EGRP Research Highlights

Epidemiology and Genetics Research Program

Web Site: epi.grants.cancer.gov

The National Cancer Institute's (NCI) Epidemiology and Genetics Research Program (EGRP), in the Division of Cancer Control and Population Sciences (DCCPS), provides opportunities for investigators to conduct population-based research to increase our understanding of cancer etiology and prevention. EGRP is the largest funder of cancer epidemiology grants nationally and worldwide, supporting approximately 400 grants and cooperative agreements annually. The following pages contain summaries of publications featuring research funded in full or in part by EGRP. The featured research was nominated by extramural investigators and selected by EGRP Program Staff based on scientific merit, innovation, and/or potential public health impact. The names of the first authors and of the EGRP-supported Principal Investigators whose grants are credited in the published papers appear in boldface print. Please visit EGRP's Web site to view highlights from many other studies: *epi.grants.cancer.gov*.

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Model Developed for Predicting Risk for Bladder Cancer

Xifeng Wu, M.D., Ph.D.

Bladder cancer is the fourth most common cancer in U.S. men and the second most common urologic malignancy. Screening for bladder cancer in the general population is not recommended currently because there is no model by which risk can be assessed.

Xifeng Wu, M.D., Ph.D., of the University of Texas M.D. Anderson Cancer Center, and colleagues developed a model that incorporates both mutagen sensitivity and epidemiologic factors to predict bladder cancer risk. The model was developed based on data from 678 white bladder cancer patients and 678 controls. Risk factors incorporated into the model included pack-years smoked; exposures to diesel, aromatic amines, cleaning fluids, radioactive materials, and arsenic; and results of an in vitro assay to test the sensitivity of blood cells to the mutagens benzo[*a*]pyrene diol-epoxide (BPDE) and gamma-radiation as a measure of DNA repair capacity.

The model is easy to use, requiring an individual to answer only a few simple questions to project individual risk for the cancer and undergo a simple blood test. It also showed good discriminatory ability and excellent concordance in the internal validation based only on answers to exposure questions (area under the curve [AUC] = 0.70; 95% Confidence Interval [CI] = 0.67 to 0.73), and discrimination was improved by including the results of the mutagen sensitivity assay (AUC = 0.80; 95% CI = 0.72 to 0.82). This work describes the first risk-prediction model for bladder cancer and could prove highly useful for identifying high-risk populations.

This research was funded in part by an EGRP grant to **Dr. Wu.**

Wu X, Lin J, Grossman HB, Huang M, Gu J, Etzel CJ, Amos CI, Dinney CP, Spitz MR. Projecting individualized probabilities of developing bladder cancer in white individuals. J Clin Oncol. 2007 Nov 1;25(31):4974-81.

Hormone Levels and Mammographic Density Independently Linked to Breast Cancer Risk in Postmenopausal Women

Rulla Tamimi, Sc.D.

Circulating sex hormone levels and mammographic density are strongly and independently related to breast cancer risk in postmenopausal women, according to a study by **Rulla Tamimi, Sc.D., of Brigham and Women's Hospital and Harvard Medical School**, and colleagues.

The investigators examined plasma levels of estradiol, free estradiol, testosterone, and free testosterone as well as mammographic density in a cohort of 253 postmenopausal women with breast cancer and 520 healthy women from the Nurses' Health Study (NHS) who were not taking hormones.

Relative risk (RR) of breast cancer associated with mammographic density (RR for highest vs. lowest quartile = 3.8; 95% CI = 2.2 to 6.6; Ptrend < .001) changed little after adjusting for circulating estradiol (RR = 3.9; 95% CI = 2.2 to 6.9; Ptrend <.001) or circulating testosterone (RR = 4.1; 95% CI = 2.3 to 7.2; Ptrend < .001). Circulating levels of estradiol (RR = 2.4; 95% CI = 1.4 to 4.0) and of testosterone (RR = 2.0; 95% CI = 1.2 to 3.1) both were associated with breast cancer risk before and after adjustment for mammographic density.

