, normal copies of genes can sometimes mask mutations in the other allele.

Conversion analysis overcomes this weakness by separating pairs of chromosomes prior to analysis, through generation of human/mouse somatic cell hybrids. Results of this study demonstrated that conversion analysis provided a 33 percent improvement in detection of mismatch repair mutations in 89 colorectal cancer cases and a 56 percent increase in the diagnostic yield of genetic testing, compared with conventional sequencing. The Colon CFR is an EGRP-funded research resource.

Casey G, Lindor NM, Papadopoulos N, Thibodeau SN, Moskow J, Steelman S, Buzin CH, Sommer SS, Collins CE, Butz M, Aronson M, Gallinger S, Barker MA, Young JP, Jass JR, Hopper JL, Diep A, Bapat B, Salem M, Seminara D, Haile R; Colon Cancer Family Registry. Conversion analysis for mutation detection in *MLH1* and *MSH2* in patients with colorectal cancer. JAMA 2005 Feb 16;293(7):799-809.

New, More Accurate Information Available on Risk of Hereditary Colorectal Cancer

Noralane Lindor, M.D.

Noralane Lindor, M.D., of the Mayo Foundation, and colleagues studied individuals with a family pedigree suggestive of hereditary nonpolyposis colorectal cancer (HNPCC) but who lacked the characteristic DNA mismatch repair gene defect (*MMR*). They found that families without the DNA defect had a lower risk of colorectal cancer, were diagnosed at a later age, and had a lower incidence of other cancers associated with HNPCC than did families with the defect. About 60 percent of families that meet criteria for a certain type of HNPCC, Amsterdam-I (AC-I), have an abnormality in a DNA *MMR* gene. Cancer incidence in AC-I families with *MMR* gene mutations is high, but the incidence for individuals in AC-I families without evidence of an *MMR* defect has been unknown. In counseling families with AC-I, clinicians now can provide more accurate and lower risk information using these new data in combination with the specific family history, report the researchers. The study included 161 families who met the AC-I criteria; most of the families were from the EGRP-funded Colon Cancer Family Registry (CFR). The research was funded by EGRP

grants supporting the Registry, for which Dr. Lindor is one of the principal investigators.

Lindor NM, Rabe K, Petersen GM, Haile R, Casey G, Baron J, Gallinger S, Bapat B, Aronson M, Hopper J, Jass J, LeMarchand L, Grove J, Potter J, Newcomb P, Terdiman JP, Conrad P, Moslein G, Goldberg R, Ziogas A, Anton-Culver H, de Andrade M, Siegmund K, Thibodeau SN, Boardman LA, Seminara D. Lower cancer incidence in Amsterdam-I criteria families without mismatch repair deficiency: familial colorectal cancer type X. JAMA 2005 Apr 27; 293(16):1979-85.

Vitamin B_6 Inversely Associated With Risk of Colorectal Cancer

Esther Wei, Sc..D.

Vitamin B_6 may be inversely associated with risk of colorectal cancer, according to findings by Esther Wei, Sc.D., of Brigham and Women's Hospital and Harvard University, and colleagues. The main circulating form of vitamin B_6, pyridoxal 5'-phosphate (PLP), is critical to DNA synthesis and methylation, processes potentially involved in carcinogenesis. The researchers conducted a prospective, nested case-control study of 32,826 women participating in the Nurses' Health Study (NHS) to evaluate the association of PLP levels with colorectal cancer risk. Women in the highest quartile of plasma PLP concentration had a 44 percent lower risk of colorectal cancer compared with women in the lowest quartile. The association between PLP concentration and colon cancer was statistically significant (58% decreased risk). Moreover, both associations were statistically significant and stronger after controlling for folate, multivitamin, and methionine intake. The research was funded partly by EGRP grants to Graham Colditz, M.D., Ph. D., Susan Hankinson, Sc.D., and Meir Stampfer, M.D., Dr.P.H.

Wei EK, Giovannucci E, Selhub J, Fuchs CS, Hankinson SE, Ma J. Plasma vitamin B_6 and the risk of colorectal cancer and adenoma in women. J Natl Cancer Inst 2005 May 4;97(9):684-92.

