EGRP Research Highlights

Epidemiology and Genetics Research Program

Web Site: epi.grants.cancer.gov

The Epidemiology and Genetics Research Program (EGRP) supports approximately 400 grants and cooperative agreements annually. Investigators 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. Please visit EGRP's Web site to view a special section with highlights from many other studies: *epi.grants.cancer.gov*.

Contents

Breast Cancer

BPC3 Consortium Finds Polymorphisms in the Androgen Receptor Are Not Linked to Breast Cancer Risk 2

BRCA1 and *BRCA2* Mutation Frequencies and Their Association With Cancer 2

Cervical Cancer

Human Papillomavirus Type 16 and 18 Variants Show Race-Related Distribution and Persistence 3

Multiple-Type Human Papillomavirus Infection Increases Risk of Cervical Cancer 3

Colorectal Cancer

Colorectal Cancer Risk Associated Jointly With Smoking and NSAID Use 4

Model Predicts Germline Mutations and Risk of Cancer in the Lynch Syndrome 4

Liver Cancer

Prediagnostic Level of Serum Retinol Associated With Decreased Risk of Hepatocellular Carcinoma 5

Pancreatic Cancer

Vitamin D Intake Associated With a Lower Risk for Pancreatic Cancer in Two Cohort Studies 5

Recent-Onset Diabetes Mellitus May Be an Early Marker for Pancreatic Cancer 6

Prostate Cancer

LTA May Modify the Association Between NSAID Use and Decreased Risk of Advanced Prostate Cancer 6

Melanoma

High-Risk Melanoma Susceptibility Genes and Pancreatic Cancer, Neural System Tumors, and Uveal Melanoma Across GenoMEL 7

EGRP Staff List 8

Sources of Information on Grant Policies, Funding, and Training 8

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

Winter 2007

BPC3 Consortium Finds Polymorphisms in the Androgen Receptor Are Not Linked to Breast Cancer Risk


Brian Henderson, M.D.


David Hunter, M.D., Sc.D.


Elio Riboli, M.D., M.Sc.


Michael Thun, M.D.

Androgens may influence breast cancer risk through mechanisms including conversion to estradiol or by binding to the estrogen receptor and/or the androgen receptor (AR) in the breast. The AR is expressed in the normal breast as well as in breast cancer tumors, and both the expression and protein levels have been correlated with tumor invasiveness. To analyze whether polymorphisms in the AR gene are associated with breast cancer risk, **Brian Henderson, M.D., of the University of Southern California/Norris Comprehensive Cancer Center, David Hunter, M.D., Sc.D., of the Harvard School of Public Health, Elio Riboli, M.D., M.Sc., of the Imperial College, London, Michael Thun, M.D., of the American Cancer Society,** and colleagues participating in the EGRP-sponsored Breast and Prostate Cancer Cohort Consortium (BPC3) Study first determined the underlying genetic variation in the AR coding regions in a panel of 95 advanced breast cancer cases and genotyped markers in a panel of 349 healthy women. They identified linkage disequilibrium relationships across the gene and selected haplotype-tagged single nucleotide polymorphisms (htSNPs) that captured the common genetic variants across the locus. The htSNPs then were genotyped in nested breast cancer cases (5,603) and controls (7,480) from the Cancer Prevention Study II, European Prospective Investigation into Cancer and Nutrition, Multiethnic Cohort, Nurses' Health Study, and Women's Health Study cohorts. The authors found no association between any genetic variation in the AR gene and breast cancer risk. They concluded that, in postmenopausal Caucasian women, common polymorphisms in AR are not associated with breast cancer risk.

Cox DG, Blanche H, Pearce CL, Calle EE, Colditz GA, Pike MC, Albanes D, Allen NE, Amiano P, Berglund G, Boeing H, Buring J, Burtt N, Canzian F, Chanock S, Clavel-Chapelon F, Feigelson HS, Freedman M, Haiman CA, Hankinson SE, Henderson BE, Hoover R, Hunter DJ, Kaaks R, Kolonel L, Kraft P, LeMarchand L, Lund E, Palli D, Peeters PH, Riboli E, Stram DO, Thun M, Tjonneland A, Trichopoulos D, Yeager M; Breast and Prostate Cancer Cohort Consortium. A comprehensive analysis of the androgen receptor gene and risk of breast cancer: results from the National Cancer Institute Breast and Prostate Cancer Cohort Consortium (BPC3). Breast Cancer Res. 2006;8(5):R54.

