BREAST CANCER AND THE ENVIRONMENT RESEARCH PROGRAM

14th Annual Meeting

Bringing Precision to the Future of Environmental Breast Cancer Research

November 7-8, 2019

The American Hotel

Atlanta, Georgia

Supported by the
National Institute of Environmental Health Sciences (**NIEHS**) and
National Cancer Institute (**NCI**)

Hosted by the University of Wisconsin-Madison

Welcome

Welcome to the BCERP Annual Meeting

The overarching goal of the Breast Cancer and the Environment Research Program (BCERP) is to support integrated scientific research to enhance our understanding of environmental and genetic factors underlying breast cancer risk throughout the lifespan, with particular focus on the influence of environmental exposures during time windows of susceptibility. Our Annual Meeting supports this goal by bringing scientists, community partners, breast cancer advocates, and others together to translate research into action to reduce breast cancer.

Multidisciplinary, community-engaged research has been a hallmark of BCERP (previously known as BCERC, or the Breast Cancer and the Environment Research Centers) since its inception in 2003. This week's meeting is the 14th public conference for BCERP and will likely be the last conference for this consortium. We hope this conference will serve as a celebration of BCERP accomplishments and a springboard for future research and outreach to reduce the burden of breast cancer.

Special Thanks

The BCERP Annual Meeting Planning Committee offers special thanks to the program co-chairs, D. Joseph Jerry, Sallie S. Schneider, Anna G. Symington, and Mia M. Gaudet for their extraordinary commitment to facilitating the development of the program for this meeting and to the entire BCERP consortium for attending and sharing their expertise and their valuable contributions to the program.

This BCERP Annual Meeting is supported by grants from the National Institute of Environmental Health Sciences and the National Cancer Institute, including grant U01ES026127 to the University of Wisconsin-Madison to support the Coordinating Center.

Table of Contents

General Information 3
Program Committee 4
Agenda 5-8
Session 3: Emerging Research Abstracts 9-11
Poster Session Map 12
Posters 13-15
Poster Abstracts 16-32
Participant List 33-36
Notes 37-39
Local Restaurant Suggestions 40
Site-seeing Attractions 41
The American Hotel Map 42
Meeting at a Glance ..... Back Cover

General Information

Registration
The registration table will be open on Thursday, Nov. 7 from 7:00 a.m. – 5:00 p.m., and on Friday, Nov. 8 from 7:30 a.m. – 12:30 p.m.

Meals
Coffee, tea and water will be available during the meeting. Light lunch options will be provided on Thursday, Nov. 7 beginning at 12:15 p.m. in Apollo B.

Poster Information
In the poster session, meeting attendees will present their work and ideas in progress. Presenters will be available to discuss their respective posters during the session in Apollo A on Thursday, Nov. 7. Posters will be eligible for poster awards. The poster session winners will be announced during the closing remarks on Friday, Nov. 8.

Poster presenters are asked to hang their posters between 7:00 a.m.-12:00 p.m. on Thursday, Nov. 7.

Presenters for odd-numbered posters are asked to stand at their posters from 1:45 – 2:30 p.m., and presenters for even-numbered posters are asked to stand at their posters from 2:30 – 3:15 p.m.

All posters are strongly encouraged to remain on display through the end of the meeting on Friday, Nov. 8 at 12:30 p.m. They should be promptly removed between 12:30-1:00 p.m.

A look back and a look forward – the impact of the BCERP
Please join us for a reception and panel session on Thursday, Nov. 7 from 5:00 – 6:30 p.m. chaired by Dr. Frank Biro as we celebrate the impact of BCERP and look forward to the future.

Wifi
For guests staying at The American Hotel and for meeting attendees, wifi is available for free using the network attwifi_meeting. A password will be provided at registration.

Post Meeting Survey
Please respond to the online survey that will be emailed at the conclusion of the meeting.

Program Committee

Jennifer Bird
University of Wisconsin-Madison
Madison, WI

Frank Biro
Cincinnati Children's Hospital Medical Center
Cincinnati, OH

Abee Boyles
National Institute of Environmental Health Sciences
Durham, NC

Celia Byrne
Uniformed Services University of the Health Sciences
Bethesda, MD

Shiuan Chen
City of Hope
Duarte, CA

Symma Finn
National Institute of Environmental Health Sciences
Durham, NC

Mia Gaudet - Co-chair
American Cancer Society
Atlanta, GA

Joseph Jerry - Co-chair
University of Massachusetts Amherst
Amherst, MA

Tram Kim Lam
National Cancer Institute
Rockville, MD

Kristen Malecki
University of Wisconsin-Madison
Madison, WI

Mary Beth Martin
Georgetown University
Washington, DC

Julie McGregor
University of Wisconsin-Madison
Madison, WI

Eileen McGuine
University of Wisconsin-Madison
Madison, WI

Sallie Schneider - Co-chair
Baystate Medical Center
Springfield, MA

Anna Symington-Co-chair
University of Massachusetts Amherst
Amherst, MA

Amy Trentham-Dietz
University of Wisconsin-Madison
Madison, WI

Claudia Vredeveld
University of Wisconsin-Madison
Madison, WI

Agenda

Thursday, November 7, 2019

7:00 a.m.-5:00 p.m.	Registration
8:00-8:15 a.m.	**Welcome and Introduction of Keynote Speaker – Apollo B** Hosts: D. Joseph Jerry, PhD, *University of Massachusetts Amherst* Sallie S. Schneider, PhD, *Baystate Medical Center* Anna G. Symington, *University of Massachusetts Amherst* Mia M. Gaudet, PhD, *American Cancer Society* Abee L. Boyles, PhD, Program Director, Population Health Branch, *National Institute of Environmental Health Sciences* Tram Kim Lam, PhD, MPH, Program Director, Environmental Epidemiology Branch, *National Cancer Institute*
8:15-9:00 a.m.	**Keynote Address – Apollo B** **The BCERP legacy: windows of susceptibility to environmental risks of disease** Gwen Collman, PhD, Director, Extramural Research & Training, *National Institute of Environmental Health Sciences*
9:00-10:30 a.m.	**Session 1: Myths, Messages, and Communicating Uncertainty – Apollo B** Chair: Anna G. Symington, *University of Massachusetts Amherst* "Breast Cancer Survivors Lend Their Voices" video Why me? Conversations with women newly diagnosed with breast cancer Grace Makari-Judson, MD, *University of Massachusetts Medical School-Baystate* Advancing cancer health equity through strategic community-engagement - Designing population and implementation research that matters Tabia Henry Akintobi, PhD, MPH, *Morehouse School of Medicine* Communicating BCERP science from the lens of uncertainty theories Kami Silk, PhD, *University of Delaware* Open discussion
10:30-10:45 a.m.	Break

Thursday, November 7, 2019 *Cont.*

10:45 a.m.-12:15 p.m. **Session 2: Interactions Between Environmental Chemicals and Inherited Risk – Apollo B**
Chair: James Shull, PhD, *University of Wisconsin-Madison*

Using the diversity outbred mice to identify gene by evironment interactions
Alison Harrill, PhD, *National Institute of Environmental Health Sciences*

Detecting gene by environment interactions in population studies
Mia M. Gaudet, PhD, *American Cancer Society*

Why genetic, environment, and epigenetic regulation matter to breast cancer risk
Rachel Miller, MD, FAAAAI, *Columbia University*

On the need to consider both genetic and windows of susceptibility for environmental exposures and breast cancer risk with the specific example of PAHs
Mary Beth Terry, PhD, *Columbia University*

Open discussion

12:15-1:45 p.m. Lunch is provided – Apollo B

1:45-3:15 p.m. **Poster Session – Apollo A**
Presenters for odd-numbered posters are asked to stand at their posters during 1:45-2:30 p.m., and presenters for even-numbered posters are asked to stand at their posters during 2:30-3:15 p.m.

3:15-3:30 p.m. Break

Thursday, November 7, 2019 *Cont.*

3:30-5:00 p.m.

Session 3: Emerging Research – Apollo B
Chair: Jennifer Ohayon, PhD, *Silent Spring Institute*

Barriers and opportunities for breast cancer organizations to focus on environmental health and disease prevention: a mixed-methods approach using website analyses, interviews, and focus groups
Jennifer Ohayon, PhD, *Silent Spring Institute*

Evaluation results of BCERP continuing medical education for pediatric health care professionals
Daniel Totzkay, PhD, *West Virginia University*

Hair dye and chemical straighteners in relation to breast cancer risk in a large US population of black and white women
Alexandra White, PhD, *National Institute of Environmental Health Sciences*

Effect of oxybenzone exposure during pregnancy and lactation on the protective effect of parity on mammary cancer in mice with p53 -/- epithelium
Karen Dunphy, PhD, *University of Massachusetts Amherst*

Combined effects of 17β-estradiol (E2), progesterone (P4), and polybrominated diphenyl ethers (PBDEs) on postmenopausal murine mammary glands at the single cell resolution
Kohei Saeki, PhD, *City of Hope*

Hormone phenotypes defined in peripubertal girls, a novel approach using principal components and cluster analysis in a longitudinal cohort
Cecily Fassler, PhD, *University of Cincinnati College of Medicine*

5:00-6:30 p.m.

Session 4: A Look Back and a Look Forward - The Impact of the BCERP - Apollo B
Chair: Frank Biro, MD, *Cincinnati Children's Hospital Medical Center*

Panel Members:
Gwen Collman, PhD, *National Institute of Environmental Health Sciences*
Caroline Dilworth, PhD, MSPH, *Thrive Integrative Wellness Coaching*
Gary Ellison, PhD, MPH, *National Cancer Institute*
Shuk-Mei Ho, PhD, *University of Arkansas for Medical Science*
Karen Miller, *Michigan State University*
Les Reinlib, PhD, *National Institute of Environmental Health Sciences*
Susan Teitelbaum, PhD, *Icahn School of Medicine at Mount Sinai*

Open discussion

Agenda

Friday, November 8, 2019

7:30 a.m.-12:30 p.m. Registration

8:30-10:00 a.m. **Session 5: Addressing risk posed by mixtures of chemicals – Apollo B**
Chair: Kristen Malecki, PhD, MPH, *University of Wisconsin-Madison*

New methods for analyzing mixtures in population based research
Mary Turyk, PhD, MPH, *University of Illinois at Chicago*

Multiple exposures to chemicals with biologic persistence do influence the levels of some reproductive hormones during female puberty
Susan Pinney, PhD, *University of Cincinnati*

Effects of estrogen, progesterone, and PBDEs on mammary gland structure after surgical menopause
Shiuan Chen, PhD, *Beckman Research Institute of the City of Hope*

Multiple chemical exposures and breast cancer risk, findings from and the California Teacher's Study
Peggy Reynolds, PhD, MPH, *University of California, San Francisco*

Open discussion

10:00-10:15 a.m. Break

10:15-11:45 a.m. **Session 6: Environmental chemicals, metabolism and immune function and breast cancer risk – Apollo B**
Chairs: Mary Beth Martin, PhD, *Georgetown University* and Shiuan Chen, PhD, *Beckman Research Institute of the City of Hope*

How the Breast Environment May Influence Breast Cancer Risk
Karin Michels, ScD, PhD, *University of California, Los Angeles*

Effect of Exercise on Metabolic Syndrome in Black Women by Family History and Predicted Risk of Breast Cancer: The FIERCE Study
Lucile Adams-Campbell, PhD, *Georgetown University Medical Center*

Endocrine Disrupting Chemicals and the Immune System: A Possible Mechanism for Facilitated Cancer Progression
Sallie Schneider, PhD, *Baystate Medical Center*

African Ancestry and Tumor Immune Responses Leading to Disparate Clinical Outcomes
Melissa Davis, PhD, *Weill Cornell Medical College*

Open discussion

11:45 a.m.-Noon **Announcement of Poster Awards and Closing Remarks – Apollo B**
Gwen Collman, PhD, Director, Extramural Research & Training, *National Institute of Environmental Health Sciences*

12:30 p.m. Meeting Adjourned

Barriers and opportunities for breast cancer organizations to focus on environmental health and disease prevention: a mixed-methods approach using website analyses, interviews, and focus groups

Authors: Ohayon JL, Nost E, Silk K, Rakoff M, Brody JG
Presenter: Jennifer Ohayon, PhD, *Silent Spring Institute*

Purpose of study: Historically, breast cancer activists successfully advocated for innovative research on the links between breast cancer and chemical exposures, and were formative in the creation of federal research programs such as BCERP. Since then, new evidence supports hypotheses that common industrial and consumer chemicals are linked to the disease, and expert panels recommend reducing exposures. We evaluated whether these research results and recommendations are translated back into the work of breast cancer organizations and what barriers and opportunities influence their ability to focus on environmental factors. Methods: We used a Python script to evaluate the frequency of environmental terms on the websites of 81 breast cancer organizations (>14,000 associated URLs), and conducted two focus groups and 20 interviews with leaders of breast cancer organizations. Results: Half of the organizations include information on environmental chemicals on their websites, but references are infrequent and rarely cite specific chemicals of concern. Most organizations (82%) discuss other risk factors such as exercise, diet, family history, or genetics. From interviews and focus groups, we identified four types of barriers to addressing environmental chemicals: 1) time and resource constraints, 2) limited knowledge of the state of the research and lack of access to experts, 3) difficulties with messaging, including concern that socioeconomic factors make it difficult for individuals to reduce their exposures, and 4) institutional obstacles, such as the downplaying of environmental risks by industry interests. Participants expressed the desire for easy-to-adopt educational programs and increased federal funding for scientist-advocate research partnerships. Conclusion: Our research underscores the need for environmental breast cancer experts and trusted cancer organizations to increase research translation activities so that breast cancer organizations can communicate new science on environmental factors in their online and in-person work. Moreover, our research highlights how most groups are focusing on providing resources to diagnosed women, including addressing problems with healthcare access, which displaces their ability to work on breast cancer prevention.

(Supported by the NIEHS/NCI grant T32ES023769 and U01ES026130.)

Evaluation results of BCERP continuing medical education for pediatric health care professionals

Authors: Silk K, Totzkay D, Thomas B, Walling B, Smith S
Presenter: Daniel Totzkay, *West Virginia University*

This study evaluated the effectiveness of two, one-hour online training modules designed to educate pediatric health care providers (PHCPs) about breast cancer and the environment via a Continuing Medical Education (CME) mechanism. The training modules were professionally produced and edited to include slides, graphics, quizzes, and full narration by BCERP researchers. A snowball sample of PHCPs (i.e., pediatricians, family physicians, nurse practitioners, and nurses; N=100) was recruited through a range of health care organizations' (i.e., AAP, NAPNAP, children's hospitals) newsletters and social media as well as through email directly to large family and pediatric health practices. At the time of the training, participants completed pre- and post-test questionnaires; approximately three weeks later, participants completed a follow-up survey (response rate=91%). Survey items measured knowledge, perceptions of training, efficacy, behavioral intentions, and subsequent behavior. ANOVA results show that using CME opportunities to train PHCPs about BCERP science and relevant communication strategies can increase knowledge on the cancer-environment connection, as well as intentions to engage in recommended practices. PHCPs improved their accuracy in identifying risk reduction strategies to recommend to their patients regarding exposure to PFOA, BPA, PBDE, and phthalates. Results demonstrated PHCPs' understanding of windows of susceptibility and their implication for breast cancer risk and an increased willingness to integrate recommendations into their practices. Findings were consistent from an immediate post-test to the three-week follow-up. This research demonstrates the feasibility of CME to diffuse emerging science to health care providers so they are able to provide evidence-based recommendations to their patients and their families.