International Consortium Conducts Large-Scale, Combined Genomewide Linkage Scan for Prostate Cancer-Susceptibility Genes

Jianfeng Xu, M.D., Dr.P.H.

Genomewide screens have been performed in more than a dozen independent studies, but few chromosomal regions have been consistently identified as regions of interest. A major difficulty is genetic heterogeneity, possibly due to multiple, incompletely penetrant prostate cancer-susceptibility genes.

Jianfeng Xu, M.D., Dr.P.H., of Wake Forest University, and other members of the International Consortium for Prostate Cancer Genetics (ICPCG) explored two approaches to overcome this difficulty. The Consortium combined linkage data from 1,233 families to increase the statistical power for detecting linkage, and identified five regions with "suggestive" linkage. It also focused on subsets of families that are more likely to segregate highly penetrant mutations, including families with large numbers of affected individuals or early age at diagnosis.

Stronger evidence of linkage in several regions was identified, including a "significant" linkage at 22q12, with an LOD score of 3.57 and five suggestive linkages (1q25, 8q13, 13q14, 16p13, and 17q21) in 269 families with at least five affected members. Additional suggestive linkages (3p24, 5q35, 11q22, and Xq12) were found in 606 families with mean age at diagnosis of ≤ 65 years. A conservative interpretation of these results would be that if major prostate cancer-susceptibility genes do exist, they are most likely located in the regions generating suggestive or significant linkage signals in this large study, report the researchers. An EGRP grant for the ICPCG, with William Isaacs, Ph.D., of Johns Hopkins University, as principal investigator, supported the research.

Xu J, Dimitrov L, Chang BL, Adams TS, Turner AR, Meyers DA, Eeles RA, Easton DF, Foulkes WD, Simard J, Giles GG, Hopper JL, Mahle L, Moller P, Bishop T, Evans C, Edwards S, Meitz J, Bullock S, Hope Q, Hsieh CL, Halpern J, Balise RN, Oakley-Girvan I, Whittemore AS, Ewing CM, Gielzak M, Isaacs SD, Walsh PC, Wiley KE, Isaacs WB, Thibodeau SN, McDonnell SK, Cunningham JM, Zarfas KE, Hebbring S, Schaid DJ, Friedrichsen DM, Deutsch K, Kolb S, Badzioch M, Jarvik GP, Janer M, Hood L, Ostrander EA, Stanford JL, Lange EM, Beebe-Dimmer JL, Mohai CE, Cooney KA, Ikonen T, Baffoe-Bonnie A, Fredriksson H, Matikainen MP, Tammela TL, Bailey-Wilson J, Schleutker J, Maier C, Herkommer K, Hoegel JJ, Vogel W, Paiss T, Wiklund F, Emanuelsson M, Stenman E, Jonsson BA, Gronberg H, Camp NJ, Farnham J, Cannon-Albright LA, Seminara D; ACTANE Consortium. A combined genomewide linkage scan of 1,233 families for prostate cancer-susceptibility genes conducted by the international consortium for prostate cancer genetics. Am J Hum Genet 2005 Aug;77(2):219-29. Epub 2005 June 29.

Cytochrome P450 Polymorphism Associated With Risk of Prostate Cancer

Angie Stone, B.S.

Members of the cytochrome P450 3A subfamily of enzymes are involved in steroid hormone metabolism. A study by Angie Stone, B.S., of the National Center for Toxicological Research, and colleagues investigated the association between the *CYP3A43*3* genotype and risk of prostate cancer in African Americans and Caucasians. Their findings suggest that the *CYP3A43 Pro^{340} Ala* polymorphism contributes to prostate cancer risk in African Americans. They found a 3-fold increased risk of prostate cancer among men with the *CYP3A43-Ala/Ala* genotype compared with men with the *CYP3A43-Pro/Pro* genotype when analyzing data on all study participants. The *CYP3A43-Ala/Ala* genotype polymorphism was more frequently found in African Americans than in Caucasians (45% versus 13%), and African Americans with the *CYP3A43-Ala/Ala* genotype had a 2.6-fold increased risk of prostate cancer. The study is one of the first case-control studies to associate polymorphisms in the *CYP3A43* gene with prostate cancer susceptibility. It included 124 African Americans and 358 Caucasians with prostate cancer and 167 African-American and 319 Caucasian controls. The research was partly supported by an EGRP grant to Nicholas Lang, M.D., of the Central Arkansas Veteran's Health Care System and Arkansas Cancer Research Center.