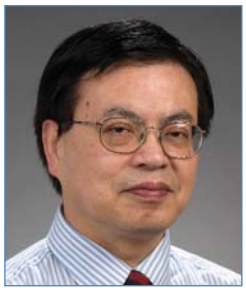
BRCA1 and *BRCA2* Mutation Frequencies and Their Association With Cancer


Harvey Risch, M.D., Ph.D.

The presence of *BRCA1* and *BRCA2* mutations in the general population and the link between these mutations and various types of cancers have not been well documented. A study by **Harvey A. Risch, M.D., Ph.D., of the Yale University School of Medicine**, and colleagues investigated the presence of *BRCA1* and *BRCA2* mutations in 1,171 unselected patients with newly diagnosed incident ovarian cancer in Ontario, Canada, with respect to cancers reported among their relatives. The patients were screened for germline mutations throughout the *BRCA1* and *BRCA2* genes. Higher risks for various cancers, including ovarian, female breast, and testicular cancer in the general Ontario population, were associated with carrying *BRCA1* mutations versus not carrying mutations (ovarian cancer relative risk (RR) = 21, 95% confidence interval (CI) = 12 to 36; female breast cancer RR = 11, 95% CI = 7.5 to 15; and testicular cancer RR = 17, 95% CI = 1.3 to 230). Similarly, higher risks were associated with carrying *BRCA2* mutations versus not carrying mutations, particularly for ovarian (RR = 7.0, 95% CI = 3.1 to 16), female and male breast (RR = 4.6, 95% CI = 2.7 to 7.8; and RR = 102, 95% CI = 9.9 to 1,050; respectively), and pancreatic (RR = 6.6, 95% CI = 1.9 to 23) cancers. Cancer risks differed according to a mutation's position on the gene. Estimated cumulative incidence to age 80 years among women carrying *BRCA1* mutations was 24% for ovarian cancer and 90% for breast cancer; in women carrying *BRCA2* mutations, the estimated cumulative incidence was 8.4% for ovarian cancer and 41% for breast cancer. For the general Ontario population, estimated carrier frequencies of *BRCA1* and *BRCA2* mutations were, respectively, 0.32% (95% CI = 0.23% to 0.45%) and 0.69% (95% CI = 0.43% to 1.10%). The researchers concluded that *BRCA1* and *BRCA2* mutations may be more frequent in general populations than previously thought and may be associated with various types of cancers. This research was supported by EGRP grants to **Dr. Risch and Steven Narod, M.D., Ph.D., University of Toronto.**

Risch HA, McLaughlin JR, Cole DE, Rosen B, Bradley L, Fan I, Tang J, Li S, Zhang S, Shaw PA, Narod SA. Population BRCAl and BRCA2 mutation frequencies and cancer penetrances: a kin-cohort study in Ontario, Canada. J Natl Cancer Inst. 2006 Dec 6;98(23):1694-706.

Human Papillomavirus Type 16 and 18 Variants Show Race-Related Distribution and Persistence



Long Fu Xi, M.D., Ph.D.

Persistent human papillomavirus (HPV) infection, particularly with HPV types 16 or 18, places women at increased risk for cervical cancer. HPV variants, which are viral isolates for any HPV type that differ by less than 2% in the L1 gene sequence, appear to segregate geographically. The persistence of these variants in certain geographic populations of infected individuals may be related to the racial composition of that population. **Long Fu Xi, M.D., Ph.D., of the University of Washington**, and colleagues studied 1,114 women in the United States participating in the Atypical Squamous Cells of Undetermined Significance/Low-Grade Squamous Intraepithelial Lesion Triage Study who were positive for HPV16 and/or HPV18 at enrollment and classified the HPV variants based on established HPV lineages. They found that 63.9% of women infected with HPV18 who self-reported as African American were infected with the HPV18 African variant (95% confidence interval (CI) = 53.5% to 73.4%); 54.2% of white women infected with HPV18 were infected with the European variant (95% CI = 46.3% to 61.9%). The likelihood of staying HPV18 positive was statistically significant and higher for African-American women if infected with the African variant compared to the European variant, and statistically significant and higher for white women if infected with the European variant compared to the African variant. The same pattern was found for HPV16 infection. This work suggests that HPV infection persists longer in a host whose race indicates an ancestral geographic distribution that once was shared with that of the infecting HPV variant. **This study was funded by an EGRP grant to Dr. Xi.**