(Supported by the NIEHS/NCI grant 5R21ES027418-02.)

Hair dye and chemical straighteners in relation to breast cancer risk in a large US population of black and white women

Authors: White AJ, Eberle C, Taylor KW, Sandler DP
Presenter: Alexandra J White, *National Institute of Environmental Health Sciences*

Purpose: Many hair products contain endocrine disrupting compounds and carcinogens potentially relevant to breast cancer. Products used predominately by black women may contain more hormonally-active compounds. In a national prospective cohort study, we examined the association between hair dye and chemical relaxer/straightener use and breast cancer risk by race. Methods: Sister Study participants (N=46,709), women ages 35-74, were enrolled between 2003-2009, and had a sister with breast cancer but were breast cancer-free themselves. Enrollment questionnaires included past 12-month hair product use. Cox proportional hazards models estimated adjusted hazard ratios (HRs) and 95% confidence intervals (95% CIs) for the association between hair products and breast cancer; effect measure modification by race was evaluated. Results: During follow-up (mean=8.3 years), 2,794 breast cancers were identified. 55% of participants reported using permanent dye at enrollment. Permanent dye use was associated with 45% higher breast cancer risk in black women (HR=1.45, 95% CI: 1.10-1.90), and 7% higher risk in white women (HR=1.07, 95% CI: 0.99-1.16) (heterogeneity p=0.04). Among all participants, personal straightener use was associated with breast cancer (HR=1.18, 95% CI 0.99-1.41); with higher risk associated with increased frequency (p for trend =0.01). Non-professional application of semi-permanent dye (HR=1.28, 95% CI 1.05-1.56) and straighteners (HR=1.27, 95% CI 0.99-1.62) to others was associated with breast cancer risk. Conclusions: In a population of women with a family history of breast cancer, we observed a higher breast cancer risk associated with any straightener use and personal use of hair dye, especially among black women. These results suggest that chemicals in hair products may play a role in breast carcinogenesis.

(Supported by the NIEHS/NCI grant Z01-ES044005.)

Effect of oxybenzone exposure during pregnancy and lactation on the protective effect of parity on mammary cancer in mice with p53 -/- epithelium

Authors: Matouskova K, Roberts A, Dunphy KA, Hagen M, Schneider SS, Jerry DJ, Vandenberg LN
Presenter: Karen Dunphy, *University of Massachusetts Amherst*

Hormones and endocrine disrupting chemicals (EDCs) are generally thought to have permanent 'organizational' effects when exposures occur during development but not adulthood. Yet, an increasing number of studies have shown that pregnant females are disrupted by EDC exposures, with some effects that are permanent. In our prior study (LaPlante et al., J Endocrine Society 2018), we observed long-term effects of exposure to oxybenzone, an estrogenic chemical found in sunscreen and personal care products, on the morphology of the mammary gland in mice exposed during pregnancy and lactation. Female mice exposed to oxybenzone from pregnancy day 0 until weaning had permanent changes to ductal density that were significantly different from both the nulliparous and vehicle groups. These effects were observed five weeks after exposures ceased. A monotonic, dose-dependent increase in cell proliferation was also observed in the oxybenzone-treated females. In the current study, we transplanted p53 -/- epithelial cells into cleared fat pads of BALB/c mice, and then separated the females into three groups: nulliparous, parous controls, and parous + oxybenzone exposure. In this latter group, females were exposed to 3000 µg/kg/d oxybenzone throughout pregnancy and lactation. As in prior studies, we found that parity was protective against tumor development. Yet, oxybenzone exposure eroded this protection, with more females developing tumors at an earlier stage compared to both parous controls and nulliparous females. Moving forward, we are evaluating each tumor histologically to determine if oxybenzone also alters tumor severity and type. The results we have obtained to date are consistent with our hypothesis that estrogenic endocrine disrupting chemicals can interfere with the known protection that pregnancy provides against breast cancer.

(Supported by the National Institutes of Health grant U01ES026140.)

Combined effects of 17β-estradiol (E2), progesterone (P4), and polybrominated diphenyl ethers (PBDEs) on postmenopausal murine mammary glands at the single cell resolution

Authors: Saeki K, Chang G, Kanaya N, Wu X, Bernal L, Rakoff M, Neuhausen SL, Chen S
Presenter: Kohei Saeki, *City of Hope*

Purpose: Development, maturation and homeostasis of the mammary gland are orchestrated by complex interaction of various cell types in the tissue mainly under control of E2 and P4. PBDEs, as flame retardants in household materials, have been found to bind and modulate the activity of estrogen through the interaction with estrogen receptor (ER). Their persistence in the environment led to great concerns about PBDEs' potential endocrine disrupting activity, which includes breast cancer development. However, this presumed causal relationship has not been definitively investigated. In this study, effects of PBDEs, together with E2 and P4 were comprehensively evaluated at the single cell resolution. Methods: 9-week-old female BALB/cj mice were ovariectomized and treated with E2, P4 and PBDEs (as a mixture of 3 major congeners; BDE-47, -100 and -153) for 1 week, after 10 or 20 weeks from surgical menopause (P10 and P20, respectively). Inguinal mammary glands were collected, digested and sequenced using the single cell RNA sequencing 10x Genomics platform. Results: A total of 9 major clusters were putatively identified; 2 luminal cell clusters (HR+ and HR-), 1 basal cell cluster, 3 fibroblast clusters characterized by Ccl2, Cxcl14 and Spon1 expression, respectively, an endothelial cell cluster, and 2 hematopoietic cell clusters. Esr1 (ER) expression was found mainly in HR+ luminal and Ccl2+ fibroblasts while Pgr (PR) expression was exclusively in HR+ luminal cells. In the untreated regressed glands, more Ccl2+ fibroblasts were found in P20. Supplementation of E2 dramatically increased the size of both luminal clusters, especially in P20, and addition of E2+P4 significantly expanded the HR- cluster. Exposure to PBDEs in addition to E2+P4 led to increase in HR+ luminal cells although impact on other clusters was variable between P10 and P20. Conclusion: Our analyses provide valuable insights into mechanism by which mammary glands increase sensitivity to sex hormones with time following menopause as well as P4 stimulates Esr1-/Pgr- putative progenitor expansion. Variability of effect of PBDEs could result from differing states of the gland, which should be further explored by quantitative analyses and by using a chemical (VCD)-induced menopausal transition model.

(Supported by grant from National Institutes of Health/NCI U01 ES026137.)

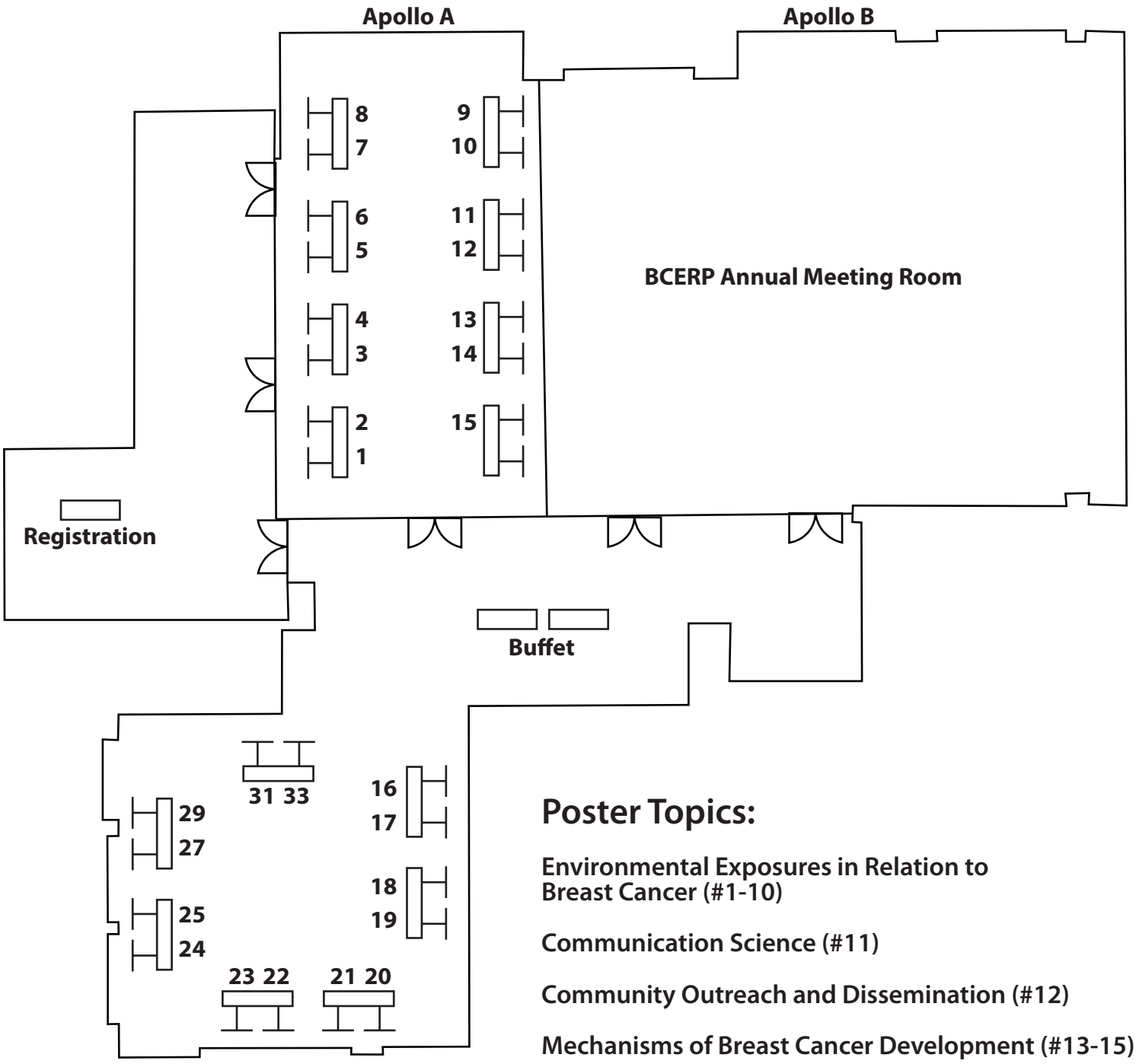
Hormone phenotypes defined in peripubertal girls, a novel approach using principal components and cluster analysis in a longitudinal cohort

Authors: Fassler CLS, Xie C, Biro FM, Pinney SM
Presenter: Cecily Fassler, *University of Cincinnati College of Medicine*

Purpose: Age at pubertal onset differs in girls, and age at menarche associated with the risk of breast cancer. We used principal component and cluster analysis to identify, for the first time, distinct hormone phenotypes of peripubertal girls in a longitudinal cohort.
Methods: We measured serum DHEA, estrone, estradiol, and testosterone at five time periods relative to onset of thelarche (time =-18,-12,-6, thelarche, and +6) in 269 peripubertal girls from the greater Cincinnati area. Principal components analysis was performed to select a subset of relevant hormone phenotypic variables. Cluster Analysis was then applied to identify phenotypes of girls based on those predictive hormone variables. After the phenotypes were defined, differences among the phenotypes with regard to demographics and other characteristics were examined to further characterize the profiles.
Results: Hormone profile Phenotype 1 (n=42) included girls with high DHEA, as well as high testosterone and estrone; Phenotype 2 (n=37) included girls with very high estradiol as well as high DHEA and testosterone; Phenotype 3 (n=74) included girls with little change in hormone values except for DHEA-S; and Phenotype 4 (n=96) consisted of girls with very low values for all hormones around the time of thelarche. When controlling for race, maternal age of menarche, caregiver education, and body mass, different phenotypes were associated with the age of pubertal events. Girls with low levels of all four hormones, Phenotype 3b, were youngest at thelarche (8.67 years), those in Phenotype 2, with the highest estradiol levels and estradiol surge six months after thelarche, were youngest at menarche (11.87 years), with shortest pubertal tempo.
Conclusions: Hormone phenotypic clustering can identify clinically relevant subgroups with differing ages of thelarche, pubarche, and menarche. These findings may enhance the understanding of timing of pubertal milestones and risk of adult disease.

(Supported by the NIEHS/NCI grants U01ES012770, U01ES019453, U01ES019457, U01ES026119, R01ES029133, T32GM063483, T32ES010957, P30 ES006096 CSTA-UL1RR026314.)