In a joint analysis of mammographic density and plasma testosterone, the risk of breast cancer was greatest in the highest tertiles of both factors relative to the lowest tertiles (RR = 6.0; 95% CI = 2.6 to 14.0). A joint analysis of mammographic density and plasma estradiol revealed a similar pattern (RR = 4.1; 95% CI = 1.7 to 9.8). This is the first study to indicate that the mechanism by which mammographic density increases breast cancer risk is independent of circulating sex hormone levels. On their own, circulating levels of sex hormones were associated with a twofold increased risk of breast cancer, and mammographic density was associated with an approximately fourfold increased risk of breast cancer.

This research was funded in part by an EGRP grant to **Susan Hankinson, Sc.D., of Brigham and Women's Hospital and Harvard Medical School**.

Tamimi RM, Byrne C, Colditz GA, Hankinson SE. Endogenous hormone levels, mammographic density, and subsequent risk of breast cancer in postmenopausal women. J Natl Cancer Inst. 2007 Aug 1;99(15):1178-87.

Hispanic Breast Cancer Patients May Have Highest Prevalence of *BRCA1* Mutation

Esther John, Ph.D.

Hispanic breast cancer patients may have the highest prevalence of *BRCA1* mutations when compared to African American, Asian-American, and non-Hispanic white patients without reported Ashkenazi Jewish ancestry, according to a new study.

Esther John, Ph.D., of the Northern California Cancer Center, and colleagues examined the prevalence of *BRCA1* mutations in Hispanic, African-American, and Asian-American female breast cancer patients compared with non-Hispanic white patients with and without Ashkenazi Jewish ancestry. All patients in this population-based study of 3,181 women were diagnosed before age 65 and were enrolled at the Northern California Breast Cancer Family Registry during the period 1996–2005. The Registry is part of the Breast Cancer Family Registry (B-CFR), an EGRP-supported research resource.

Among patients without reported Ashkenazi Jewish ancestry, estimated *BRCA1* mutation prevalence was 3.5 percent for Hispanic breast cancer patients (95% CI = 2.1% to 5.8%), 2.2 percent for non-Hispanic white patients (95% CI = 0.7% to 6.9%), 1.3 percent for African-American patients (95% CI = 0.6% to 2.6%), and 0.5 percent for Asian-American patients (95% CI = 0.1% to 2.0%). Patients with reported Ashkenazi Jewish ancestry had a *BRCA1* mutation prevalence of 8.3 percent (95% CI = 3.1% to 20.1%).

Within each racial/ethnic group, prevalence estimates decreased with age at diagnosis and were higher among patients who reported a family history of breast or ovarian cancer. Among African-American women diagnosed before the age of 35 years, the estimated *BRCA1* mutation prevalence was particularly high, 16.7 percent (95% CI = 7.1% to 34.3%).

This research was funded by EGRP grants to **Dr. John; Frederick Li, M.D., of the Dana-Farber Cancer Institute; and Alice Whittemore, Ph.D., of the University of Southern California.**

Access EGRP's Web site for the B-CFR to learn about using this resource (*epi.grants.cancer.gov/CFR*).

John EM, Miron A, Gong G, Phipps AI, Felberg A, Li FP, West DW, Whittemore A. Prevalence of pathogenic *BRCA1* mutation carriers in 5 US racial/ethnic groups. JAMA. 2007 Dec 26;298(24):2869-911.

Radiotherapy Reduces Mortality in Patients With Recurrent Contralateral Breast Cancer

Mario Schootman, Ph.D.

Radiotherapy following breast-conserving surgery is the standard of care to reduce the risk of same-side recurrence of primary breast cancer in women. However, no studies have been conducted on its effect on mortality in women who subsequently develop a second primary or metachronous contralateral breast cancer (MCBC).

Using data from NCI's Surveillance, Epidemiology, and End Results (SEER) Program, **Mario Schootman, Ph.D., of Washington University School of Medicine,** and colleagues retrospectively investigated the impact of radiotherapy on cause-specific and all-cause mortality in women who were treated surgically following the diagnosis of MCBC.

The study's analysis was based on data from 1,083 women 40 to 69 years of age diagnosed with stage 0 to III second primary contralateral breast cancer who were identified from SEER data for the period 1985 to 2000 and who subsequently were treated with breast conserving surgery. Based on misclassification-corrected analyses, 43.2 percent of 1,083 women with MCBC did not receive radiotherapy after breast conserving surgery.