Xi LF, Kiviat NB, Hildesheim A, Galloway DA, Wheeler CM, Ho J, Koutsky LA. Human papillomavirus type 16 and 18 variants: race-related distribution and persistence. J Natl Cancer Inst. 2006 Aug 2;98(15):1045-52.

Multiple-Type Human Papillomavirus Infection Increases Risk of Cervical Cancer

Helen Trottier, Ph.D.

Human papillomavirus (HPV) infection is one of the most common sexually transmitted diseases and plays the main causal role in cervical carcinogenesis. Certain HPV genotypes, such as HPV16, are associated with a high risk of cervical cancer, but little is known about the effects of infection with multiple HPV genotypes on cervical cancer. **Helen Trottier, Ph.D., and Eduardo Franco, Dr.P.H., of McGill University**, and colleagues used PCR to type HPV present in cervical specimens from 2,462 Brazilian women and assessed the relationship between infection with multiple HPV types and any-grade squamous intraepithelial lesions (SIL) and high-grade SIL (HSIL). Infection with multiple HPV types was associated positively with HSIL risk. Relative to women consistently negative for HPV infection, after a 1-year followup for HSIL, women infected with a single type of HPV had an odds ratio (OR) of 41.5, 95% confidence interval (CI) = 5.3 to 323.2; women infected with two to three types of HPV had an OR of 91.7, 95% CI = 11.6 to 728.1; and women infected with four to six types had an OR of 424.0, 95% CI = 31.8 to 5,651.8. The excess risk associated with multiple HPV-type infection persisted after excluding women infected with HPV16 or other high-risk HPV types, or for persistent infections, particularly for any-grade SIL. This work suggests that infection with multiple HPV types may act synergistically in cervical carcinogenesis, or that harboring multiple HPV types may be a marker for a decreased immune response to HPV and thus to greater risk. **This research is supported in part by an EGRP grant to Dr. Franco.**

Eduardo Franco, Dr.P.H.

Trottier H, Mahmud S, Costa MC, Sobrinho JP, Duarte-Franco E, Rohan TE, Ferenczy A, Villa LL, Franco EL. Human papillomavirus infections with multiple types and risk of cervical neoplasia. Cancer Epidemiol Biomarkers Prev. 2006 Jul;15(7):1274-80.

Colorectal Cancer Risk Associated Jointly With Smoking and NSAID Use

Victoria Chia, Ph.D.

Smoking has been associated with an increased risk of colorectal cancer, and nonsteroidal anti-inflammatory drugs (NSAIDs) have been associated with a reduced risk of colorectal cancer. In a population-based case-control study, **Victoria M. Chia, Ph.D., of the Fred Hutchinson Cancer Research Center and the University of Washington**, and colleagues evaluated the joint association between smoking and regular NSAID use with colorectal cancer risk and examined these associations stratified by tumor microsatellite instability (MSI high/low: MSI-H, MSI-L). They analyzed 1,792 incident colorectal cancer cases and 1,501 population controls in the Seattle area from 1998 to 2002, and assessed MSI in tumors of 1,202 cases. Individuals who had ever smoked had an increased risk of developing colorectal cancer. Individuals who were currently using NSAIDs had a 30% lower risk of developing colorectal cancer compared to non-NSAID users. The data also demonstrated that, relative to current NSAID users who never smoked, individuals who had both smoked for more than 40 years and had never used NSAIDs had the highest risk for colorectal cancer. Compared with nonsmokers, a greater number of tumors in smokers were classified as MSI-H than as MSI-L. NSAID use did not reduce the risk of MSI-H or MSI-L tumors in long-term smokers. Yet smokers who never used NSAIDs had a higher likelihood of having MSI-L tumors. The researchers concluded that there seems to be a synergistic inverse association that implies protection against colorectal cancer overall as a result of NSAID use and nonsmoking, but the risk of MSI-H colorectal cancer remains elevated among smokers even when they have a history of NSAID use. This research was supported in part by an EGRP grant to **John Potter, M.D., Ph.D., of the Fred Hutchinson Cancer Research Center and the University of Washington, and through cooperative agreements with members of the Colon Cancer Family Registry and Principal Investigators.**