Poster Session Map



Poster Topics:

Environmental Exposures in Relation to Breast Cancer (#1-10)

Communication Science (#11)

Community Outreach and Dissemination (#12)

Mechanisms of Breast Cancer Development (#13-15)

Special Topics (#16-20)

Emerging Scholars (#21-23)

Windows of Susceptibility (#24-33)

Posters

Environmental Exposures in Relation to Breast Cancer (#1-10)

Poster #	Presenter	Title
1-T	Amye L. Black	Inter-individual variation in response to estrogen and xenoestrogens in normal human breast epithelial cells
2	Bethsaida Cardona	Chemicals that increase synthesis of estradiol and progesterone are potential risk factors for breast cancer
3	Yuan Chun Ding	Association of PBDEs and BPA and epigenome modulation in sera of women at the menopausal transition
4-T	Reyhane Hoshyar	Effect of oxybenzone on eosinophil recruitment and expression of immune-related gene products in p53-null mice exposed to a high saturated animal fat diet
5-T	Stephanie Morin	BP3 and PBDE facilitate metastatic growth of an ER negative breast cancer cell line
6	Olena Morozova	Effects of high fat diet and oxybenzone on recrutment of tumor-associated cd206 macrophages
7	Jennifer Ohayon	Can a state law reduce population-level exposures to breast cancer-relevant chemicals? A case study of California Proposition 65
8	Susan M. Pinney	The association between sex hormones, pubertal milestones and benzophenone-3 exposure, measured by urinary biomarker or questionnaire
9	Samantha Puvanesarajah	Associations of alcohol and smoking with breast cancer subtypes in the Breast Cancer Association Consortium
10	Katherine Reeves	Estrogenic activity following BPA exposure from consumption of canned soup

Communication Science (#11)

Poster #	Presenter	Title
11	Daniel Totzkay	Perceptions of breast density in states with and without dense breast notification legislation

Community Outreach and Dissemination (#12)

Poster #	Presenter	Title
12	Katherine Boronow	A new model for environmental health literacy about endocrine disrupting chemicals

Mechanisms of Breast Cancer Development (#13-15)

Poster #	Presenter	Title
13	Kelly Gregory	The use of patient-derived breast tissue explants to study resident macrophage polarization and the effects of xenoestrogen exposure

*-T indicates trainee poster

Posters

Mechanisms of Breast Cancer Development (#13-15) *Cont.*

Poster #	Presenter	Title
14-T	Kohei Saeki	Combined effects of 17β-estradiol (E2), progesterone (P4), and polybrominated diphenyl ethers (PBDEs) on postmenopausal murine mammary glands at the single cell resolution
15-T	Debashish Sahay	Prenatal PAH induces mammary hyperplasia and altered ERα in grand-offspring adult female mice

Special Topics (#16-20)

Poster #	Presenter	Title
16	Erin Bailey	Barriers and opportunities for breast cancer screening and risk reduction among African American women
17	Amanda Hernandez	A social and economic systems approach to advance environmental cancer prevention
18	Kristin Marks	Epigenetic and metabolic features associated with breast cancer in the Michigan Polybrominated Biphenyl Registry
19	Sharima Rasanayagam	A Breast Cancer Primary Prevention Plan for the State of California
20-T	Jennifer Bird	Celebrating the Accomplishments of BCERP

Emerging Scholars (#21-23)

Poster #	Presenter	Title
21-T	Raymond Lin	To replace toxins linked with breast cancer: the development of antimicrobial sustainable food packaging films utilizing bilayer emulsion compositions
22-T	Timothy Liu	Developing a safer, biocompatible polymer for tissue engineering which shows potential for breast reconstruction
23-T	Subhana Zafar	Earthworms (Eisenia fetida) recover from Roundup® exposure

Windows of Susceptibility (#24-33)

Poster #	Presenter	Title
24-T	Rebecca Kehm	Physical activity and breast tissue composition during the adolescent window of susceptibility
25	Nhi M Dang	PFOA and Zeranol exposure during puberty affect the transcriptomic profile of the rat mammary gland
27	Nhi M Dang	Immune response of the rat mammary gland to the pubertal exposure to PFOA+ZERANOL
29	Nhi M Dang	Pubertal exposure to different doses of Zeranol induces unique changes in the transcriptomic profile of rat mammary glands

*-T indicates trainee poster

Windows of Susceptibility (#24-33) *Cont.*

Poster #	Presenter	Title
31	Nhi M Dang	Butyl benzyl phthalate and Perfluorooctanoic acid induce long term impact on the gene expression profile of mammary gland in exposed rats during puberty
33	Nhi M Dang	Single or combined butyl benzyl phthalate (BBP) and Zeranol exposure during puberty affect the expression profile in the rat mammary gland at older age

*-T indicates trainee poster

Environmental Exposures in relation to Breast Cancer

#1: Inter-individual variation in response to estrogen and xenoestrogens in normal human breast epithelial cells

Black AL, Dunphy KA, Roberts AL, Schneider SS, Jerry DJ

Background: Previous work has highlighted the role of estrogen in both contributing to and reducing breast cancer risk. Exposure to xenoestrogens (XEs), a class of endocrine disrupting chemicals which can mimic the actions of endogenous estrogens, may affect the balance between the protective and harmful effects of estrogen on breast cancer risk. The effects of xenoestrogens may differ among individual women, as genetic diversity is known to impact breast cancer susceptibility and responses to chemicals. We hypothesize that there are subsets of individuals uniquely sensitive to estrogen and xenoestrogens.
Methods: To examine variation in response to 17β-estradiol (E2) and XEs benzophenone-3 (BP3) and propylparaben (PP), we utilized conditionally immortalized primary human mammary epithelial cells (ciHMEC) and stably immortalized HMECs. Because HMECs rapidly lose expression of estrogen receptors in 2D culture, we transiently transfected ESR1 and/or ESR2 into cells. Responses to E2, BP3, and PP were measured using dual luciferase assays. Stably immortalized HMEC lines were infected with a lentiviral plasmid containing tetracycline inducible ESR1. Proliferation assays were then used to examine E2-induced responses in ESR1 expressing HMECs.
Results: Our results show that BP3 and PP have estrogenic activity in HMECs primarily through estrogen receptor alpha. In all stably and conditionally immortalized HMECs, we observed significant responses to E2, BP3, and PP. We observed a 2.5-fold variation in response to E2, 2.3-fold variation to BP3, and 3.6-fold variation to PP treatment at concentrations that are relevant to environmental exposures. In stably immortalized HMECs expressing inducible ESR1, we observed modest proliferative responses in 2/3 lines tested.
Conclusions: Our ciHMECs demonstrate substantial variation in responses to XEs. As cells expressed saturating amounts of ESR1, it indicates inter-individual variation in intracellular signaling that is determined by either genetic variants or epigenetic differences among individuals. We also find proliferative effects of E2 differ among patient cell lines. These HMEC lines can be used to determine the mechanistic differences in E2/XE sensitivity among women.

(Supported by National Institute of Environmental Health Sciences of the National Institutes of Health # U01ES026140, US Department of Defense # W81WH-15-1-0217, and the Rays of Hope Center for Breast Cancer Research)

#2: Chemicals that increase synthesis of estradiol and progesterone are potential risk factors for breast cancer

Cardona B, De La Rosa V, Rudel RA

Established breast cancer risk factors such as hormone replacement therapy and reproductive history are thought to be related to increased estrogen and progesterone levels or activity. While most research about environmental risk factors has focused on chemicals that bind to and activate the estrogen receptor, little attention has been paid to chemicals that may affect steroidogenesis, for example by increasing synthesis of estradiol or progesterone systemically or in the breast. Using publicly available high throughput screening data from EPA's ToxCast, we identified 418 chemicals that increased levels of estradiol and/or progesterone in a H295R steroidogenesis assay –a well-established in vitro tool that uses adrenal cortical cells to measure the effects of endocrine disrupting chemicals on steroidogenesis. We prioritized these active chemicals based on their potency using their lowest active concentration, AC50 from dose-response experiments, and an integrated measure of effects on the steroid synthesis pathway (adjusted maximum mean Mahalanobis distance). We further prioritized chemicals with highest expected general population exposures using output from EPA's high throughput exposure modeling. EPA's exposure model generates median population exposure estimates by using a chemical's structure and physiochemical properties, exposure predictors such as production volume, and known or predicted use in consumer products, food, pesticides, and industrial processes. To provide BCERP grantees with this information for the chemicals in their own studies, we also tested BCERP chemicals in H295R, if they had not been tested by EPA, through a contract laboratory. This work highlights chemicals that are a priority for breast cancer research based on their ability to increase levels of estradiol and progesterone, and prioritizes these based on potency and their likely

populationthese based on potency and their likely population exposures. We will present these priority chemicals, and include findings from BCERP chemicals. Logical next steps are to measure human exposure to these chemicals, especially in high-exposed subpopulations, and to conduct experimental studies to determine effects on mammary gland development and carcinogenesis.itself decreased apoptosis in epithelial tumors. In spindle cell tumors, BP-3 increased proliferation in tumors from all diet groups.

(Supported by NIEHS Breast Cancer and Environmental Risk Factors 2017 Opportunity Fund Grant)

#3: Association of PBDEs and BPA and epigenome modulation in sera of women at the menopausal transition

Ding YC, Steele L, Hurley, S, Reynolds P, Chen S, Neuhausen SL

Growing evidence from epidemiology and experimental model studies suggests that exposure to endocrine- disrupting chemicals in the environment may modify risk of developing breast cancer. We hypothesize that during the menopausal transition, polybrominated diphenyl ethers (PBDEs) and bis-phenol A (BPA) promote development of hormone-responsive breast cancers.
Methods: PBDE levels were measured in serum of 655 (184 for BPA) female breast cancer cases and controls between ages 40 to 58 years from the California Teachers Study (CTS). We measured gene-specific methylation using the Illumina EPIC Methylation chip for 316 controls. Multivariable linear regression adjusting for covariates was used to identify differentially methylated sites (DMSs) and differentially methylated regions (DMRs) associated with chemical exposure. Using targeted bisulfite next generation sequencing (tbNGS) for a subset of these biomarkers of exposure, we measured methylation levels in 133 invasive breast cancer cases and 301 age-matched controls. Conditional logistic regression models were used to test association of the DMRs and DMSs and risk of developing breast cancer.
Results: We identified 223 DMSs (corrected FDR < 0.25) and 157 DMRs (minimum FDR < 0.05) associated with exposure to three PBDE congeners and BPA. Among genes at DMRs, top pathways enriched were cellular metabolism and Aryl Hydrocarbon Receptor signaling. Based on the magnitude of association, the top 10 DMSs and 38 DMRs were selected for validation using tbNGS. In 30 of 38 DMRs at gene promoter regions, decreased methylation (hypomethylation) was found, indicating that the PBDE congeners and BPA more often increase gene expression. This result is concordant with our in-vitro analysis of PBDE-treated cell lines where more sites were hypomethylated than hypermethylated. Of 25 DMRs and 3 DMSs validated by tbNGS, 5 DMRs and 1 DMS were associated with risk of developing invasive breast cancer (multiple testing adjusted $P < 0.05$); of which 5 were significantly hypomethylated in invasive cases compared to healthy controls. In conclusion, we identified methylation differences associated with BPA and PBDE serum levels. Six biomarkers of BPA/PBDE exposure were significantly associated with development of breast cancer.

(Supported by the NIEHS/NCI grant U01ES026137, R01 CA77398, CBCRP grant #16ZB-8501, and the City of Hope Integrative Genomics Core supported by the NCI of the NIH under award number P30CA033572)

#4: Effect of oxybenzone on eosinophil recruitment and expression of immune-related gene products in p53-null mice exposed to a high saturated animal fat diet

Hoshyar R, McSween MI, Knickerbocker KM, Kariagina A, Morozova O, Haslam SZ, Schwartz RC

Purpose: We previously demonstrated that a high-fat diet (HFD) had promoted the mammary tumorigenesis in a p53-null transplant BALB/c mouse model. In published studies, we found that HFD stimulates M2 macrophage polarization, likely through eosinophil recruitment and IL-4 production in the mammary gland. These results motivated us to study the impact of HFD and oxybenzone (benzophenone-3, BP-3, a sunscreen agent) on eosinophil recruitment and expression of IL-4 and other eosinophil-related markers, including IL5ra, CCL3, and CCL24 at times prior to tumor development.
Methods: p53-null mammary gland-transplanted BALB/c mice were fed by the following diets with or without BP-3 from 3 to 26 weeks of age: low-fat diet (LFD, 10% kcal fat); high-fat diet (HFD, 60% kcal fat); LFD switched to HFD at 10 weeks of age (L-H); HFD switched to LFD at 10 weeks of age (H-L). At 6, 7, 8, 13 and 26 weeks, inguinal mammary glands were excised, RNA isolated, and specific RNAs identified by qRT-PCR.

Eosinophils were identified by vital new red staining.
Results: Mammary glands of mice fed HFD showed BP-3-induced increases in eosinophils at 8 and 13 weeks, as well as increases in IL4, IL5ra, CCl3, and CCl24 RNAs at 13 and 26 weeks. There were no significant differences in these RNAs at earlier time-points. A similar but weaker induction of these RNAs in response to BP-3 was observed in mice fed LFD. For all of the RNAs measured, the highest expression level was observed at 26 weeks in HFD-fed mice exposed to BP-3. Interestingly, BP-3 also enhances the expression of these immunity-related genes in mice fed either switch diet (L-H and H- L) at 13 and 26 weeks.
Conclusions: The expression of RNAs related to macrophage polarization and eosinophil recruitment were significantly increased in mammary glands of mice fed with HFD + BP-3 at 13 and 26 weeks. Different patterns of gene expression were observed at the earlier time points (6, 7 and 8 weeks). High levels of IL5ra and CCl24 RNAs are particularly consistent with eosinophil recruitment, and elevated IL4 may induce M2 macrophage polarization in pre-tumor mice. These results suggest an additive effect of HFD and BP-3 treatment in the early immune response to mammary tumorigenesis in the p53-null system.

(Supported by the Breast Cancer and the Environment Research Program Grant U01ES026119 from the National Institute of Environment Health Science (NIEHS) and the National Cancer Institute (NCI), NIH, DHHS)

#5: BP3 and PBDE facilitate metastatic growth of an ER negative breast cancer cell line
Morin SM, Gregory KJ, Ser-Dolansky J, Schwartz R, Chen S, Schneider S

Purpose: BP3 and PBDE have been demonstrated to have agonistic effects on ER+ breast cancer cell proliferation in vitro, which can be inhibited by ICI, indicating an ER dependent effect. In agreement with this, BP3 had a minimal effect on proliferation in the ER negative breast cancer cell line, 4T1, in vitro. In vivo studies are more complex due to estrogen receptors or estrogen related receptors that can be found on some stromal cells, including fibroblasts and immune cells. In fact, these stromal cells can contribute to the metastatic behavior of tumors. Thus, the purpose of this study was to determine whether a pubertal exposure to BP3 or a PBDE mixture could alter the growth or metastasis of 4T1 cells in vivo suggestive of an EDC dependent signal from the stroma to the epithelium.
Methods and Animal Experiments: 4T1 cells were grown in RPMI+10% FBS and treated with 30 μM BP3. MTS assay was used to assess proliferation and Boyden chambers were used to assess invasion. BALB/c mice were treated with BP3 (3 mg/kg and 70 mg/kg body weight/day), the PBDE 47, 100, 153 mixture (1, 0.056, and 0.126 mg/kg bw/day, respectively) or control chow starting at weaning and continuing through puberty to the end of the experiment (n=10/treatment). At 8 weeks of age the animals were injected with 4T1 cells via the lateral tail vein. Organs were harvested and changes in immune populations were analyzed through the use of IHC and RT-QPCR at day 15.
Results: ER negative 4T1 cells treated with BP3 did not demonstrate increased proliferation or invasion in vitro. However, the animals exposed to high levels of BP3 and PBDE during puberty and challenged with intravenous administration of 4T1 cells demonstrated increased size and numbers of metastatic foci in the lungs. While no significant differences were noted in the number of T cells (Cd4+; Cd8+) present across the groups, significant decreases in the FoxP3 T regulatory cell population were observed in the high BP3 and PBDE groups. Analysis of macrophage and stromal populations are ongoing.
Conclusions: Pubertal exposure to BP3 and PBDE resulted in increased growth and metastasis of ER negative cancer cells. Future studies will be aimed at studying the potential contribution of the stromal population.