The cause-specific and all-cause mortalities were compared in proportional hazard models. The investigators

used the method of propensity scores to negate the problem of confounding by indication and adjusted for misclassification of radiotherapy use. After adjustment, the women who had not received radiotherapy had 2.2 times greater risk of cause-specific and 1.7 times greater risk of all-cause mortality.

Based on these findings, the investigators recommended that discussions about the benefits and side effects of radiotherapy be held with patients being treated with breast conserving surgery for stage 0 to III primary MCBC.

This research was funded in part by an EGRP grant to **Dr. Schootman.**

Schootman M, Jeffe DB, Gillanders WE, Yan Y, Aft R. The effects of radiotherapy for the treatment of contralateral breast cancer. Breast Cancer Res Treat. 2007 May;103(1):77-83. Epub 2006 Oct 11.

Common Genetic Variation in *CYP17* Not Associated With Increased Risk of Breast and Prostate Cancer


V. Wendy Setiawan, Ph.D.

The *CYP17* gene is involved in the synthesis of steroid hormones, and it is thought that it may be related to risk of developing breast and prostate cancer. Although steroid hormones play an important role in breast and prostate carcinogenesis, genetic variation in *CYP17* has been associated only inconsistently with risk of the cancers.

To further analyze this association, **V. Wendy Setiawan, Ph.D., of the University of Southern California**, and colleagues participating in the Breast and Prostate Cancer Cohort Consortium (BPC3), characterized genetic variation in *CYP17* by sequencing its coding region and using a haplotype-based analysis to characterize common patterns of variation across the entire *CYP17* locus in five racial/ethnic populations.

Nine haplotype-tagged single nucleotide polymorphisms (SNPs) from areas of strong linkage disequilibrium were genotyped in 8,138 prostate cancer cases and 9,033 controls, and 5,333 breast cancer cases and 7,069 controls, from BPC3. Two SNPs, rs2486758 and rs6892, had a borderline significant association with prostate cancer. Two haplotypes and two SNPs also tagged these haplotypes, rs4919687 and rs4919682, and were marginally significantly associated with breast cancer. Because no common variation at *CYP17* was associated with levels of circulating hormones, further research is needed to characterize functional SNPs.

"Our analysis provides evidence against a strong main effect between the overall risk of breast and prostate cancer and circulating sex steroid hormone levels and variants in *CYP17* that are common among whites [of European ancestry]," concluded the investigators. "Weak associations between several *CYP17* SNPs and the cancers should be evaluated in future studies," they added.

This study was funded in part by EGRP grants to **David Hunter, M.D., Sc.D., of Harvard School of Public Health; Michael Thun, M.D., of the American Cancer Society; Elio Riboli, M.D., M.Sc., of the Imperial College, London; and Brian Henderson, M.D., of the University of Southern California. The Division of Cancer Epidemiology and Genetics, which is the intramural epidemiology component of NCI, also is part of BPC3 and participated in this research.**

With support from EGRP, the BPC3 pools data and biospecimens from 10 large prospective cohorts to conduct research on gene-environment interactions in the etiology of breast and prostate cancer. Access EGRP's Web site to learn more about the BPC3 (*epi.grants.cancer.gov/BPC3*).

Setiawan VW, Schumacher FR, Haiman CA, Stram DO, Albanes D, Altshuler D, Berglund G, Buring J, Calle EE, Clavel-Chapelon F, Cox DG, Gaziano JM, Hankinson SE, Hayes RB, Henderson BE, Hirschhorn J, Hoover R, Hunter DJ, Kaaks R, Kolonel LN, Kraft P, Ma J, Le Marchand L, Linseisen J, Lund E, Navarro C, Overvad K, Palli D, Peeters PH, Pike MC, Riboli E, Stampfer MJ, Thun MJ, Travis R, Trichopoulos D, Yeager M, Ziegler RG, Spencer Feigelson H, Chanock SJ. *CYP17* genetic variation and risk of breast and prostate cancer from the National Cancer Institute Breast and Prostate Cancer Cohort Consortium (BPC3). Cancer Epidemiol Biomarkers Prev. 2007 Nov;16(11):2237-46.

Ovarian Cancer Risk May Vary With Hormone Therapy

Mary Anne Rossing, Ph.D., D.V.M.