Chia VM, Newcomb PA, Bigler J, Morimoto LM, Thibodeau SN, Potter JD. Risk of microsatellite-unstable colorectal cancer is associated jointly with smoking and nonsteroidal anti-inflammatory drug use. Cancer Res. 2006 Jul 1;66(13):6877-83.

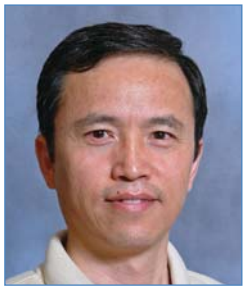
Model Predicts Germline Mutations and Risk of Cancer in the Lynch Syndrome

Sining Chen, Ph.D.

The Lynch Syndrome (hereditary nonpolyposis colorectal cancer, HNPCC), the most common familial colorectal cancer, can be caused by germline deleterious mutations of DNA mismatch repair (MMR) genes. **Sining Chen, Ph.D., of the Johns Hopkins Bloomberg School of Public Health,** and colleagues developed the MMRpro model to estimate the probability of an individual carrying a deleterious mutation in mismatch repair genes *MLH1, MSH2,* and *MSH6* and developing colorectal or endometrial cancer. The probability is assessed on the basis of a detailed family history of colorectal and endometrial cancer for an individual and his or her first- and second-degree relatives. To validate the MMRpro model, the model's predictions were compared with the results of highly sensitive germline mutation detection techniques for 279 individuals from 226 clinic-based families in the United States, Canada, and Australia (referred between 1993 and 2005). In this independent evaluation, MMRpro provided a concordance index of 0.83 (95% confidence interval (CI) = 0.78 to 0.88) and a ratio of observed-to-predicted cases of 0.94 (95% CI = 0.84 to 1.05), demonstrating that the model is more sensitive and more specific than current clinical guidelines for identifying individuals who may benefit from MMR germline testing. Importantly, this model can be used among individuals for whom tumor samples are not available or whose germline DNA tests find no mutation. Some patients who were in this study belong to EGRP's **Colon Cancer Family Registry**, an international research infrastructure for investigators interested in conducting population- and clinic-based interdisciplinary studies on the genetic and molecular epidemiology of colon cancer and its behavioral implications.

Chen S, Wang W, Lee S, Nafa K, Lee J, Romans K, Watson P, Gruber SB, Euhus D, Kinzler KW, Jass J, Gallinger S, Lindor NM, Casey G, Ellis N, Giardiello FM, Offit K, Parmigiani G; Colon Cancer Family Registry. Prediction of germline mutations and cancer risk in the Lynch syndrome. JAMA. 2006 Sep 27;296(12):1479-87.

Prediagnostic Level of Serum Retinol Associated With Decreased Risk of Hepatocellular Carcinoma



Jian-Min Yuan, M.D., Ph.D.