(Supported by NIEHS BCERP U01 ES026127-01 and the 2018 Opportunity Fund supplement)

#6: Effects of high fat diet and oxybenzone on recrutment of tumor-associated cd206 macrophages
Morozova O, Kariagina A, Haslam SZ, Schwartz RC

Background and Purpose: Previous studies (Zhu Y et al., 2016) found that a diet high in saturated animal fat (HFD) increased the incidence of Trp53-null mammary tumors. Notably, pubertally restricted exposure to HFD promoted tumorigenesis in adulthood. Irrespective of the life stage of exposure, HFD resulted in tumors that showed increased proliferation, vascularity, and recruitment of macrophages. Oxybenzone (benzophenone-3, BP-3), an ingredient in sunscreen and many personal care products, is a putative endocrine disrupting chemical that may alter mammary

gland development and function, and thus may influence mammary tumorigenesis. We are currently studying the interactions between HFD and BP-3 exposure during puberty and adulthood, and its influence on tumorigenesis. In this particular study, we are examining the effects of HFD and BP-3 on M2 macrophage polarization in tumor progression.
Methods: Fragments of mammary glands from 8-week-old Trp53-knockout mice were transplanted to the cleared inguinal fat pads of 3-week-old BALB/c mice. Mouse diets: low fat diet (LFD; 10% kcal fat) +/- BP-3; HFD (60% kcal fat) switched at 10 weeks of age to LFD +/- BP-3 for the entire treatment period; and LFD switched at 10 weeks of age to HFD +/- BP-3 for the entire treatment period. The ingested dose of BP-3 was about 70 mg/kg body weight/day.
Results: Tumors were sectioned and immunofluorescently stained for CD206. CD206-positive cells were enumerated. Number of CD206-positive macrophages is expressed as the mean of CD206-positive cells per image. Quantitation is ongoing. We will present the final results.
Conclusions: Preliminary data show that in epithelial tumors, HFD at either puberty or adulthood significantly increases the number of CD206-positive cells. At the same time, pubertal HFD exposure increases CD206-positive cells to a greater extent than adult exposure. BP-3 exposure in the LFD group shows a trend toward increased CD206-positive macrophages. No significant BP-3 or diet effects are found in spindle cell tumors.

(Supported by Breast Cancer and the Environment Research Program Grant U01ES026119 from the National Institute of Environment Health Science (NIEHS) and the National Cancer Institute (NCI), NIH, DHHS)

#7: Can a state law reduce population-level exposures to breast cancer-relevant chemicals? A case study of California Proposition 65

Ohayon J, Schwarzman M, Dodson R, Knox K, Rudel R, Polsky C

Study Purpose: We are evaluating whether and how a state-based toxics law, Proposition 65, has reduced population-level exposures to chemicals relevant to breast cancer in California and beyond. At the BCERP meeting, we will share preliminary project findings, as well as more broadly present methodological approaches to evaluating the impacts of chemical policies.

Methods: We are analyzing data from CDC's National Health and Nutrition Examination Survey (NHANES) for chemicals for which biomonitoring data is available from both before and after Proposition 65-listing and comparing trends to closely related unlisted chemicals. We conducted >20 interviews with representatives of large manufacturers and retailers to learn about decision-making for selection of consumer product ingredients. Finally, using California Air Resources Board data, we are analyzing shifts over time in product compositions for listed chemicals.
Results: Preliminary analysis of NHANES data suggests that Proposition 65 is impacting population-level chemical exposures. Business interviews underscore that the law directly influences industry (re)formulations through Proposition 65 enforcement actions, as well as indirectly by informing Restricted Substances Lists and increasing supplier transparency. A case study of diesel exhaust suggests several key enforcement actions significantly reduced local-level exposures to diesel particulate matter, while dramatic state-wide reductions in emissions are likely attributable to Proposition 65 enabling California to designate diesel exhaust as a toxic air contaminant. Preliminary data also suggests that private enforcement actions do not always target the most public health-relevant exposures and that Proposition 65 does little to steer informed substitutions in product reformulations.
Conclusion: Our research identifies successful elements of Proposition 65, including its role in driving safer product (re)formulations, and highlights opportunities for improving the law's effectiveness in reducing chemical exposures. The impacts of Proposition 65, including consumer product reformulations, extend beyond California, and state-based chemical policies can thus have public health benefits that are national in scope.

(Supported by the California Breast Cancer Research Program)

#8: The association between sex hormones, pubertal milestones and benzophenone-3 exposure, measured by urinary biomarker or questionnaire

Giannini CM, Huang B, Schwartz RC, Fassler CLS, Biro FM, Chandler DW, Pinney SM

Introduction: Experimental studies have suggested endocrine disruption by benzophenone-3 (BP-3), an active ingredient in sunscreen, with widespread exposure around the world.
Methods: Girls were recruited at ages 6 – 7 years and returned semi-annually for pubertal maturation staging (thelarche, pubarche), and had blood drawn to be analyzed subsequently for serum hormone analyses [estradiol, estrone, testosterone, dehydroepiandrosterone-sulfate (DHEA-S)], and urine for BP-3 assay. Parents completed yearly questionnaires to report participants' past-year sunscreen use and attainment of menarche. Quantile regression was used to determine the relationship between each log-transformed sex hormone with BP-3 exposure, measured by either the BP- 3 urinary biomarker (with half-life of about 16 hours) or reported sunscreen use (use over the last year). Cox-proportional hazards models examined whether BP-3 exposure was associated with age-of-pubertal milestone.
Results: The median value of baseline BP-3 measurements was 25.0 µg/g-creatinine (N=353), with detection in 98.9% of samples. The median number of days sunscreen was used in the past year was 48 days (N=302). There was no evidence of associations between level of the BP-3 biomarker and levels of any of the four hormones. We found a significant negative linear association between amount of reported sunscreen use and testosterone levels during the thelarche window (N=157, adjusted β= -0.0163, 97.5 % CI: -0.0300, -0.0026). There was no evidence of association between amount of reported sunscreen use and levels of other hormones at any time points. The 2nd quartile of the BP-3 biomarker had a greater risk of earlier thelarche compared to the 1st quartile (N=282, adjusted HR=1.584, 97.5% CI: 1.038-2.415). Risk of attaining menarche and pubarche were not different among participants in different biomarker or questionnaire quartiles.
Conclusions: Results suggest that higher report of sunscreen use was associated with lower testosterone levels during thelarche as well as a non-linear relationship between the BP-3 urinary biomarker and the age of thelarche. Exposure assessment captured by sunscreen questionnaire and biomarker are not completely consistent, thus conclusions should be interpreted with caution.

(Supported by U01ES012770, U01ES012771, U01ES012801, U01ES019453, U01ES019454, U01ES019435, U01ES026119, T32GM063483, T32ES010957)

#9: Associations of alcohol and smoking with breast cancer subtypes in the Breast Cancer Association Consortium

Puvanesarajah S, Jung AY, Ahearn TU, Behrens S, Schmidt MK, Chatterjee N, Garcia-Closas M, Terry MB, Chang-Claude J, Gaudet MM

Breast cancer is a heterogeneous disease made up of several etiological subtypes defined by estrogen receptor (ER), progesterone receptor (PR) and HER2. Little is known about modifiable lifestyle factors for the most aggressive subtypes, including non-luminal-HER2+ (ER- and PR- and HER2+) and triple negative (ER- and PR- and HER2-), in part because of their relatively small proportion of all breast cancers. Using 56,835 cases and 73,321 controls from 33 population-based studies in the Breast Cancer Association Consortium (BCAC), we examined associations of alcohol and smoking with subtype-specific breast cancer risk. Alcohol variables studied include cumulative lifetime intake and age at starting regular drinking relative to first full term birth (FFTP). Smoking variables include cigarette smoking in last year (never/former/current) before study entry/ breast cancer diagnosis and age at initiation relative to age at FFTP. Subtypes were defined using two classification schemes: 1) by ER status (ER+, ER-) and 2) by invasive intrinsic-like subtypes [Luminal A (HR+, HER2-, grade 1/2), Luminal B/HER2- (HR+, HER2-, grade 3), Luminal B/HER2+ (HR+, HER2+), non- luminal-HER2+, and triple negative]. In preliminary univariate analyses, associations differed mainly by subtypes defined by ER status. Specifically, we observed increased risk of ER+ cancer when comparing ever vs. never smokers and all categories of drinkers (≤1 drink/day, 1-3 drinks/day, >3 drinks/day) vs. never drinkers. Associations with risk of ER- breast cancer were largely null. Initiation of cigarette smoking years before FFTP was associated with increased risk of breast cancer overall and, specifically, for ER+ cancer.

For example, women who began smoking >10 years before FFTP were 1.41 times (95% CI:1.33-1.50) as likely to develop breast cancer overall and 1.53 times (95% CI:1.42-1.64) as likely to develop ER+ cancer vs. never smokers. Future analyses will further explore intrinsic subtypes using a two-stage multivariate model that captures the correlational structure across the markers and will allow for analysis of rarer subtypes with increased power. These results will direct the study of interactions between these modifiable risk factors and breast cancer susceptibility genes by tumor subtype.

#10: Estrogenic activity following BPA exposure from consumption of canned soup
Reeves KW, Vandenberg L

Bisphenol-A (BPA) activates the estrogen receptor (ER) in both in vitro and in vivo experiments. ER signaling is a potential mechanism by which exposure to this environmental contaminant could promote breast cancer, though human data are lacking. We conducted an innovative pilot study to evaluate the effects of dietary BPA exposure on estrogenicity in healthy, postmenopausal women not taking postmenopausal hormone therapy (n=6). Participants consumed one serving of either boxed (BPA-) soup or canned soup (BPA+) and then provided blood and urine samples approximately 45 minutes after eating. Each experimental condition was administered twice on separate days (total of 4 days). Total and unconjugated BPA was measured in urine samples using high performance liquid chromatography with tandem mass spectrometry. ER activation by plasma samples was measured using a chemically activated luciferase gene expression (CALUX) assay in T47D-KBluc breast cancer cells. Mean urinary BPA concentrations were 30 times higher following consumption of BPA+ versus BPA- soup. Plasma estrogenicity was higher in postmenopausal women following consumption of BPA+ (92.51 pM estradiol equivalents) versus BPA- soup (78.54 pM estradiol equivalents; p=0.04). Our results suggest that BPA exposure from canned soup has an immediate physiologic impact on estrogen signaling. These results demonstrate, for the first time in humans, estrogenic effects associated with BPA exposure. Future studies in larger populations will be useful in establishing this relationship and providing a potential causal pathway by which BPA could increase breast cancer risk.

(Supported by University of Massachusetts Amherst)

Communication Science

#11: Perceptions of breast density in states with and without dense breast notification legislation
Totzkay D, Silk KJ

To better plan for future messaging around breast density and its relation to the cancer-environment connection, two studies were conducted to first capture women's understanding of breast density and then to assess the potential impact of the breast density notifications. In a quasi-experiment between states with and without breast density notification laws, women likely to receive a notification (i.e., aged 40 to 50 years who have recently received a mammogram and have no cancer history) (N=190) reported whether they had heard about breast density and from where they had received that information. Most of the surveyed women reported never hearing about breast density, with no statistically significant difference between states with versus without notification legislation. The women who had heard about breast density reported hearing about it from health care providers or medical staff, interpersonal sources such as coworkers, family, and friends, and mediated sources such as the Internet and the news. Then, an experiment was conducted by exposing a similar sample of women (N=540) either to a mammogram report with a breast density notification embedded or a control mammogram report with no density information. After viewing the notification, women reported a greater intention to speak with their health care provider about breast density, greater self-efficacy regarding that conversation, greater worry about breast density, and more accurate knowledge regarding breast density's complication of mammogram interpretation and that other options were available for cancer screening. Women also believed cancer was present, despite the letter explicitly stating no cancer was found. Taken together, these findings suggest women already know some details about breast density, perhaps in part due to dense breast notifications. However, these data suggest other sources of density information that should be

examined, as well as potential negative consequences of feeling worried after reading the notification. Overall, these data show women already have pre-existing knowledge of breast density and BCERP messaging will need to not only reflect that, but additionally overcome some misperceptions and potentially negative outcomes associated with the experience of worry.

(Supported by the BCERP Opportunity Fund)

Community Outreach and Dissemination

#12: A new model for environmental health literacy about endocrine disrupting chemicals
Boronow KE, Brody JG

Purpose: Endocrine disrupting chemicals (EDCs) in everyday environments and consumer products can interfere with the body's natural hormone signaling. Examples include some pesticides, fragrances, flame retardants, ingredients in plastics and personal care products, and highly fluorinated substances. To make personal and public decisions about EDCs, people need a functional understanding about these chemicals, including where they come from, how they enter the body and affect health, and options for reducing exposure. This project elicited expert insight to define goals for public understanding about EDCs.
Methods: We conducted three focus groups with researchers and community leaders (n = 39) from the Breast Cancer and Environment Research Program and Partnerships for Environmental Public Health networks who conduct research or outreach about EDCs. Focus groups were organized around what "regular" people need to know about six topics to have environmental health literacy about EDCs. Focus group transcripts were coded in Dedoose. Using a mental models approach, we constructed an expert influence diagram for EDCs and distilled focus group discussion into main messages that people need to know to be able to make decisions about EDCs. Results: We identified the following key elements of an action-oriented understanding of EDCs: People encounter EDCs every day in their food, products, and work and home environments. EDCs can affect nearly all the systems of the body and many small exposures can add up to pose a health risk. Exposures to EDCs pose greater risk to health during periods when the body is developing. Individual action can lower some personal exposure to EDCs, and advocacy can change public policy that protects everyone. Currently, chemicals are not tested for safety before they are used in products sold in the US. Information about specific EDCs should be tailored to the exposure situation of the individual or community.
Conclusions: EDC researchers and community partners support communicating a proactive, precautionary approach toward EDC exposure reduction. In addition, communications should focus on correcting misconceptions about how EDCs are regulated in the US, which is a key node influencing personal choices and public engagement related to EDCs.

(Supported by the NIEHS grant R03ES027884)

Mechanisms of Breast Cancer Development

#13: The use of patient-derived breast tissue explants to study resident macrophage polarization and the effects of xenoestrogen exposure
Gregory KJ, Morin SM, Ser-Dolansky J, Schneider SS

Purpose: Genetic variation among women likely contributes to differences in susceptibility to breast cancer. We employed an ex vivo patient derived explant (PDE) system to analyze the impact of EDC exposures with normal complex heterotypic interactions. Of particular interest was the impact of EDCs on macrophages and their cross talk with epithelial cells, as these immune cells are important in ductal/lobular development as well as tumorigenesis. BP3 has been shown to increase the expression genes associated with M2 polarization in mouse studies, so we sought to determine whether BP3 had a similar effect in human tissue. These effects were confirmed using purified human monocytes.
Methods: Fresh human breast tissue and blood was obtained from women who consented to be part of in the Rays of Hope Breast Research Patient Registry. The tissue fragments were placed on Surgifoam gelatin in 30 mm tissue culture dishes. After 24 hours to allow for hormone clearance, the media was supplemented with either vehicle (DMSO/EtOH), 10 nM E2, 30 μM BP3, 10 nM E2 +30 μM BP3, or 3 nM PCB126 for 72 hours. For the macrophage polarization study, explant cultures were treated the same day they were set up with vehicle (DMSO), 100 nM IFNγ + 100 nM LPS, or 20 nM IL-4 + 20 nM IL-13 in the presence and absence of 30 μM BP3 for 72 hrs. The tissues were flash frozen for PCR analysis and formalin fixed and paraffin embedded for IHC analysis.