Few studies have examined whether ovarian cancer risk varies according to the recency and duration of the specific menopausal hormone therapy (HT) used. A study by **Mary Anne Rossing, Ph.D., D.V.M., of Fred Hutchinson Cancer Research Center,** and colleagues assessed the risk of ovarian cancer among users of unopposed menopausal estrogen (ET) and combined estrogen/progestogen therapy (EPT).

In a population-based study in Washington State, the investigators interviewed 812 women diagnosed with ovarian cancer between 2002 and 2005 and 1,313 controls about HT use and other characteristics. Women who used a single form of HT (ET or EPT) were compared with women who never used HT. Women who had used only ET had a slightly higher increased risk, and women who had used only EPT had a slightly reduced risk of ovarian cancer. Among current users, the increased risk among women who used ET for 5 years or more remained evident (OR = 1.6; 95% CI = 1.1 to 2.5). Among former ET users, a 5 years or more duration of use was associated with increased risk primarily among women who last used ET relatively recently. The OR was 1.8 (95% CI = 0.8 to 3.7) among women with 5 or more years of use who had stopped less than 3 years earlier, and 1.2 (95% CI = 0.6 to 2.7) among women who had used ET for a similar duration but had stopped 3 or more years earlier. In contrast, no increase in risk was found among women who used only EPT, regardless of duration. Compared with women who never used HT, current EPT users had an OR of 1.1 (95% CI = 0.8 to 1.5), and risk declined with increasing time since stoppage. Long-term ET use may be associated with an increased ovarian cancer risk that wanes after use ceases. No similar increased risk was observed with EPT use, and a reduction in risk became increasingly evident as time after stoppage increased. If replicated, these results may have public health implications and influence future chemoprevention strategies.

This research was funded by an EGRP grant to **Dr. Rossing.**

Rossing MA, Cushing-Haugen KL, Wicklund KG, Doherty JA, Weiss NS. Menopausal hormone therapy and risk of epithelial ovarian cancer. Cancer Epidemiol Biomarkers Prev. 2007 Dec;16(12):2548-56.

Endogenous Estrogens May Increase Colorectal Cancer Risk

Marc Gunter, Ph.D.

Despite evidence strongly suggesting that obesity and hyperinsulinemia are risk factors for colorectal cancer, few prospective studies have directly measured the effects of insulin or insulin-like growth factor-1 (IGF-1) on risk. Additionally, estrogen may affect colorectal cancer risk, and studies suggest that use of oral hormone replacement therapy is associated with decreased risk. However, no studies have evaluated the association between endogenous estradiol and risk of colorectal cancer.

Marc Gunter, Ph.D., of Albert Einstein College of Medicine, and colleagues conducted a case-cohort investigation of colorectal cancer among nondiabetic women enrolled in the Women's Health Initiative Observational Study. The investigators collected fasting baseline serum specimens from 438 women with colorectal cancer and 816 women in a random subcohort and measured insulin, glucose, total IGF-1, free IGF-1, IGF binding protein-3 (IGFBP-3), and estradiol. Insulin, waist circumference, and free IGF-1 were each positively associated with colorectal cancer incidence, but these associations became nonsignificant when adjusted for one another.

In contrast, endogenous estradiol levels were positively associated with risk of colorectal cancer (hazard ratio for high versus low levels = 1.53; 95% CI = 1.05 to 2.22), even after controlling for insulin, free IGF-1, and waist circumference. The authors suggest that these data indicate two independent biological pathways affecting colorectal cancer risk: the first involves endogenous estradiol, and the second is associated broadly with obesity, hyperinsulinemia, and free IGF-1.

This research was funded by an EGRP grant to **Howard Strickler, M.D., M.P.H., of Albert Einstein College of Medicine.**

Gunter MJ, Hoover DR, Yu H, Wassertheil-Smoller S, Rohan TE, Manson JE, Howard BV, Wylie-Rosett J, Anderson GL, Ho GY, Kaplan RC, Li J, Xue X, Harris TG, Burk RD, Strickler HD. Insulin, insulin-like growth factor-I, endogenous estradiol, and risk of colorectal cancer in postmenopausal women. Cancer Res. 2008 Jan 1;68(1):329-37.

Putative Tumor Suppressor Gene Associated With Aggressive Prostate Cancer

David Duggan, Ph.D.