Stone A, Ratnasinghe LD, Emerson GL, Modali R, Lehman T, Runnells G, Carroll A, Carter W, Barnhart S, Rasheed AA, Greene G, Johnson DE, Ambrosone CB, Kadlubar FF, Lang NP. *CYP343 Pro^{340} Ala* polymorphism and prostate cancer risk in African Americans and Caucasians. Cancer Epidemiol Biomarkers Prev 2005 May;14(5):1257-61.

Statins Associated With Decreased Risk of Advanced Prostate Cancer

Elizabeth Platz, Sc.D., M.P.H.

The longer men take cholesterol-lowering drugs such as statins, the far less likely they are to develop advanced prostate cancer, according to findings by Elizabeth Platz, Sc.D., M.P.H., of Johns Hopkins University, and colleagues. The researchers tracked use of cholesterol-lowering drugs and diagnosis of prostate cancer among 34,428 men participating in the Health Professionals Follow-up Study (HPFS) and followed them for more than a decade. Men who used these medications had one-half the risk of advanced prostate cancer and one-third of the risk of metastatic or fatal prostate cancer, compared with men who did not use cholesterol lowering drugs. Risk of advanced prostate cancer fell with increasing duration of use of the drugs. Use of cholesterol-lowering drugs did not have any influence on prostate cancer confined within the organ. The researchers believe that most of the protective effect comes from statins, because by 2000 more than 90 percent of the men who reported using cholesterol-lowering drugs said that they were using statins. It is not known whether the apparent benefit of statins is due to their cholesterol-lowering effect or their other properties, such as anti-inflammatory activity or effects on post-translational modification of proteins. The research was partly funded by an EGRP grant to Edward Giovannuci, M.D., Sc.D., of Harvard School of Public Health. EGRP has supported the HPFS since its establishment in the early 1980s.

American Association for Cancer Research Annual Meeting, 2005;46:4374.

Western Diet Associated With Increased Risk of Non-Hodgkin's Lymphoma

Ellen Chang, Sc.D.

The incidence of non-Hodgkin's lymphoma (NHL) has increased rapidly worldwide in recent decades for reasons that are largely unknown. Ellen Chang, Sc.D., of the Karolinska Institutet, and colleagues examined the role of diet in the development of NHL in a population-based case-control study in Sweden. Higher intakes of dairy products and fried meat, especially red meat, were associated with increased risk of some NHL subtypes in both men and women. The odds ratio (OR) of NHL for individuals in the highest quartile of intake of dairy products to the lowest quartile was 1.5. The OR for the highest quartile of intake of fried red meat was 1.5. Higher intakes of fruits and vegetables were associated with decreased risk of NHL, particularly follicular lymphoma, among women (OR=0.3). The researchers concluded that spread of the Western diet, with its high intake of dairy products and cooked and processed meats and low intake of fruits and vegetables, could account for a moderate proportion of the worldwide increase in NHL incidence, and that dietary modifications could help prevent some of the more common subtypes of NHL. The study included 597 cases and 467 controls. The research was partly funded by an EGRP grant to Dr. Chang.

Chang ET, Smedby KE, Zhang SM, Hjalgrim H, Melbye M, Ost A, Glimelius B, Wolk A, Adami HO. Dietary factors and risk of non-Hodgkin lymphoma in men and women. Cancer Epidemiol Biomarkers Prev 2005 Feb;14(2):512-20.

Epidemiology Report Identifies Barriers, Gaps, and Opportunities in Tobacco, Diet/Energy Balance, and Genetic Research

The meeting summary for The 1st NCI Epidemiology Leadership Workshop: Tobacco, Diet/Energy Balance, and Genetic Research, sponsored by EGRP, is available at *epi.grants.cancer.gov/Conference*. Seasoned principal investigators funded through EGRP were asked to this by-invitation-only-meeting in September 2004 to identify barriers and gaps in cancer epidemiology and to advance solutions to the study of tobacco diet/energy balance, and genes.