Monocytes were purified from PBMCs using Stem Cell technology EasySEP monocyte isolation kit. They were grown in RPMI +10% FBS 5 days and treated as above. Results: Estrogen, BP3, and PCB 126 differentially affect target gene expression among women. Additionally, we present a novel finding that we can polarize tissue macrophages within normal breast PDEs towards M1 or M2 as evidenced by elevated expression levels of M1 markers (HLA-DRA and CXCL10) or M2a markers (CD209 and CCL18). Furthermore, BP3 increased and M2 associated gene expression in PDEs and in monocytes. Conclusions: Our PDE model system is an outstanding preclinical model to study the effect of EDC exposures and immune responses. BP3 can affect both epithelial and stromal responses and induces increased secretion of CCL18 and other changes associated with M2 like cells.

(Supported by NIEHS BCERP U01 ES026127-01)

#14: Combined effects of 17β-estradiol (E2), progesterone (P4), and polybrominated diphenyl ethers (PBDEs) on postmenopausal murine mammary glands at the single cell resolution

Saeki K, Chang G, Kanaya N, Wu X, Bernal L, Rakoff M, Neuhausen SL, Chen S

Purpose: Development, maturation and homeostasis of the mammary gland are orchestrated by complex interaction of various cell types in the tissue mainly under control of E2 and P4. PBDEs, as flame retardants in household materials, have been found to bind and modulate the activity of estrogen through the interaction with estrogen receptor (ER). Their persistence in the environment led to great concerns about PBDEs potential endocrine disrupting activity, which includes breast cancer development. However, this presumed causal relationship has not been definitively investigated. In this study, effects of PBDEs, together with E2 and P4 were comprehensively evaluated at the single cell resolution.
Methods: 9-week-old female BALB/cj mice were ovariectomized and treated with E2, P4 and PBDEs (as a mixture of 3 major congeners; BDE-47, -100 and -153) for 1 week, after 10 or 20 weeks from surgical menopause (P10 and P20, respectively). Inguinal mammary glands were collected, digested and sequenced using the single cell RNA sequencing 10x Genomics platform.

Results: A total of 9 major clusters were putatively identified; 2 luminal cell clusters (HR+ and HR-), 1 basal cell cluster, 3 fibroblast clusters characterized by Ccl2, Cxcl14 and Spon1 expression, respectively, an endothelial cell cluster, and 2 hematopoietic cell clusters. Esr1 (ER) expression was found mainly in HR+ luminal and Ccl2+ fibroblasts while Pgr (PR) expression was exclusively in HR+ luminal cells. In the untreated regressed glands, more Ccl2+ fibroblasts were found in P20. Supplementation of E2 dramatically increased the size of both luminal clusters, especially in P20, and addition of E2+P4 significantly expanded the HR- cluster. Exposure to PBDEs in addition to E2+P4 led to increase in HR+ luminal cells although impact on other clusters was variable between P10 and P20.
Conclusion: Our analyses provide valuable insights into mechanism by which mammary glands increase sensitivity to sex hormones with time following menopause as well as P4 stimulates Esr1-/Pgr- putative progenitor expansion. Variability of effect of PBDEs could result from differing states of the gland, which should be further explored by quantitative analyses and by using a chemical (VCD)-induced menopausal transition model.

(Supported by the NIEHS/NCI grant U01 ES026137)

#15: Prenatal PAH induces mammary hyperplasia and altered ERα in grand-offspring adult female mice

Sahay D, Rivera JA, Jezioro JR, Yan B, Szabolcs M, Terry MB, Miller

Polycyclic aromatic hydrocarbons (PAH) are ubiquitous environmental pollutants possessing both carcinogenic and endocrine-disrupting properties. Despite accumulating epidemiological data supporting the association between PAH exposure and breast cancer risk, few experiments have tested the prenatal time window of exposure on subsequent BC risk. We hypothesize that prenatal PAH exposure induces proliferation and cellular hyperplasia in mammary tissue. Second, we propose this may be due to PAH effects on ERα and consequential downstream events, as demonstrated through altered expression and DNA methylation of ERα pathway genes (ERα, PR, p53, AHRR, ARNT, BRCA1) in grand-offspring mammary tissue of adult mice. Pregnant Balb/c dams (F0) were exposed to a nebulized mixture of 9 PAH vs negative control aerosol

5 days a week, for 3 weeks. Female offspring (F1) on postnatal day (PND) 60 were mated with unexposed male mice for 1 week to generate female grand-offspring (F2) mice. On postnatal day (PND) 60, F2 mammary tissues were assessed for histological hyperplasia, atypia and inflammation following H&E staining, and ERα and Ki67 following immunohistochemistry. Reverse transcriptase-real time PCR (for ERα, PR, p53, AHRR, ARNT and BRCA1) and pyrosequencing (ERα and AHRR) in mammary tissues were assessed as well. Prenatal PAH induced more frequent cellular hyperplasia in grand-offsprings compared to controls (Control 1/11 vs. PAH 6/11 mice), but not proliferation, in mammary tissues. Prenatal PAH reduced ERα mRNA levels by 40% ±25%, protein levels by 45% ±29%, and induced higher methylation of several CpG sites at the ERα promoter region by over 40% ±23% when compared to control exposure ($p<0.05$ for each). Prenatal PAH reduced mRNA expression of ER α downstream gene PR ($p<0.05$), but not p53. Prenatal PAH exposure also lowered AHRR mRNA expression by 28% ±19% ($p<0.05$) and induced higher AHRR promoter methylation at CpG-329 ($p<0.05$) by 18% ±11%, but not other sites when compared to control. Finally, prenatal PAH exposure lowered mRNA levels of BRCA1 ($p<0.05$), but not ARNT, when compared to control exposure. Preliminary results suggest that prenatal PAH exposure may induce more frequent mammary cellular hyperplasia, higher methylation of ERα and AHRR,and lower BRCA1 levels.

(Supported by 1U01ES026122)

Special Topics

#16: Barriers and opportunities for breast cancer screening and risk reduction among African American women

Bailey EN, Eggen AT, O'Regan RM, Malecki KMC

Purpose: Little information on women's knowledge of breast cancer risk factors and screening among high risk patients is known, particularly for high risk populations for which this information is often culturally informed. African Americans in Wisconsin, similar to other parts of the country experience higher mortality, later diagnosis and often more aggressive forms of breast cancer. Aims of this study were to assess knowledge of personal breast cancer risk including neighborhood environment context and life experiences, knowledge of and barriers to breast cancer screening that may alter breast cancer risk.
Methods: The focus population was African American women living in areas served by the UW Carbone Cancer Center. A multi-pronged approach was used to partner with community organizations and "county champions" to conduct one focus group in each of six counties followed by a larger quantitative survey (n=250). All focus groups were audio recorded, transcribed and analyzed using NVivo software, and coded inductively for themes. Quantitative survey results are currently being analyzed.
Results: Among the 50 women participating in focus groups, 80% were older than 45 years, the majority (58%) received employee health insurance, 38% received government funded health care, and 32% had a household income <$25,000. Breast cancer was identified among their top eight health concerns. Participants were not knowledgeable about screening guidelines and deferred to their health care providers for health information. Genetics/heredity, chemicals and cleaning supplies, diet and other environmental factors were among the top five possible causes of breast cancer identified in the focus groups. Costs, insurance, transportation and childcare were also among the top reasons cited for lack of access to care and barriers to screening and prevention.
Conclusion: While women are aware of breast cancer screening, they are less knowledgeable about screening guidelines and more severe types of breast cancer. Women are more informed about family risk factors than other environmental factors suggesting opportunities for both primary and secondary prevention in this population. Data will help providers and researchers in developing further risk reduction strategies.

(Supported by the University of Wisconsin Carbone Cancer Center's (UWCCC) Breast Cancer Research Fund, NIH/NCI P30 CA014520- UW Comprehensive Cancer Center Support)

#17: A social and economic systems approach to advance environmental cancer prevention

Hernandez A, Witherspoon N, Jacobs M, Brody J, Hoppin P

Purpose: As scientists learn more about which chemicals can influence breast cancer and other diseases, we need innovative, interdisciplinary efforts to reduce the burden of cancer by eliminating these chemicals and replacing them with safer materials.

To succeed, these efforts require an analysis of the complex social and economic systems that contribute to the production of cancer-causing chemicals. The Cancer Free Economy Network (CFEN) was founded in 2014 to address systems-level cancer prevention. This poster/presentation reports on CFENs system mapping to identify factors that impede or hold promise for a transition to a safer, more equitable economy and identifies strategies for breast cancer scientists and advocates to engage in societal-level prevention.
Methods: A steering committee recruited scientists, environmental advocates, community and worker-based organizations, health professional groups, and green engineering leaders to join CFEN. Through in-person and remote meetings, the Network conducted systems mapping and analysis to set priorities and identify leverage points for cancer prevention.
Results: 45 groups and individuals joined CFEN and organized into three nodes: Health/Science, Building Power, and Market Shift. The systems analysis showed opportunities to strengthen capacity of key constituencies to support environmental carcinogen reduction. Factors impeding progress include lack of training for health professionals, assumptions that government already regulates toxic chemicals, and over-balanced investment in secondary and tertiary prevention. Factors driving progress include new scientific evidence on chemicals, public awareness, and analysis of economic costs. To further address these factors, CFEN developed a training program on chemicals for workers and impacted communities, co-led a webinar with the Bladder Cancer Advocacy Network, and contributed to the first environmental carcinogenesis conference of the American Association for Cancer Research.
Conclusions: System-level changes to increase interdisciplinary research, engage communities, and harness market forces should be prioritized to integrate the reduction of environmental carcinogens into cancer prevention research and practice.

(Supported by the Garfield Foundation)

#18: Epigenetic and metabolic features associated with breast cancer in the Michigan Polybrominated Biphenyl Registry

Marks KJ, Liu C, Kaufman JA, McCullough LE, Curtis SW, Walker DI, Terrell ML, Smith AK, Jones DP, Marder ME, Barr DB, Marcus M

The Michigan Polybrominated Biphenyl (PBB) Registry is a cohort of individuals exposed to high PBB levels via a widespread agricultural contamination in 1973-74. Research on this cohort has revealed endocrine disrupting effects of PBBs on pubertal development, reproductive success, thyroid dysfunction, and breast cancer. In epigenetic analyses, PBBs were associated with steroid hormone pathways, as well as stochastic epigenetic mutations (SEMs) and epigenetic age acceleration, which are both associated with cancer. Untargeted metabolomics analyses suggest PBB associates with mitochondrial energy metabolism (which underlies cancer). Given the evidence suggesting that PBBs could induce carcinogenesis via epigenetic and metabolic changes, this pilot study aims to examine SEMs, age acceleration, and metabolomics among breast cancer cases and cancer-free controls in the exposed population with complete epigenetic and metabolomic data. Cases (n=9) are women from the Registry who report a prior breast cancer diagnosis. Controls (n=28) were a random selection of cancer-free women from the Registry age-matched to cases. We used conditional logistic regression to assess the association of SEMs and age acceleration with breast cancer. The software apLCMS was used for metabolic feature extraction following untargeted high resolution metabolic profiling. Two sample t-tests were utilized for the metabolome-wide association study (MWAS) comparing the metabolic profiles between cases and controls. Median PBB levels were similar among cases and controls (0.28 vs 0.29 ppb). Associations with SEMs and age acceleration measures were null, though increased age acceleration was weakly associated with breast cancer (e.g., intrinsic, odds ratio: 1.07, 95% CI: 0.85, 1.34). No interaction by PBB level was observed in the associations of SEMs and age acceleration with breast cancer. In the MWAS, ferulate was higher in cases by 0.52 standard deviations (SD) (95% CI 0.06- 0.99) and ascorbate was lower in cases by 0.79 SD (95% CI -1.48, -0.10). Neither passed the Benjamini- Hochberg false discovery rate (0.2) after correcting for multiple testing.

While this pilot study is limited by small sample size, there are suggestions of epigenetic and metabolic alterations by breast cancer status.

(Supported by the National Institute of Environmental Health Sciences of the National Institutes of Health under award numbers R01ES025775, R24ES028528, and R01ES024790)

#19: A Breast Cancer Primary Prevention Plan for the State of California

Rasanayagam S, Engel C, Sarantis H, Nudelman J, Buermeyer N

Purpose of the study: We are developing the first-ever comprehensive Breast Cancer Primary Prevention Plan for California. It explores the full range of prevention opportunities, at the individual, community and societal levels, that can contribute to a statewide effort to reduce the incidence of breast cancer.
Methods: We conducted a scoping review of the scientific literature from 2012-2018 on risk factors for breast cancer including environmental exposures to toxic chemicals; exposure to ionizing radiation and other physical factors; barriers to healthy diets and activity levels, such as food deserts and the built environment; workplace exposures; alcohol consumption and tobacco use; and the impact of racism and poverty. We held a series of Listening Sessions around the state to learn from local communities how they experience the identified risk factors and their priorities for action.
Results and Conclusions: The project is on-going, so outcomes and results are not final. While all states have cancer plans, this project offers a unique approach:

• It focuses on societal rather than individual changes. The recommendations address systemic, society-level issues that increase risks for breast cancer. For instance, recommending to individuals that they become more physically active may be less effective than creating safe, walkable neighborhoods with recreational opportunities.

• It incorporates the perspectives of impacted communities into the recommended interventions. California's communities hold valuable information about the issues relevant to their local context and have an intimate understanding of their strengths and barriers to effective change.

• We have a strong commitment to ensure social equity and environmental justice permeate the Plan recommendations.

The process is guided by a multi-stakeholder advisory committee including some of California's leading breast cancer, public health, workplace safety, social and environmental justice experts. The project will generate a California policy agenda to reduce the incidence of breast cancer in the state. BCPP will work with stakeholders to implement aspects of the agenda in the coming years. The Plan will provide a road map for other states and the nation to prioritize primary prevention of breast cancer.

(Supported by California Breast Cancer Research Program Grant # 22QB-7101)

#20: Celebrating the Accomplishments of BCERP

Bird J, Trentham-Dietz A

In 2003, the Breast Cancer and the Environment Research Centers (BCERC) were established to study the impact of pre-pubertal exposures that may affect pubertal development and predispose a woman to breast cancer. During this time, the Puberty Cohort Studies were conducted as a collaborative multi-site epidemiologic study of young girls to investigate and identify predictors of early age at onset of puberty. In 2009, the name of the consortium was changed to the Breast Cancer and the Environment Research Program (BCERP) to reflect an expanded scope including a multidisciplinary network of scientists, clinicians, and community partners to examine the effects of environmental exposures at multiple windows of susceptibility that may predispose a woman to breast cancer. Numerous accomplishments of BCERP members including scientific papers, educational toolkits, and input on policies have resulted in a dramatic expansion of our understanding of the role of the environment in breast cancer risk and steps that women can take to reduce this risk.