S. Lilly Zheng, Ph.D.

A variant of a tumor suppressor gene, *DAB21P,* may be associated with an increased risk of aggressive prostate cancer, according to a study by **David Duggan, Ph.D., of the Translational Genomics Research Institute, S. Lilly Zheng, Ph.D., of Wake Forest University**, and colleagues. Consistent findings that prostate cancer has a genetic component have led investigators to suspect there are genetic variants that predispose some men to develop the disease. The investigative team combined the results from two genome-wide association studies (GWAS) on aggressive prostate cancer to identify single nucleotide polymorphisms (SNPs) that may be associated with aggressive disease.

In the first phase of the study, the investigators performed an exploratory GWAS scan in 498 men with aggressive prostate cancer and 494 men who were cancer-free from a population-based study in Sweden. The results of the scan were combined with data on 737 patients with aggressive prostate cancer and 1,105 controls from a GWAS performed by NCI's Cancer Genetic Markers of Susceptibility (CGEMS) project. By combining results of these two studies, the investigators identified 11 SNPs that had statistically significant associations with aggressive prostate cancer, based on a threshold of $P < 0.01$, seven of which had consistent risk associations in both studies.

These exploratory findings then were confirmed in two independent populations: one cohort consisting of 1,032 European Americans and 571 controls, and the other cohort of 210 African Americans and 346 controls. The investigators found a statistically significant association with aggressive prostate cancer for the SNP rs1571801 at 9q33 in the European Americans ($P = 0.004$) and African Americans ($P = 0.02$). This SNP, located in the *DAB2IP* gene, was associated with aggressive prostate cancer among men of European and African-American descent. *DAB2IP* encodes a novel Ras GTPase-activating protein and putative prostate tumor suppressor. Reduced *DAB2IP* expression has been shown to correlate with increased expression of *EZH2*, which is one of the strongest markers for aggressive prostate cancer.

This research was funded in part by EGRP grants to **William Isaacs, Ph.D., of The Johns Hopkins Medical Institutions, and Jianfeng Xu , M.D., Dr.P.H., of Wake Forest University School of Medicine.**

CGEMS is an NCI initiative to identify common genetic variants associated with risk for prostate and breast cancer. It is coordinated through the NCI's Division of Cancer Epidemiology and Genetics, Core Genotyping Facility, and Office of Cancer Genomics.

Duggan D, Zheng SL, Knowlton M, Benitez D, Dimitrov L, Wiklund F, Robbins C, Isaacs SD, Cheng Y, Li G, Sun J, Chang BL, Marovich L, Wiley KE, Bälter K, Stattin P, Adami HO, Gielzak M, Yan G, Sauvageot J, Liu W, Kim JW, Bleecker ER, Meyers DA, Trock BJ, Partin AW, Walsh PC, Isaacs WB, Grönberg H, Xu J, Carpten JD. Two genome-wide association studies of aggressive prostate cancer implicate putative prostate tumor suppressor gene *DAB2IP*. J Natl Cancer Inst. 2007 Dec 19;99(24):1836-44. Epub 2007 Dec 11.

Alcohol and Tobacco Do Not Increase Risk in Patients With HPV16-Related Head and Neck Cancer

Kate Applebaum, Sc.D.

The effect of human papillomavirus type 16 (HPV16) infection on the association between smoking and drinking alcohol and risk of head and neck squamous cell carcinoma (HNSCC) is not fully understood. Research conducted by **Kate Applebaum, Sc.D., while a postdoctoral fellow at the Harvard School of Public Health,** and colleagues suggests that the etiology of HPV16-related HNSCC may be distinct from cancers of those sites attributed to tobacco or alcohol use. These findings of different patterns of risk for differing cancer sites have important implications for the development of targeted treatments.

The investigators conducted a case-control study of 485 patients in the Boston area who were diagnosed with

HNSCC between 1999 and 2003 and 549 control subjects who were cancer-free. The study groups were matched closely for age, gender, and town of residence. All participants completed questionnaires about smoking and alcohol consumption and received antibody tests to detect the presence of HPV16 in the blood.

Polytomous logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CI) for the association of HPV serology, alcohol, and tobacco use for oral, pharyngeal, and laryngeal cancer.

Although HPV16 serology was not a strong confounder for the association of HNSCC risk and smoking and alcohol, HPV16 seropositivity was a strong risk factor for HNSCC (OR = 4.5, 95% CI = 3.1 to 6.5). The strongest risk factors by tumor site were smoking for laryngeal cancer, alcohol consumption for oral cancer, and HPV16 for pharyngeal cancer.