Retinol and its derivatives (retinoids) are antioxidants that promote cell differentiation and may protect against the development of hepatocellular carcinoma (HCC) by controlling hepatocellular differentiation and reducing inflammatory responses. Few prospective epidemiologic studies of serum retinol and other antioxidants in relation to HCC risk have been conducted, however. This study by **Jian-Min Yuan, M.D., Ph.D., of the University of Minnesota**, and colleagues examined the relationship between concentrations of antioxidant micronutrients in prediagnostic serum samples and the risk of developing HCC in 213 patients with HCC and 1,087 controls from a cohort of 18,244 men in Shanghai, China, who were monitored from 1986 through 2001. The micronutrients measured included retinol, specific carotenoids, tocopherols, and selenium. Odds ratios (ORs) and 95% confidence intervals (CIs) were estimated by quartile (Q) of serum micronutrient concentrations using logistic regression, with adjustments for smoking status, alcohol intake, history of physician-diagnosed hepatitis or liver cirrhosis, and seropositivity for hepatitis B surface antigen (HBsAg). The researchers found that higher prediagnostic serum levels of retinol were associated with a statistically significant reduced risk of developing HCC in these middle-aged or older Chinese men (Q2 versus Q1, OR= 0.37, CI = 0.22 to 0.61; Q3 versus Q1, OR = 0.30, CI = 0.17 to 0.50; Q4 versus Q1, OR = 0.13, CI = 0.06 to 0.26; $P_{trend} < .001$). The association between serum retinol levels and HCC risk was present in both chronic carriers and noncarriers of the hepatitis B virus. Statistically significant interaction regarding HCC risk between low retinol levels and HBsAg positivity also was found; HBsAg-positive men in the lowest tertile of retinol had a greater than 70-fold higher risk (OR = 72.7, CI = 31.6 to 167.4) of HCC than HBsAg-negative men in the highest tertile of retinol ($P_{interaction} = .018$). Given that HCC is highly fatal, these findings may have implications for clinical practice and prevention efforts. **This research was supported by EGRP grants to Dr. Yuan.**

Yuan JM, Gao YT, Ong CN, Ross RK, Yu MC. Prediagnostic level of serum retinol in relation to reduced risk of hepatocellular carcinoma. J Natl Cancer Inst. 2006 Apr 5;98(7):482-90.

Vitamin D Intake Associated With a Lower Risk for Pancreatic Cancer in Two Cohort Studies

Charles Fuchs, M.D., M.P.H.

Vitamin D and its analogs show strong antitumor effects in a variety of tissues, including the pancreas. A study by Halcyon Skinner, Ph.D., of Northwestern University, and colleagues looked at associations between dietary intake of vitamin D, calcium, and retinol and subsequent risk for pancreatic cancer in two large, EGRP-supported prospective cohort studies: the Health Professionals Follow-up Study, which includes 46,771 men ages 40 to 75 years as of 1986, and the Nurses' Health Study, which includes 75,427 women ages 38 to 65 years as of 1984. Researchers collected information on Vitamin D dietary intake, documented incident pancreatic cancer through the year 2000, and identified 365 pancreatic cancer cases. Compared with participants in the lowest category of total vitamin D intake (< 150 IU/d), those participants who consumed ≥ 300 IU/d decreased their risk for pancreatic cancer (relative risk = 0.59). Calcium and retinol intakes were found not to be associated with pancreatic cancer risk. The researchers concluded that higher intakes of vitamin D were associated with lower risks for pancreatic cancer in these two U.S. cohorts, suggesting a potential role for vitamin D in the pathogenesis and prevention of pancreatic cancer. This research was supported in part by EGRP grants to **Walter Willett, M.D., Dr.P.H., and Graham Colditz, M.D., Dr.P.H., both of Harvard University and Brigham and Women's Hospital; and Charles Fuchs, M.D., M.P.H. (pictured), of Harvard University, Brigham and Women's Hospital, and Dana-Farber Cancer Institute.**

Skinner HG, Michaud DS, Giovannucci E, Willett WC, Colditz GA, Fuchs CS. Vitamin D intake and the risk for pancreatic cancer in two cohort studies. Cancer Epidemiol Biomarkers Prev. 2006 Sep;15(9):1688-95.

Recent-Onset Diabetes Mellitus May Be an Early Marker for Pancreatic Cancer

Elizabeth Holly, Ph.D., M.P.H.