Emerging Scholars

#21: To replace toxins linked with breast cancer: the development of antimicrobial sustainable food packaging films utilizing bilayer emulsion compositions

Lin R

Purpose: The purpose of this study is to examine the implementation of varying compositions of poly-γ- glutamic acid (γPGA), chitosan (CH), sophorolipid butyl ester (SLBE), and essential oils to produce a food-safe film with favorable physical, antimicrobial, and mechanical properties. By employing a bilayer structure, these films are capable of replacing common preservatives, such as sulfites, formaldehydes, sorbates, nitrates, and benzoates, which have been associated with adverse health effects. These films could also reduce the need for food packaging and wrappers produced using BPA and PFOA, toxic chemicals linked to breast cancer.
Methods: Bilayer film materials were produced using γ PGA and CH crosslinking between solution and emulsion phases. CH bottom layers and γPGA top layers were cast utilizing varying component concentrations. Vial imaging, microscopy, z-average droplet diameter, zeta potential, viscosity, and pH were analyzed to measure emulsion stability. Imaging, thickness, moisture content, water- solubility, antimicrobial assays, and dynamic mechanical tests were performed to evaluate film properties. These measurements provided an overall assessment of material stability and functionality.
Summary: Bilayer films employing γPGA, CH, SLBE, and oregano oil were prepared and formed with the desired stability, antimicrobial capabilities, and mechanical properties required for a viable food packaging film. By modifying the systems tested, the influences of varying component concentrations were determined, informing the development of a functional and sustainable material.
Conclusions: Oregano oil emulsions generally remained more stable compared to other essential oil emulsions with comparable component concentrations. Bilayer films of different compositions demonstrated varying antimicrobial activity against gram-positive bacteria, fungi, and gram-negative bacteria. Formulation procedures and components similarly influenced the mechanical properties of the films.
The novel materials developed have excellent potential to prevent microbial spoilage, replacing common preservatives and
packaging materials linked with breast cancer. Future research will incorporate antimicrobial peptides such as picidine-3, as well as a coacervation procedure.

(Supported by Great Neck Breast Cancer Coalition, New York State Department of Health Grant)

#22: Developing a safer, biocompatible polymer for tissue engineering which shows potential for breast reconstruction

Liu TC, Gross R

Purpose: The purpose of this study was to develop a safer, biodegradable and biocompatible polymer that can be used as a scaffold for tissue engineering. This unique non-toxic process shows potential for breast reconstruction for breast cancer patients. This was done by improving the properties via molecular weight enhancement of poly(glycerol sebacate) (PGS). PGS is a biodegradable elastomer properties desirable for use as a tissue scaffold. However, pure PGS has a low molecular weight and requires the use of a carrier polymer to allow for electrospinning into fibers, yet these copolymers often manifest characteristics of the carrier over those of PGS. 1,8-octanediol was copolymerized with PGS and catalyzed with Novozym 435 in order to increase molecular weight and allow for electrospinning.
Methods: Poly(1,8-octanediol-glycerol sebacate) (POGS) with glycerol, octanediol, and sebacic acid in ratios of 1:1:2, 1:3:4, and 1:4:5 was synthesized with Novozym 435 in a parallel reactor for 71 hours. Gel permeation chromatography, nuclear magnetic resonance, and differential scanning calorimetry assays were performed on each synthesized product. POGS with the highest molecular weight was electrospun.
Summary: Enzymatically synthesized POGS exhibited markedly higher molecular weights as compared to PGS. 1:3:4 POGS had a significantly higher molecular weight than other POGS ($p<0.05$). 1:3:4 POGS was unable to be electrospun and fibers weren't fully visible with the naked eye. Melting and crystallization temperatures drastically increased with increasing molar ratio of 1,8-octanediol.
Conclusions: The data indicates that utilization of Novozym 435 and copolymerization of 1,8-octanediol improved PGS's thermal and physical properties.

Although 1:3:4 POGS did not produce visible fibers, POGS synthesized in a ratio of 1:2:3 was previously able to be electrospun into fibers. SEM imaging can be done to ascertain the presence of nanofibers in electrospun POGS. Further modification of PGS based materials is a promising area of research for soft tissue engineering, granting ability to manipulate elasticity to match those of tissue. Through developing a non-toxic polymer, POGS scaffolds show potential for breast soft tissue (i.e. glandular or adipose tissue) engineering.

(Supported by New York State Department of Health Grant)

#23: Earthworms (Eisenia fetida) recover from Roundup® exposure

Zafar S, Pochron S, Mirza A, Mezic M, Chung E, Ezedum Z, Gepiraci G, Mari J, Meiselbach C, Shamberger O, Smith R, Tucker W

Pesticides are substances used to inhibit or control an organism's growth. A popular pesticide Roundup® contains an active ingredient of glyphosate, a probable carcinogenic chemical. This weed killer can be found in our drinking water, rain, soil, and food, and may have detrimental effects on general public health and ecosystems. Pesticides are endocrine disrupting chemicals, which can contribute to the development of diseases later in life, which we are particularly susceptible to in certain windows of life, like puberty. Within soil ecosystems, microbes are supported by earthworms when decomposing material such as glyphosate. The combination of microbes, earthworms, glyphosate, and Roundup® form a complex relationship within the soil. By observing the rate at which soil microbes and earthworms can recover from Roundup® exposure can help us understand how the contamination impacts the organisms responsible for degrading the glyphosate. Our experiment was conducted to determine if earthworms could recover from a single contamination event involving a popular Roundup® formulation, as well as to see how the microbe biomass responded. We discovered that earthworms suffered the greatest impact during the first week post exposure, surviving a stress test for 21.2% fewer minutes than did their counterparts living in clean soil. Despite the effects on the earth worms, soil microbe biomass did not respond to contamination in the first week. By the second week, earthworms began to recover, surviving the stress test 12.2% fewer minutes than the control worms. Conversely, microbe biomass drastically decreased by the second week. By the third week, both the worms and the microbes had recovered. Earthworms response to Roundup® preceded the soil microbe response, suggesting that earthworm health drove microbe biomass. Contamination did not impact worm body mass at any time point. Does this elude that the worms adapt? How did their bodies change to do that? What does that say about human exposure? I would like to acknowledge the expertise and supervision of researcher Dr. Sharon Pochran of Stony Brook University throughout the experimental process as well as the abstract submission.

Windows of Susceptibility

#24: Physical activity and breast tissue composition during the adolescent window of susceptibility

Kehm RD, Lilge L, Walter JE, Zeinomar N, Tehranifar P, Herbstman J, Perera F, Miller R, Terry MB

Purpose of the Study: While studies show that adolescent physical activity is associated with reduced breast cancer risk, it is not known if the association is due to long-term changes in breast tissue composition (BTC). In this study, we examined concurrent associations of physical activity with BTC in adolescent girls.
Methods: We studied 144 adolescent girls from the Columbia Center for Children's Environmental Health birth cohort, which recruited African American and Dominican pregnant women from 3 low-income neighborhoods in New York City (1998-2006). As part of Columbia's BCERP Study, girls participated in a follow-up clinic visit in adolescence (ages 11-19, median=15 years) during which they reported by questionnaire average minutes per week of physical education, average minutes per day walking/biking to/from school, and total minutes in the past week and year participating in organized sports and other unorganized activities. At the clinic visit, BTC was measured by optical spectroscopy (OS), a novel and non-invasive tool capturing variation in the amount of water, lipid, oxy-hemoglobin, deoxy-hemoglobin, and collagen, as well as overall cellular and connective tissue density. OS measured red and near-infrared light transmission of 7 wavelengths (650-1060 nm) at 4 source-detector distances in each breast quadrant resulting in 16 overlapping tissue volumes. Principal component analysis was used to reduce spectral data by generating 7 principal component (PC) scores for each participant averaged over both breasts.

We used multivariable linear regression to examine associations adjusted for age, ethnicity, and body mass index (BMI) measured at the time of OS.
Summary of Results: 28% of girls participated in organized sports in the past week, with an average duration of 3.3 hours per week. Any participation in organized sports in the past week (versus none) and duration of past week participation were statistically significantly associated with 3 of the 7 OS PCs ($p<0.05$). These 3 PCs were not associated with BMI and physical activity associations with OS PCs were not modified by BMI.
Statement of Conclusions: Preliminary results support that the association between adolescent physical activity and breast cancer risk might be through changes in BTC.

(Supported by Columbia University site of BCERP - U01ES026122-05)

#25: PFOA and Zeranol exposure during puberty affect the transcriptomic profile of the rat mammary gland
Dang NM, Su Y, Santucci-Pereira J, Russo J

Perfluorooctanoic acid (PFOA) and Zeranol (Zer) are known as endocrine disrupting chemicals (EDCs), however, their potential either alone or in combination of increasing the susceptibility to breast cancer is still not fully understood. Here, we hypothesize that pubertal exposure to these EDCs has an impact on the transcriptomic profile of the mammary glands which explain the less differentiated phenotype and increased susceptibility to carcinogenesis when challenged with a chemical carcinogens like DMBA. Female Sprague-Dawley rats at 21 days of age were assigned to four groups, receiving daily gavage for three weeks with PFOA and Zer alone or in combination, or sesame oil only for control. At 50 days & 100 days of age (d50 & d100, respectively) mammary glands were collected, the RNA was extracted, and a RNA-seq library was constructed and sequenced. We detected differentially expressed genes (DEGs, absolute fold change ≥ 2 & FDRp< 0.05) at d50 by PFOA 136 (106 up-, 30 down-regulated), Zer 394 (370 up, 24 down), and PFOA+Zer 630 (244 up, 386 down); and at d100, PFOA 74 DEGs (23 up, 51 down), Zer 405 (11 up, 394 down), and PFOA+Zer 669 (98 up, 571 down). Zer and PFOA+Zer caused the remarkable number of genes with expression alteration at both of two endpoints. Gene ontology analyses revealed that Zeranol is affecting immune response at d50 but majorly associated with tissue development at d100; whereas PFOA is affecting cell development at d50 but mainly related to metabolic process and morphogenesis at d100. Meanwhile, PFOA+Zer is associated with metabolic process at both of two endpoints. KEGG pathway analysis demonstrated that PFOA+Zer's effects at both d50 & d100 were notably mediated through PI3K-Akt and PPAR signaling pathways. A relevant and significant finding is that each compound induces a different transcriptomic profile, and when both compounds are administered together the transcriptomic profile differs from that induced by each individual one. The major EDCs affected pathways are PI3K-Akt and PPAR signaling indicating that the long term damaging effect of the combination of EDCs explain the less differentiated phenotype and its higher susceptibility to develop mammary cancer when challenged with a chemical carcinogen.

(Supported by the NIEHS/NCI grant U01ES026130 and NCI/NIH Cancer Center Support Grant P30-CA006927)

#27: Immune response of the rat mammary gland to the pubertal exposure to PFOA+ZERANOL
Nelson C, Hrdy B , Singh B, Rogers CW, Cox N, Grimaldi P, Steele S, Su Y, Santucci-Pereira J, Dang NM, Ser-Dolansky J, Schneider S, Russo J

Background: Perfluorooctanoic acid (PFOA) and Zeranol are known as endocrine disrupting chemicals (EDCs), however, their potential combination of affecting the immune response of mammary gland is still not fully understood. In this study, we aimed to determine the effect of pubertal exposure to PFOA+Zeranol on the immune responses in rat mammary glands.
Methods: Sprague Dawley rats during the pubertal period, 21 and 42 days of age, were treated by gavage with PFOA+Zeranol dissolved in sesame oil. A control group was only gavaged with the solvent. Mammary glands of seven randomly selected rats of each group at the age of 50 days were collected and formalin-fixed.

Sections (4-µm) of paraffin-embedded tissue were used for immunohistochemistry staining against rabbit monoclonal anti-CD3 (1:100), rat monoclonal anti-mouse CD8a (1:100), rabbit polyclonal anti-CD68 (1:100) and chloroacetate esterase (CAE) for mast cell, performed on a DakoCytomation autostainer using the Envision HRP Detection system (Dako, Carpinteria, CA) at Baystate Medical Center. Digitalized images were taken using Vectra® Imaging System, Stained cells were counted using the scale bar provided by CellSense Entry's software and area correction by ImageJ. Data were statistically analyzed by Sigmaplot version 12.
Results: In total, 20,582 images for four different staining against CD3, CD8, CD68 Abs and mast cells were analyzed with 55,601 counted stained cells. Number of CD8a positive cells in the group exposed to PFOA+Zeranol was 4.01 times increased (12.976 cells/mm2 for PFOA+Zeranol vs 3.231 cells/mm2 for control, $p<0.01$, 3054 images). A 2.4 times higher number of CD3 T-cells were also found in PFOA+Zer treated group in comparison with control rats (3.832 cells/mm2 for PFOA+Zeranol vs 1.571 cells/mm2 for control, $p=0.073$, 5761 images). No significant differences of CD68 positive cells or mast cells between treated and control groups were observed.
Conclusion: The data presented are showing that the combined exposure of PFOA and Zeranol in pubertal rats increase the CD8a and total T-cells in the mammary gland indicating that the EDC are eliciting a cell-mediated immune response.

(Supported by the NIEHS/NCI grant U01ES026130 and NCI/NIH Cancer Center Support Grant P30-CA006927)

#29: Pubertal exposure to different doses of Zeranol induces unique changes in the transcriptomic profile of rat mammary glands
Dang NM, Gallagher D, Su Y, Santucci-Pereira J, Russo J

Background: Puberty in human and animals has been identified as a window of susceptibility to endocrine disrupting chemicals. Zeranol, a synthetic non-steroid estrogen is widely used as a growth promoter in the beef industry in the United States. In this study we are determining the effect of pubertal exposure to different doses of Zeranol in the transcriptomic profile of the rat mammary glands.
Methods: Sprague Dawley rats were gavaged daily during the critical pubertal window (21 to 42 days of age) with sesame oil as a control, or a low dose of zeranol (0.01 mg/kg), or a high dose of zeranol (0.1 mg/kg). After the rats had reached an age of 100 days, isolated RNA from rat mammary glands were used for library construction and RNA sequencing using Illumina platform.
Results: There were 401 genes that exhibited a 2 fold change from the Zeranol low dose exposed animals compared with the control group. Of these, 17 were up-regulated and 384 were down-regulated. In the animals exposed to the higher dose 1134 genes were found to have a significant difference with the control and 281 of these were up regulated and 853 were down-regulated. Gene ontology enrichment showed that Zeranol at low dose is affecting down-regulated genes related to tissue development while the high dose of Zeranol is up-regulating genes associated with immune-inflammatory and cell activation and migration; and down-regulating genes mainly related to the cell cycle and DNA metabolic process. KEGG pathway analysis indicated that calcium and CGMP-PKG signaling pathways are affected by low dose exposure to Zeranol while the P53 signaling pathway and DNA repair are down-regulated by high doses of Zeranol.
Conclusion: Pubertal exposure to Zeranol at different doses induce different transcriptomic profiles that are detected in 100 day old animals, indicating the long term effect of this agent in a variety of molecular pathways and particularly with DNA repair impairment at high dose exposure.