The summary includes presentations on the state-of-the-science in these areas and on opportunities to explore new collaborations and reports from the four working groups: (1) Challenges to Diet/Energy Balance Epidemiology Research, (2) Haplotypes Versus Genotypes, (3) Design Issues and Strategies in the Study of Rare Cancer, and (4) Susceptibility in Tobacco Carcinogenesis: Genotypes Versus Phenotypes. The Web site also includes the speakers' presentations (PDF format).

Report Published on Cancer Risk Prediction Models

Cancer researchers, clinicians, and the public are increasingly interested in statistical models designed to predict the occurrence of cancer. As the number and sophistication of cancer risk prediction models have grown, so too, has interest in ensuring that they are appropriately applied, correctly developed, and rigorously evaluated. A report on an NCI-sponsored workshop, held in May 2004 and cosponsored by EGRP, focusing on statistical models for predicting a person's risk of developing cancer is now published. Research priorities and resources are identified in the areas of (1) revising existing breast cancer risk assessment models and developing new models, (2) encouraging the development of new risk models, (3) obtaining data to develop more accurate risk models, (4) supporting validation mechanisms and resources, (5) strengthening model development efforts and encouraging coordination, and (6) promoting effective cancer risk communication and decision-making.

Freedman AN, Seminara D, Gail MH, Hartge P, Colditz GA, Ballard-Barbash R, Pfeiffer RM. Cancer risk prediction models: a workshop on development, evaluation, and application. J Natl Cancer Inst 2005 May 18;97(10):715-23.

Epidemiology and Genetics Research Program (EGRP) Staff

- **Epidemiology and Genetics Research Program**
  Telephone: 301-496-9600; Fax: 301-435-6609
  Web site: *epi.grants.cancer.gov*

- **Edward Trapido, Sc.D., EGRP Associate Director**
  Linda Anderson, M.P.A., Communications Director
  Jay Choudhry, M.S., Program Director
  Barbara Guest, M.S.W., M.P.H., Program Analyst
  Shannon Lemrow, Ph.D., Program Director
  Julian Smith, Program Analyst

- **Mukesh Verma, Ph.D., Acting Chief, Analytic Epidemiology Research Branch (AERB)**
  Nancy Emenacker, Ph.D., M.Ed., R.D., Health Science Administrator
  Virginia (Ginny) Hartmuller, Ph.D., R.D., Program Director
  A.R. (Joe) Patel, Ph.D., Program Director
  Vaurice Starks, Program Director
  Carmina Valle, M.P.H., Program Analyst

- **Deborah Winn, Ph.D., Chief, Clinical and Genetic Epidemiology Research Branch (CGERB)**
  J. Fernando Arena, M.D., Ph.D., Program Director
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  Carol Kasten, M.D., Program Director
  Shannon Lynch, M.P.H., Career Research Training Award Fellow
  Isis Mikhail, M.D., M.P.H., Dr.P.H., Program Director
  Scott Rogers, M.P.H., Research Associate
  Daniela Seminara, Ph.D., M.P.H., Program Director

- **Intergovernmental Personnel Act Employees (Spending some time each month at EGRP)**
  Leslie Bernstein, Ph.D., University of Southern California/Norris Comprehensive Cancer Center
  Graham Colditz, M.D., Ph.D., Brigham and Women's Hospital and Harvard University
  Margaret Spitz, M.D., M.P.H., The University of Texas M.D. Anderson Cancer Center

Sources of Information on Grant Policies, Funding, and Training

- Our Division of Cancer Control and Population Sciences (DCCPS) Home page: *cancercontrol.cancer.gov* for grant policy alerts and information on funding opportunities.
- DCCPS Tobacco Control Research Branch: *cancercontrol.cancer.gov/tcrb*
- NCI's Division of Extramural Activities (DEA): *deainfo.nci.nih.gov*
- NCI's Research Resources (directory of more than 100 products and services): *resresources.nci.nih.gov*
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- NCI's cancer research training, career development, and education opportunities Web site: *cancertraining.nci.nih.gov;* EGRP's training Web site: *epi.grants.cancer.gov/training*
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