Diabetes has been hypothesized to be both a risk factor for and a consequence of pancreatic cancer. Patients with diabetes have been shown to have an approximately 2-fold risk of developing pancreatic cancer, and new-onset diabetes may be caused by pancreatic cancer. Furong Wang, M.D., of the University of California, San Francisco, and colleagues performed a population-based case-control study of pancreatic cancer in the San Francisco Bay area, involving 532 cases with newly diagnosed pancreatic cancer and 1,701 controls. They found that participants with pancreatic cancer were more likely to report a history of diabetes than controls (odds ratio = 1.5, 95% confidence interval (CI) = 1.1 to 2.1). Diabetics in the case group reported a shorter duration of diabetes and a larger proportion of insulin users. Risk for pancreatic cancer decreased as the duration of diabetes increased. There was no association with pancreatic cancer and insulin use for 5 or more years, but insulin use for less than 5 years was associated with a 6.8-fold increased risk for pancreatic cancer (95% CI = 3.7 to 12). The authors concluded that recent-onset diabetes may be a complication of or an early marker for pancreatic cancer. **This research was supported by EGRP grants to Elizabeth Holly, Ph.D., M.P.H., (pictured) of the University of California, San Francisco, and Stanford University.**

Wang F, Gupta S, Holly EA. Diabetes mellitus and pancreatic cancer in a population-based case-control study in the San Francisco Bay Area, California. Cancer Epidemiol Biomarkers Prev. 2006 Aug;15(8):1458-63.

LTA May Modify the Association Between NSAID Use and Decreased Risk of Advanced Prostate Cancer

Graham Casey, Ph.D.

Studies show that nonsteroidal anti-inflammatory drugs (NSAIDs) have protective effects against prostate cancer, but this may exist only among certain subgroups of men, such as those with particular variants in inflammatory response genes. Xin Liu, M.D., Ph.D., of the University of California, San Francisco, and colleagues conducted a case-control study (n = 1,012) of the association between NSAID use and more advanced prostate cancer and evaluated whether the association was modified by a functional polymorphism in the lymphotoxin alpha *(LTA)* gene (*LTA* C+80A, where the CC genotype results in higher *LTA* production). The *LTA* protein modulates the immune and inflammatory response to pathogens. The researchers found an inverse association between NSAID use and disease (odds ratio (OR) = 0.67, 95% confidence interval (CI) = 0.52 to 0.87), which was modified by the *LTA* C+80A variant (p for interaction = 0.03). In men with the CC genotype, the inverse association between NSAID use and prostate cancer was substantially stronger (OR = 0.43, 95% CI = 0.28 to 0.67). NSAID use was not found to be associated with disease for men without the CC genotype (p = 0.30). Similar associations were observed when dose/duration of NSAID use were studied. These results suggest that prostate cancer chemoprevention by NSAIDs may be most appropriate for men with the *LTA* +80CC genotype. This research was supported by EGRP grants to **Graham Casey, Ph.D., of The Cleveland Clinic Foundation, and John Witte, Ph.D., of the University of California, San Francisco (both pictured).**

John Witte, Ph.D.

Liu X, Plummer SJ, Nock NL, Casey G, Witte JS. Nonsteroidal antiinflammatory drugs and decreased risk of advanced prostate cancer: modification by lymphotoxin alpha. Am J Epidemiol. 2006 Nov 15;164(10):984-9. Epub 2006 Aug 24.

High-Risk Melanoma Susceptibility Genes and Pancreatic Cancer, Neural System Tumors, and Uveal Melanoma Across GenoMEL

Alisa Goldstein, Ph.D.

David Elder, M.B., Ch.B.

The Melanoma Genetics Consortium (GenoMEL) is an international consortium of familial melanoma research groups from North America, Europe, Asia, and Australia. This study, by **Alisa Goldstein, Ph.D., of the National Cancer Institute, David Elder, M.B., Ch.B., of the University of Pennsylvania, Julia Newton Bishop, M.D., of the University of Leeds, UK,** and colleagues used the largest familial melanoma sample currently available, taken from across 17 GenoMEL centers, to assess the high-risk melanoma susceptibility genes *CDKN2A/alternative reading frames* (*ARF,* which encodes *p16* and *p14ARF*) and *CDK4,* and their relationship with pancreatic cancer, neural system tumors, and uveal melanoma. The study included 2,137 cutaneous malignant melanoma patients from 466 melanoma-prone families with at least three melanoma patients per family. Forty-one percent ($n = 190$) of families had mutations in one of three known high-risk melanoma susceptibility genes, most of which involved *p16* ($n = 178$). There were similar frequencies (2–3%) in mutations in *CDK4* ($n = 5$) and *p14ARF* ($n = 7$). The researchers found a strong association between prostate cancer and *CDKN2A* mutations ($P < 0.0001$), which differed by mutation. The group found