(Supported by the NIEHS/NCI grant U01ES026130 and NCI/NIH Cancer Center Support Grant P30-CA006927)

#31: Butyl benzyl phthalate and Perfluorooctanoic acid induce long term impact on the gene expression profile of mammary gland in exposed rats during puberty
Dang NM, Byrne R, Su Y, Santucci-Pereira J, Russo J

Background: Butyl benzyl phthalate (BBP), a plasticizer, and Perfluorooctanoic acid (PFOA), a surfactant are known environmental disruptors. Mammary gland development takes place during puberty that is considered a critical window of cancer susceptibility.

In this study we are using BBP and PFOA alone or in combination at the lower level of exposure found in human to determine if pubertal exposure induces long term effect in the transcriptomic profile of the rat mammary gland.
Methods: Sprague Dawley rats were gavaged daily during the critical pubertal window (21 to 42 days of age) with sesame oil as a control or BBP (0.5 mg/kg of body weight) and PFOA (0.01 mg/kg of body weight), either alone or in combination. When the rats had reached the age of 100 days, RNA from the mammary glands was extracted for library construction and RNA sequencing using Illumina platform.
Results: Combination of BBP and PFOA exposure affect 1123 genes, 403 of them are up- and 720 are down-regulated. Instead BBP alone affect 213 genes, 152 are up- and 61 are down-regulated, whereas PFOA affect 96 genes, 53 are up- and 43 are down-regulated. Up-regulated genes induced by BBP+PFOA are involved in the regulation of the cell differentiation, immune response and negative regulation of RNA transcription while the down-regulated genes are found to be related to muscle structure and function. BBP alone induces the up-regulation of genes mainly associated with tissue development and cell adhesion while PFOA causes the up-regulation of genes involved with metabolic process, morphogenesis, immune system, and no significant process was found for down-regulated genes. KEGG pathway analysis shows that BBP+PFOA affect pathways associated with ribosomes and biosynthesis of amino acids while BBP affects up-regulation of genes related to the estrogen and p53 signaling pathways and PFOA affects up-regulated genes involved in rap1, CAMP, H1F-1 signaling pathways.
Conclusion: The transcriptomic profile of the rat mammary gland of animals exposed with BBP and PFOA alone or in combination at puberty clearly demonstrate that the changes are long lasting and that the effect is more pronounced and affecting different molecular pathways when combination of environmental agents are used.

(Supported by the NIEHS/NCI grant U01ES026130 and NCI/NIH Cancer Center Support Grant P30-CA006927)

#33: Single or combined butyl benzyl phthalate (BBP) and Zeranol exposure during puberty affect the expression profile in the rat mammary gland at older age

Gift O, Dang NH, Su Y, Pereira JS, Russo J

Background: Butyl benzyl phthalate (BBP) used as plasticizer, and Zeranol used in the beef industry to induce cattle growth have been found to be environmental disruptors affecting both human and animals. Puberty is a crucial time for breast development and is considered a window of susceptibility to carcinogenesis. In this study we are using BBP and Zeranol alone or in combination at the lower level of exposure found in human to determine if pubertal exposure induces long term effect in the transcriptomic profile of the rat mammary gland.
Methods: Sprague Dawley rats were gavaged daily during puberty (21 to 42 days of age) with sesame oil as a control or Zeranol (0.01 mg/kg) and BBP (0.5 mg/kg), either alone or in combination. When the rats had reached the age of 100 days, RNA from the mammary glands was extracted for library construction and RNA sequencing.
Results: Combination exposure of BBP and Zeranol had 970 genes affected and 709 of those genes were down-regulated. BBP and Zeranol alone affect 214 and 402 genes respectively. GO analysis revealed that up-regulated genes induced by BBP were related to cell development & differentiation, adhesion, and cell-cell junction organization while Zeranol induced gene expression alteration associated with tissue development. KEGG analysis revealed that these gene expression changes induced by BBP were associated with estrogen signaling pathway while calcium and CGMP-PKG signaling pathways are affected by exposure to Zeranol. BBP+Zer caused the up-regulated genes associated with the metabolic process while actin filament-based processes, muscle structure development, and transmembrane transport regulation were associated with down-regulated genes. KEGG analysis showed that up-regulated genes induced by BBP+Zer were majorly associated with metabolic processes, complement-coagulation cascades & PPAR signaling whereas down-regulated genes are shown to be associated with the MAPK, calcium and CGMP-PKG signaling pathway.

Conclusions: The transcriptomic profile of the rat mammary gland of animals exposed with BBP and Zeranol alone or in combination at puberty clearly demonstrate that the changes are long lasting and that the effect of the combination is more pronounced and affecting different molecular pathways.

(Supported by the NIEHS/NCI grant U01ES026130 and NCI/NIH Cancer Center Support Grant P30-CA006927)

Participant List

Emily Adam
ORISE fellow at Centers for Disease Control and Prevention
Brookhaven, GA
emily-adam@uiowa.edu

Lucile Adams-Campbell
Georgetown University
Washington, DC
lla9@georgetown.edu

Alexandrina Balanean
Georgia State University
Decatur, GA
abalanean1@student.gsu.edu

Erin Bailey-Winston
University of Wisconsin Carbone Cancer Center
Madison, WI
enbailey@wisc.edu

Jennifer Bird
University of Wisconsin -Madison
Madison, WI
jbird@wisc.edu

Frank Biro
Cincinnati Children's Hospital Medical Center
Cincinnati, OH
frank.biro@cchmc.org

Amye Black
University of Massachusetts Amherst
Amherst, MA
alblack@umass.edu

Katherine Boronow
Silent Spring Institute
Newton, MA
boronow@silentspring.org

Abee Boyles
National Institute of Environmental Health Sciences, National Institutes of Health
Durham, NC
abee.boyles@nih.gov

Julia Brody
Silent Spring Institute
Newton, MA
brody@silentspring.org

Kimberly Burke
Columbia University
New York, NY
krb2160@cumc.columbia.edu

Celia Byrne
Uniformed Services University of the Health Sciences
Bethesda, MD
celia.byrne@usuhs.edu

Bethsaida Cardona
Silent Spring Institute
Newton, MA
cardona@silentspring.org

Gregory Chang
City of Hope
Duarte, CA
gregchang@coh.org

Shiuan Chen
City of Hope
Arcadia, CA
schen@coh.org

Gwen Collman
National Institute of Environmental Health Sciences
Durham, NC
collman@niehs.nih.gov

Nhi Dang
Fox Chase Cancer Center
Philadelphia, PA
nhi.dang@fccc.edu

Melissa Davis
Weill Cornell Medicine
New York, NY
mbd4001@med.cornell.edu

Caroline Dilworth
Thrive Integrative Wellness Coaching
Chapel Hill, NC
caroline.dilworth@gmail.com

Yuan C Ding
City of Hope
Duarte, CA
ycding@coh.org

Karen Dunphy
University of Massachusetts Amherst
Amherst, MA
kdunphy@vasci.umass.edu

Calvin Ellison
Pink Panel Initiative
Stone Mountain, GA
Columbia University
dr.calellison@gmail.com

Gary Ellison
National Cancer Institute
Bethesda, MD
ellisong@mail.nih.gov

Shaoqi Fan
National Cancer Institute
Rockville, MD
shaoqi.fan@nih.gov

Cecily Fassler
University of Cincinnati
Cincinnati, OH
shimpcl@mail.uc.edu

Stacey Fedewa
American Cancer Society
Atlanta, GA
stacey.fedewa@cancer.org

Ronald Gangnon
University of Wisconsin-Madison
Madison, WI
ronald@biostat.wisc.edu

Mia M. Gaudet
American Cancer Society
Atlanta, GA
mia.gaudet@cancer.org

Armen Ghazarian
National Cancer Institute
Rockville, MD
armen.ghazarian@nih.gov

Participant List

Owen Gift
Fox Chase Cancer Center
Pottstown, PA
oeg26@drexel.edu

Ann Goding Sauer
American Cancer Society
Atlanta, GA
ann.godingsauer@cancer.org

Amelia Grant-Alfieri
Columbia University
Ann Arbor, MI
amelia.grant.alfieri@gmail.com

Susanna Greer
American Cancer Society
Atlanta, GA
Susanna.greer@cancer.org

Kelly Gregory
Pioneer Valley Life Sciences Institute-Baystate Medical Center
Springfield, MA
kelly.gregory@baystatehealth.org

Mary Gullatte
Emory Healthcare and Winship Cancer Institute of Emory University
Atlanta, GA
mfgulla@emory.edu

Rhonda Hamilton
Georgetown University
Washington, DC
rnh12@georgetown.edu

Alison Harrill
National Institute of Environmental Health Sciences
Chapel Hill, NC
alison.harrill@nih.gov

Jaime Hart
Brigham and Women's Hospital & Harvard Medical School; Harvard TH Chan School of Public Health
Boston, MA
jaime.hart@channing.harvard.edu

Terry Hartman
Emory University
Atlanta, GA
tjhartm@emory.edu

Tabia Henry Akintobi
Morehouse School of Medicine
Atlanta, GA
takintobi@msm.edu

Amanda Hernandez
Silent Spring Institute
Newton, MA
hernandez@silentspring.org

Shuk-Mei Ho
University of Arkansas for Medical Sciences
Little Rock, AR
shukmeiho@uams.edu

Dawn Holman
Centers for Disease Control and Prevention, Division of Cancer Prevention and Control
Chamblee, GA
dholman@cdc.gov

Reyhane Hoshyar
Michigan State University
East Lansing, MI
hooshyar@msu.edu

Joseph Jerry
University of Massachusetts Amherst
Amherst, MA
jjerry@vasci.umass.edu

Anastasia Kariagina
Michigan State University
East Lansing, MI
kariagin@msu.edu

John Kaufman
Emory University
Atlanta, GA
jakauf2@emory.edu

Rebecca Kehm
Columbia University
New York, NY
rk2967@cumc.columbia.edu

Tram Kim Lam
National Cancer Institute
Rockville, MD
lamt@mail.nih.gov

Teri Larkins
Centers for Disease Control and Prevention
Atlanta, GA
tlarkins@cdc.gov

Lisa Levine
Great Neck Breast Cancer Coalition
New York, NY
nutritiontransition@gmail.com

Raymond Lin
Great Neck Breast Cancer Coalition
Great Neck, NY
raymondjlin@gmail.com

John Lin
Great Neck Breast Cancer Coalition
Great Neck, NY
jehlin@hotmail.com

Chang Liu
Emory University
Decatur, GA
chang.liu2@emory.edu

Timothy Liu
Huntington Breast Cancer Action Coalition
Melville, NY
friends@hbcac.org

Susan Lloyd
Columbia University Mailman School of Public Health
New York, NY
sl4279@cumc.columbia.edu

Grace Makari-Judson
Rays of Hope Center for Breast Cancer Research/ Baystate Regional Cancer Program
Springfield, MA
grace.makari-judson@baystatehealth.org

Participant List

Kristen Malecki
University of Wisconsin- Madison
Madison, WI
kmalecki@wisc.edu

Michele Marcus
Emory University
Atlanta, GA
mmarcus@emory.edu

Kristin Marks
Emory University
Decatur, GA
kristin.marks@emory.edu

Polly Marshall
Breast Cancer Over Time
San Francisco, CA
polly@breastcancerovertime.org

Mary Beth Martin
Georgetown University
Washington, DC
martinmb@georgetown.edu

Julie McGregor
University of Wisconsin - Madison
Madison, WI
jmcgregor@wisc.edu

Eileen McGuine
University of Wisconsin -Madison
Madison, WI
emcguine@wisc.edu

Karin Michels
University of California, Los Angeles
Los Angeles, CA
k.michels@ucla.edu

Karen Miller
Huntington Breast Cancer Action Coalition (HBCAC)
Melville, NY
friends@hbcac.org

Rachel Miller
Columbia University
New York, NY
rachel.miller2@mssm.edu

Stephanie Morin
University of Massachusetts Amherst
South Hadley, MA
smorin@umass.edu

Olena Morozova
Michigan State University
East Lansing, MI
morozov1@msu.edu

Leah Moubadder
Emory University Rollins School of Public Health
Decatur, GA
lmoubad@emory.edu

Sherieda Muthra
Georgetown University
Washington, DC
STM36@Georgetown.edu

Susan Neuhausen
Beckman Research Institute of City of Hope
Duarte, CA
sneuhausen@coh.org

Jennifer Ohayon
Silent Spring Institute
Newton, MA
ohayon@silentspring.org

Lucy Peipins
Centers for Disease Control and Prevention
Chamblee, GA
LBP6@CDC.GOV

Ana Pereira
Institute of Nutrition and Food Technology, University of Chile
Santiago, RM
anitapereiras@gmail.com

Susan Pinney
University of Cincinnati College of Medicine
Cincinnati, OH
susan.pinney@uc.edu

Samantha Puvanesarajah
American Cancer Society
Atlanta, GA
samantha.puvanesarajah@cancer.org

Michele Rakoff
City of Hope
Beverly Hills, CA
rakoffm@yahoo.com

Sharima Rasanayagam
Breast Cancer Prevention Partners (BCPP)
San Francisco, CA
sharima@bcpp.org

Katherine Reeves
University of Massachusetts Amherst
Amherst, MA
kwreeves@umass.edu

Les Reinlib
National Institute of Environmental Health Sciences
Research Triangle Park, NC
reinlib@niehs.nih.gov

Peggy Reynolds
University of California, San Francisco
Berkeley, CA
peggy.reynolds@ucsf.edu

Kim Robien
Milken Institute School of Public Health, George Washington University
Washington, DC
krobien@gwu.edu

Ruthann Rudel
Silent Spring Institute
Newton, MA
rudel@silentspring.org

Kohei Saeki
City of Hope
Monrovia, CA
ksaeki@coh.org

Debashish Sahay
Columbia University Medical Center
New York City, NY
sd3057@cumc.columbia.edu

Participant List

Sallie S. Schneider
Pioneer Valley Life Sciences Institute
/Baystate Medical Center
Springfield, MA
sallie.schneider@baystatehealth.org

Richard Schwartz
Michigan State University
East Lansing, MI
schwart9@msu.edu

Jim Shull
University of Wisconsin-Madison
Madison, WI
shull@oncology.wisc.edu

Kami Silk
University of Delaware
Newark, DE
kamisilk@udel.edu

Anna G. Symington
University of Massachusetts Amherst
South Hadley, MA
asannagee33@gmail.com

Susan Teitelbaum
Icahn School of Medicine at Mount Sinai
New York, NY
susan.teitelbaum@mssm.edu

Mary Beth Terry
Columbia University
New York, NY
mt146@columbia.edu

Brandon Thomas
Michigan State University
Lansing, MI
Brandondht@gmail.com

Claudia Thompson
National Institute of Environmental
Health Sciences
Morrisville, NC
thomps14@niehs.nih.gov

