Health Care Appropriations Subcommittee

November 17, 2015
1:00 PM – 3:00 PM
Webster Hall (212 Knott)

Action Packet

Steve Crisafulli
Speaker

Matt Hudson
Chair

Committee Meeting Notice

HOUSE OF REPRESENTATIVES

Health Care Appropriations Subcommittee

Start Date and Time:	Tuesday, November 17, 2015 01:00 pm
End Date and Time:	Tuesday, November 17, 2015 03:00 pm
Location:	Webster Hall (212 Knott)
Duration:	2.00 hrs

Overview and Update by the Department of Health on:

- Biomedical/Alzheimer's Research
- The Florida Cancer Registry
- The Biomedical Research Advisory Council with Daniel Armstrong, Ph.D., Chair

NOTICE FINALIZED on 11/10/2015 3:45PM by LAL

COMMITTEE MEETING REPORT

Health Care Appropriations Subcommittee

11/17/2015 1:00:00PM

Location: Webster Hall (212 Knott)

Summary: No Bills Considered

Committee meeting was reported out: Tuesday, November 17, 2015 3:11:39PM

COMMITTEE MEETING REPORT

Health Care Appropriations Subcommittee

11/17/2015 1:00:00PM

Location: Webster Hall (212 Knott)

Attendance:

	Present	*Absent*	*Excused*
Matt Hudson (Chair)	X		
Michael Bileca	X		
Jason Brodeur	X		
Janet Cruz	X		
W. Travis Cummings	X		
Gayle Harrell	X		
Shawn Harrison	X		
MaryLynn Magar	X		
Jared Moskowitz			X
Amanda Murphy	X		
Cary Pigman	X		
David Richardson	X		
Kenneth Roberson	X		
Totals:	**12**	**0**	**1**

Committee meeting was reported out: Tuesday, November 17, 2015 3:11:39PM

COMMITTEE MEETING REPORT

Health Care Appropriations Subcommittee

11/17/2015 1:00:00PM

Location: Webster Hall (212 Knott)

Presentation/Workshop/Other Business Appearances:

Biomedical Research Advisory Council (BRAC)
Armstrong, Daniel (At Request Of Chair) - Information Only
Biomedical Research Advisory Council (BRAC)
Chair
PO Box 016820
Miami FL 33101
Phone: (305) 243-6801

Biomedical/Alzheimer's Research & Florida Cancer Registry
Philip, Dr. Celeste (State Employee) (At Request Of Chair) - Information Only
Department of Health
Deputy Secretary for Health/Deputy State Health Officer for CMS
4052 Bald Cypress Way
Tallahassee FL 32399
Phone: (850) 245-4245

Committee meeting was reported out: Tuesday, November 17, 2015 3:11:39PM

Bankhead-Coley Cancer Research Program

Institution	Grant Amount	Life to Date Expenditures	Type of Grant	Project Title	Project Abstract
H. Lee Moffitt Cancer Center	$ 1,290,000.00	$ 107,500.00	Discovery Science	An Integrated Computational and Biological Approach to Curing Prostate to Bone Metastases	The American Cancer Society predicts that approximately 6 Floridian men will die from prostate cancer each day in 2014. These deaths are due to the cancer spreading to secondary sites. Prostate cancer frequently metastasizes to the skeleton where it promotes extensive bone destruction and formation causing great pain to the patient. Clearly, understanding how prostate cancer cells communicate with normal bone cells in order to establish and grow can yield new therapeutic targets. Traditional biological experimentation has enhanced our understanding but a major limitation is an inability to investigate multiple parallel cellular interactions. To circumvent this limitation, we propose to use computational modeling. Just like computational models can predict hurricane patterns, they can also be used to predict how prostate cancer grows and interacts with the bone environment or responds to applied therapies. We have used our biological observations to fuel a computational model that has predicted; 1) transforming growth factorβ (TGFβ) is crucial for the growth of the cancer in bone, 2) bone destroying osteoclast cells contribute to prostate cancer growth in a cyclical manner and, 3) specialized cells known as mesenchymal stem cells (MSCs) contribute to prostate cancer growth and bone formation. The objectives of this proposal are to test the accuracy of the computational model predictions and whether computational models can be used to optimize the efficacy and potency of established and emerging therapies. To achieve this, we will use in vivo mouse models of bone metastatic prostate cancer that mimic the human disease. We expect our results will yield robust computational model of bone metastatic prostate cancer that can be used identify new therapeutic strategies. Most importantly, the generation and validation of the computational model ensures its application as a research tool to examine a broad range of human cancers afflicting Floridians.
Mayo Cancer Clinic	$ 1,200,953.00	$ 100,079.42	Discovery Science	Development of Assays for Individualized Breast Cancer Risk Prediction	More than 1 million women in the US every year undergo breast biopsies for mammographic abnormalities or palpable lesions. The majority of these women have nonmalignant breast lesions that are classified as benign breast disease (BBD). Because they have BBD, these women are known to have significantly elevated risk of progression to breast cancer, but at present there is little information that a woman with BBD can use to determine her individual risk. Two key clinical questions arise from these observations. Can we identify which of these women are most likely to develop breast cancer? If we can identify high risk patients, then what can we do to reduce cancer mortality among them? The first part of our proposal focuses on identification of women who are at risk for developing estrogen receptor-positive breast cancer and who thus would benefit from chemo preventive endocrine therapy. A parallel aim is to identify women who are at risk of developing aggressive breast cancers for which current treatment methods are not as effective, and for which more frequent mammography could be recommended to identify disease at the earliest possible stage. We propose to develop a rapid and inexpensive clinical assay that uses RNA from benign breast biopsies to assess molecular markers as the basis for an individualized model for breast cancer risk prediction. A robust breast cancer risk model would help focus chemoprevention and surveillance efforts towards those women who would benefit most from them, and could also identify women who are at low risk, reducing unnecessary patient anxiety and helping providers to establish an appropriately informed schedule for future surveillance. Successful completion of our aims thus will be "practice changing" and will decrease both the incidence of and the mortality associated with breast cancer among women who have been diagnosed with BBD.