Julia Newton Bishop, M.D.

little evidence of an association between *CDKN2A* mutations and neural system tumors or uveal melanoma and only a marginally significant association between neural system tumors and *ARF* ($P = 0.05$). Researchers also found that the proportion of families with the most frequent founder mutations differed by locale, with similarities between Sweden and the Netherlands; between France, Spain, and Italy; and between the United Kingdom and Australia ($P = 0.0009$). This GenoMEL study provides the most extensive characterization to date of mutations in high-risk melanoma susceptibility genes in families with three or more melanoma patients. This research was supported in part by EGRP grants to **Dr. Elder, Lisa Cannon Albright, Ph.D., of the University of Utah, and Nicholas Hayward, Ph.D., of the Queensland Institute of Medical Research.**

Goldstein AM, Chan M, Harland M, Gillanders EM, Hayward NK, Avril MF, Azizi E, Bianchi-Scarra G, Bishop DT, Bressac-de Paillerets B, Bruno W, Calista D, Cannon Albright LA, Demenais F, Elder DE, Ghiorzo P, Gruis NA, Hansson J, Hogg D, Holland EA, Kanetsky PA, Kefford RF, Landi MT, Lang J, Leachman SA, Mackie RM, Magnusson V, Mann GJ, Niendorf K, Newton Bishop J, Palmer JM, Puig S, Puig-Butille JA, de Snoo FA, Stark M, Tsao H, Tucker MA, Whitaker L, Yakobson E; Melanoma Genetics Consortium (GenoMEL). High-risk melanoma susceptibility genes and pancreatic cancer, neural system tumors, and uveal melanoma across GenoMEL. Cancer Res. 2006 Oct 15;66(20):9818-28.

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*
Deborah Winn, Ph.D., EGRP Acting Associate Director

Office of the Associate Director

Stephanie Eber, M.P.H., Associate Communications Coordinator
Diane Horn-Cruder, B.A., Program Analyst
Shannon Lemrow, Ph.D., Program Director

Analytic Epidemiology Research Branch (AERB)

Mukesh Verma, Ph.D., Acting Chief
Virginia (Ginny) Hartmuller, Ph.D., R.D., Program Director
Chinonye (Nonye) Harvey, M.P.H., Program Analyst
Shannon Lynch, M.P.H., Program Analyst
Britt Reid, D.D.S., Ph.D., Program Director
Sheri Dixon Schully, Ph.D., Program Director
Vaurice Starks, B.S., Program Director

Clinical and Genetic Epidemiology Research Branch (CGERB)

Deborah Winn, Ph.D., Chief
J. Fernando Arena, M.D., Ph.D., Program Director
John Fox, M.P.H., Program Analyst
Carol Kasten, M.D., Program Director
Isis Mikhail, M.D., M.P.H., Dr.P.H., Program Director
Scott Rogers, M.P.H., Program Analyst
Daniela Seminara, Ph.D., M.P.H., Program Director

SBIR (R43/R44) and STTR (R41/R42) Grants

Jay Choudhry, M.S., Program Director

Sources of Information on Grant Policies, Funding, and Training

- Our NCI Division of Cancer Control and Population Sciences (DCCPS) Home page: *cancercontrol.cancer.gov* for grant policy alerts and information on funding opportunities.
- NCI Division of Extramural Activities (DEA): *deainfo.nci.nih.gov*
- *Grants.gov* (central resource to find and apply for U.S. grants)
- NIH Electronic Submission of Grant Applications Web site: *era.nih.gov/ElectronicReceipt/index.htm*
- Subscribe to:
  - **NCI Cancer Bulletin** (weekly newsletter): *cancer.gov/ncicancerbulletin*
  - 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: *ebers@mail.nih.gov*
- NCI Research Resources (directory of more than 100 products and services): *resresources.nci.nih.gov*
- NCI-Sponsored Training Opportunities: *www.cancer.gov/researchandfunding/training*
  EGRP's training Web site: *epi.grants.cancer.gov/training*
- **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*.)**