Daniel Totzkay
West Virginia University
Morgantown, WV
daniel.totzkay@mail.wvu.edu

Mary Turyk
University of Illinois at Chicago
Chicago, IL
mturyk1@uic.edu

Amy Trentham-Dietz
University of Wisconsin-Madison
Madison, WI
trentham@wisc.edu

Claudia Vredeveld
University of Wisconsin-Madison
Madison, WI
vredeveld@wisc.edu

Desiree Walker
Columbia University
New York, NY
healthadvocateny@gmail.com

Susan Webb
Prisma Health Upstate
Clemson, SC
susan.webb@prismahealth.org

Laura Weinberg
Great Neck Breast Cancer Coalition
Great Neck, NY
lpw513@gmail.com

Mary White
Centers for Disease Control and
Prevention (CDC)
Chamblee, GA
mxw5@cdc.gov

Alexandra White
National Institute of Environmental
Health Sciences
Research Triangle Park, NC
alexandra.white@nih.gov

Kevin Wright
George Mason University
Springfield, VA
kwrigh16@gmu.edu

Subhana Zafar
Huntington Breast Cancer Action
Coalition
Melville, NY
friends@hbcac.org

Notes

Notes

Notes

Local Restaurant Suggestions

Pittypat's Porch
0.1 miles from The American Hotel
"Southern comfort food, a rocking chair lounge, & a full bar with mint juleps in a quaint space."
Phone: (404) 525-8228
Hours: 4:30 PM – 9 PM
pittypatsrestaurant.com

Aviva by Kameel
0.1 miles from The American Hotel
225 Peachtree St NE, Atlanta, GA 30303
"Easygoing counter-serve cafe & juice bar providing locally sourced, classic Mediterranean meals.
Phone: (404) 698-3600
Hours: 7:30 AM – 4 PM
avivabykameel.com

Sun Dial
0.1 miles from The American Hotel
210 Peachtree St NW, Atlanta, GA 30303
"Spinning hotel restaurant & bar with 360-degree views of the city & frequent live jazz."
Phone: (404) 589-7506
Hours: 11:30 AM – 2:30 PM, 6 PM – 10 PM
sundialrestaurant.com

Aamar Indian Cuisine
0.2 miles from The American Hotel
100 Luckie St NW, Atlanta, GA 30303
"Tiny, counter-serve institution with unpretentious Indian dishes, plus late hours & outdoor seating."
Phone: (404) 257-6959
Hours: 4 PM – 11:30 PM
aamarindianatlanta.com

White Oak Kitchen & Cocktails
0.3 miles from The American Hotel
270 Peachtree St NW, 100, Atlanta, GA 30303
"Airy, modern-rustic hangout serving Southern-tinged New American fare & specialty cocktails"
Phone: (404) 524-7200
Hours: 11:30 AM – 10:30 PM
whiteoakkitchen.com

Polaris
0.3 miles from The American Hotel
265 Peachtree St, Atlanta, GA 30303
"Iconic rotating restaurant featuring a retro peach daiquiri & Southern fare in sleek environs."
Phone: (404) 460-6425
Hours: 5 PM – 12 AM
polarisatlanta.com

Taqueria on Broad
0.4 miles from The American Hotel
54 Broad St NW, Atlanta, GA 30303
"Warm, rustic wood-lined stop for quick-serve Mexican tacos, burritos & a range of Jarritos."
Phone: (678) 732-0693
Hours: 10AM – 6PM
taqueriaonbroad.com

Dua Vietnamese Noodle Soup
0.4 miles from The American Hotel
53 Broad St NW, Atlanta, GA 30303
"Cozy, casual Vietnamese stop with a colorful space is known for quick-serve pho & lemongrass tofu."
Phone: (404) 589-8889
Hours: 10 AM – 4 PM

Blossom Tree
0.4 miles from The American Hotel
64 Peachtree St NW, Atlanta, GA 30303
"Casual Korean kitchen dishing up tacos with Asian-inspired fillings, plus familiar entrees & sides."
Phone: (404) 223-7500
Hours: 10:30 AM – 7:30 PM
blossomtreeatl.com

Ponce City Market
2 miles from The American Hotel
675 Ponce De Leon Ave NE, Atlanta, GA 30308
"Vibrant converted historic Sears building, now a market with a food hall, shops & living space."
Phone: (404) 900-7900
Hours: 10 AM – 9 PM
poncecitymarket.com

Ted's Montana Grill
0.1 miles from The American Hotel
133 Luckie St NW, Atlanta, GA 30303
"Old West-style steakhouse chain known for its burgers & chops made from bison."
Phone: (404) 521-9796
Hours: 11 AM – 10 PM
tedsmontanagrill.com

Site-seeing Attractions

World of Coca-Cola
0.4 miles from The American Hotel
121 Baker St NW, Atlanta, GA 30313
"Memorabilia, films & exhibits (some interactive) about soft drink that debuted in Atlanta in 1886"
Phone: (404) 676-5151
Hours: 9 AM – 5:30 PM
worldofcoca-cola.com

Georgia Aquarium
0.7 miles from The American Hotel
225 Baker St NW, Atlanta, GA 30313
"One of world's largest aquariums, with permanent exhibits, interactive galleries & animals galore."
Phone: (404) 581-4000
Hours: 10 AM – 9 PM
georgiaaquarium.org

Centennial Olympic Park
0.3 miles from The American Hotel
265 Park Ave W NW, Atlanta, GA 30313
"Home of the 1996 Olympic games now filled with walking paths, water gardens & the Fountain of Rings."
Phone: (404) 223-4412
Hours: 7 AM – 11 PM
gwcca.org

SkyView Atlanta
0.3 miles from The American Hotel
168 Luckie St NW, Atlanta, GA 30303
"20-story Ferris wheel in Centennial Park providing scenic views from climate-controlled gondolas"
Phone: (678) 949-9023
Hours: 12 PM – 11 PM
skyviewatlanta.com

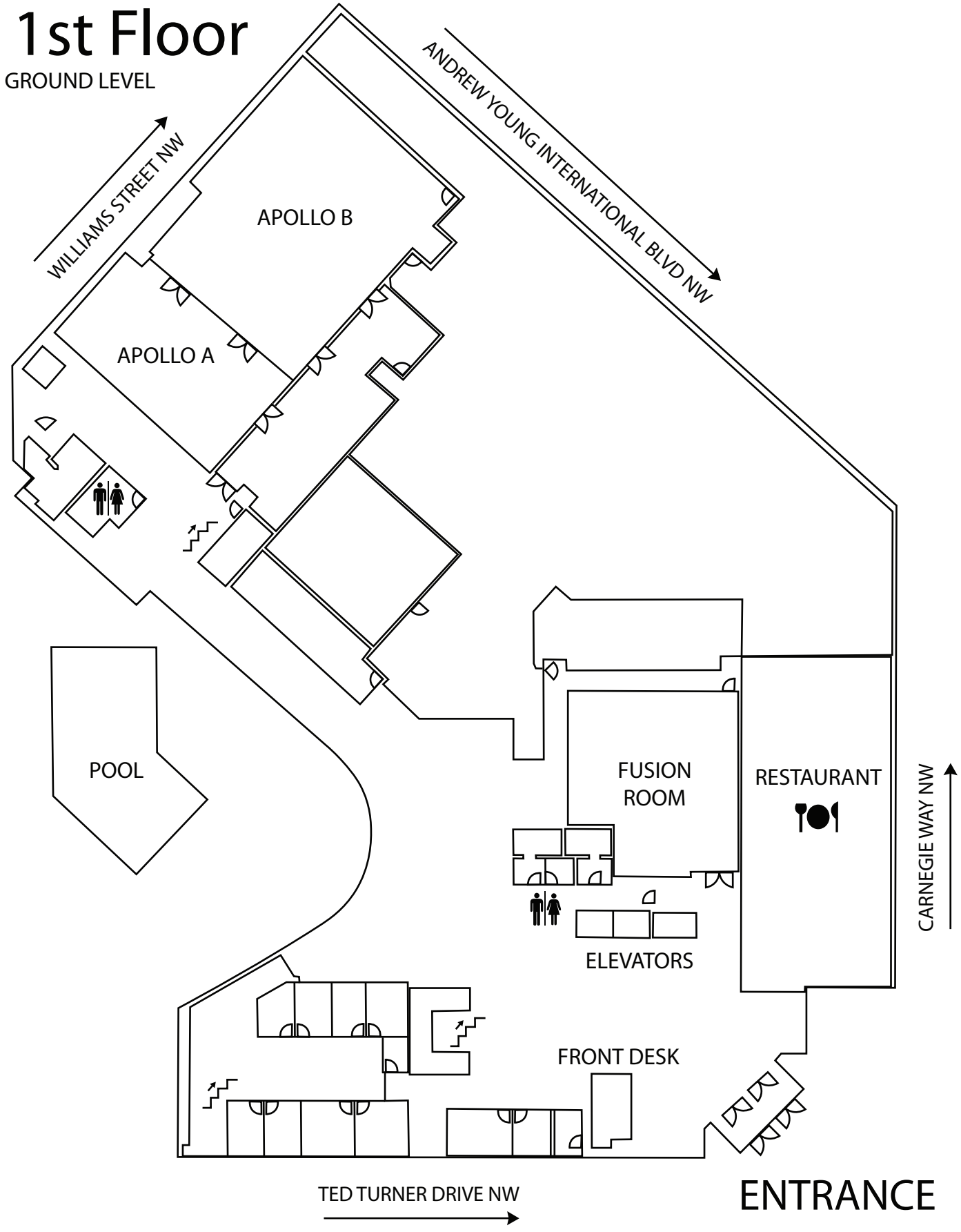
Fox Theatre
1.1 miles from The American Hotel
660 Peachtree St NE, Atlanta, GA 30308
"Restored opulent theater built in 1920s with 3,600-pipe organ hosts ballet, movies & other events"
Phone: (404) 881-2100
Foxtheatre.org

Underground Atlanta
0.6 miles from The American Hotel
50 Upper Alabama St, Atlanta, GA 30303
"This historic area is now a shopping & entertainment center with eateries, events & tours."
Phone: (404) 963-5415
Hours: 10 AM – 7 PM
undergroundatl.com

National Center for Civil and Human Rights
0.6 miles from The American Hotel
100 Ivan Allen Jr Blvd NW, Atlanta, GA 30313
"Cultural attraction presents artifacts, art & photos from the Civil Rights Era & about human rights."
Phone: (678) 999-8990
Hours: 10 AM – 5 PM
civilandhumanrights.org

College Football Hall of Fame
0.6 miles from The American Hotel
250 Marietta St NW, Atlanta, GA 30313
"Sleek modern museum offers exhibits on famous college football players & an indoor field for events."
Phone: (404) 880-4800
Hours: 10 AM – 5 PM
cfbhall.com

High Museum of Art
2.3 miles from The American Hotel
1280 Peachtree St NE, Atlanta, GA 30309
"Museum known for its diverse art collection & modern architecture by Richard Meier & Renzo Piano."
Phone: (404) 733-4400
Hours: 10 AM – 5 PM
high.org


1st Floor
GROUND LEVEL
WILLIAMS STREET NW
ANDREW YOUNG INTERNATIONAL BLVD NW
APOLLO B
APOLLO A
POOL
FUSION ROOM
RESTAURANT
CARNEGIE WAY NW
ELEVATORS
FRONT DESK
TED TURNER DRIVE NW
ENTRANCE

2019 BCERP Annual Meeting Program At a Glance

Detailed program agenda on pages 5-8

Thursday, November 7, 2019
7:00 a.m.-5:00 p.m. Registration

8:00-8:15 a.m. **Welcome and Introduction of Keynote Speaker - Apollo B**

8:15-9:00 a.m. **Keynote Address - Apollo B**
The BCERP legacy: windows of susceptibility to environmental risks of disease

9:00-10:30 a.m. **Session 1: Myths, Messages, and Communicating Uncertainty - Apollo B**
- "Breast Cancer Survivors Lend Their Voices" video
- Why me? Conversations with women newly diagnosed with breast cancer
- Advancing cancer health equity through strategic community-engagement- designing population and implementation research that matters
- Communicating BCERP science from the lens of uncertainty theories

10:30-10:45 a.m. Break

10:45 a.m.-12:15 p.m. **Session 2: Interactions between Environmental Chemicals and Inherited Risk - Apollo B**
- Using the diversity outbred mice to identify gene by environment interactions
- Detecting gene by environment interactions in population studies
- Why genetic, environment, and epigenetic regulation matter to breast cancer risk
- On the need to consider both genetic and windows of susceptibility for environmental exposures and breast cancer risk with the specific example of PAHs

12:15-1:45 p.m. **Lunch – Apollo B**

1:45-3:15 p.m. **Poster Session – Apollo A**

3:15-3:30 p.m. Break

3:30-5:00 p.m. **Session 3: Emerging Research - Apollo B**
- Barriers and opportunities for breast cancer organizations to focus on environmental health and disease prevention: a mixed-methods approach using website analyses, interviews, and focus groups
- Evaluation results of BCERP continuing medical education for pediatric health care professionals
- Hair dye and chemical straighteners in relation to breast cancer risk in a large US population of black and white women
- Effect of oxybenzone exposure during pregnancy and lactation on the protective effect of parity on mammary cancer in mice with p53 -/- epithelium
- Combined effects of 17β-estradiol (E2), progesterone (P4), and polybrominated diphenyl ethers (PBDEs) on postmenopausal murine mammary glands at the single cell resolution
- Hormone phenotypes defined in peripubertal girls, a novel approach using principal components and cluster analysis in a longitudinal cohort

5:00-6:30 p.m. **Session 4: A Look Back and a Look Forward - The Impact of the BCERP - Apollo B**

Friday, November 8, 2019
7:30 a.m.-12:30 p.m. Registration

8:30-10:00 a.m. **Session 5: Addressing Risk Posed by Mixtures of Chemicals - Apollo B**
- New methods for analyzing mixtures in population based research, Findings from the PRIME network
- Multiple exposures to chemicals with biologic persistence do influence the levels of some reproductive hormones during female puberty
- Multiple chemical exposures and breast cancer risk, findings from and the California Teacher's Study
- Effects of estrogen, progesterone, and PBDEs on mammary gland structure after surgical menopause

10:00-10:15 a.m. Break

10:15-11:45 a.m. **Session 6: Environmental Chemicals, Metabolism and Immune Function and Breast Cancer Risk - Apollo B**
- How the breast environment may influence breast cancer risk
- Effect of exercise on metabolic syndrome in black women by family history and predicted risk of breast cancer: The FIERCE Study
- Endocrine disrupting chemicals and the immune system: a possible mechanism for facilitated cancer progression
- African ancestry and tumor immune responses leading to disparate clinical outcomes

11:45 a.m.-12:30 p.m. **Announcement of Poster Awards and Closing Remarks by Gwen Collman, Director, Extramural Research & Training, NIEHS – Apollo B**

12:30 p.m. Meeting Adjourned