The research was funded in part by EGRP grants to **Karl Kelsey, M.D., M.O.H., of Brown University.** Dr. Applebaum is now on the faculty at Boston University and is a Visiting Scientist at Harvard University.

Applebaum KM, Furniss CS, Zeka A, Posner MR, Smith JF, Bryan J, Eisen EA, Peters ES, McClean MD, Kelsey KT. Lack of association of alcohol and tobacco with HPV16-associated head and neck cancer. J Natl Cancer Inst. 2007 Dec 5;99(23):1801-10.

HPV Load May Predict Prevalence of Cervical Cancer

Patti Gravitt, Ph.D.

Melinda Butsch Kovacic, Ph.D., M.P.H.

Finding markers that identify which infections will progress to high-grade cervical intraepithelial neoplasia (≥CIN2) potentially could eliminate the need for many unnecessary gynecologic procedures for women who test positive for human papillomavirus (HPV) on Pap smears. Determining whether HPV viral load in cervical specimens is valuable as a marker of progression is an important research question.

Patti Gravitt, Ph.D., of The Johns Hopkins Bloomberg School of Public Health, and Melinda Butsch Kovacic, Ph.D., M.P.H., formerly of NCI, and colleagues analyzed HPV viral load and risk of prevalent and incident histologically confirmed ≥CIN2 by conducting a prospective, cross-sectional, population-based study of women infected with HPV in Costa Rica, where rates of cervical cancer historically are high.

The final analysis looked at 1,296 women with single HPV infections and 696 women with multiple HPV infections and measured viral load by oligonucleotide probe hybridization signal intensity. In followup over 7 years, investigators found that high viral load for most high-risk HPV genotypes is associated with prevalent ≥CIN2. However, only women infected with HPV16 alone (odds ratio = 27.2; 95% CI = 3.5 to 213.5) had a strong association between high viral load and incident ≥CIN2. Cumulative risk of diagnosis of the cancer was much higher for HPV16 (20.6%) compared to women with non-HPV16 carcinogenic types (5.1%) or other noncarcinogenic types (1.6%).

These findings support emerging evidence that HPV16 possesses unique properties compared to other carcinogenic HPV genotypes. In view of the fact that HPV16 accounts for almost 50 percent of all cervical cancers worldwide, viral load testing for this specific genotype only may be beneficial. The authors advise caution in applying their findings to clinical practice, in part because the complex research assays used to measure viral load are not yet available clinically.

The study was funded by an EGRP grant to **Robert Burk, M.D., of Albert Einstein College of Medicine.** Dr. Kovacic is now at Cincinnati Children's Hospital Medical Center.

Gravitt PE, Kovacic MB, Herrero R, Schiffman M, Bratti C, Hildesheim A, Morales J, Alfaro M, Sherman ME, Wacholder S, Rodriguez AC, Burk RD. High load for most high risk human papillomavirus genotypes is associated with prevalent cervical cancer precursors but only HPV16 load predicts the development of incident disease. Int J Cancer. 2007 Dec 15;121(12):2787-93.

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- NCI Division of Extramural Activities (DEA): *deainfo.nci.nih.gov*
- *Grants.gov* (central resource to find and apply for U.S. grants)
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  - NCI directory of more than 175 products: *resresources.nci.nih.gov*
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  - NIH Guide for Grants and Contracts: *grants.nih.gov/grants/guide/listserv.htm*
  - NIH Inside eRA for Partners (Electronic Research Administration or "The Commons") (occasional updates): *era.nih.gov/eranews*
  - NIH Extramural Nexus (bimonthly newsletter for grantees): *grants.nih.gov/grants/nexus.htm*
  - EGRP's Listserv (occasional Bulletins, News Flashes) contact: *kaeferc@mail.nih.gov*
- **Everything you wanted to know about the NCI Grants Process...but were afraid to ask** (2005). Access online at *www.cancer.gov/admin/gab* or order a print copy via NCI's online Publications Locator: *https://cissecure.nci.nih.gov/ncipubs*. **(The publication does not include information about NIH's mandatory transition to electronic submission of applications and the new form; see *era.nih.gov/ElectronicReceipt/index.htm*.)**

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