Bankhead-Coley Cancer Research Program

Institution	Grant Amount	Life to Date Expenditures	Type of Grant	Project Title	Project Abstract
University of Florida	$ 86,000.00	$ 43,000.00	Bridge	Molecular Regulation of CNS Leukemia Development	The invasion of malignant leukemic cells to the central nervous system (CNS) is common and often fatal for patients with acute lymphoblastic leukemia, the most common blood cancer mainly affecting children and adolescents. Current intensified CNS-directed approaches have improved survival outcome, but caused adverse complications for children such as secondary tumors, impaired growth, chronic health problems, and toxicity-related death. New effective, less toxic strategies for managing CNS leukemia will require a better understanding of the pathogenesis of CNS leukemia. In this application we propose to elucidate molecular events critical for the development and progression of CNS leukemia. This research will provide insights into the molecular pathogenesis of CNS leukemia and likely reveal novel therapeutic targets for effectively blocking CNS leukemia.
University of Florida	$ 1,107,000.00	$ -	Discovery Science	Novel Agents that Simultaneously Target HER2, EGFR, and HER3 for Treating Breast Cancer and Overcoming Therapeutic Resistance	The HER2 oncoprotein is overexpressed in 20-25% of human breast cancers. While the HER2-specific antibody Trastuzumab (Herceptin) has improved the survival of HER2-positive breast cancer, primary and acquired therapeutic resistance are critical problems. A major source of resistance is signaling through the HER2 family members EGFR and HER3 after HER2 has been inactivated. We have identified compounds that downregulate HER2, EGFR, and HER3 protein levels in parallel. These agents potently block the growth of HER2-positive human breast tumor xenografts with no evidence of toxicity. These compounds function through a novel mechanism that involves disruption of the intricate array of disulfide bonds in the evolutionarily conserved extracellular domains of HER2, EGFR, and HER3. We refer to these agents as Disulfide bond Disrupting Agents (DDAs) and have defined a common pharmacophore among these compounds. Uncontrolled signaling through the Phosphatidylinositol 3'-Kinase (PI3K) pathway constitutes an important mechanism of acquired resistance to HER2-specific drugs. Preliminary studies indicate that DDAs cooperate with the HER2/EGFR tyrosine kinase inhibitor Lapatinib to block mitogenic/survival signaling and induce the death of breast cancer cells that are refractory to each of the drugs applied individually. This project has three major goals. Goal 1 is to maximize the anticancer activity of DDAs through an improved understanding of their chemical and biochemical mechanisms of action. Goal 2 is to test optimized DDA derivatives for activity against patient-derived breast cancer xenografts. Goal 3 is to assess the ability of novel combination therapies employing DDAs, Lapatinib, and mTOR/PI3K inhibitors to induce the regression of Trastuzumab and Lapatinib-resistant HER2-positive human breast tumors in animal models.

James and Esther King Biomedical Research Program

Institution	Grant Amount	Life to Date Expenditures	Type of Grant	Project Title	Project Abstract
University of Miami	$ 1,953,000.00	$ 122,062.50	Clinical Research	Addressing Racial/Ethnic Tobacco Health Disparities via Group Intervention	The importance of reducing tobacco-associated health disparities between cannot be understated. Racial/ethnic minorities are less likely to quit smoking, and tend to have elevated stress and depressive symptoms, which may contribute to cessation disparities. Cognitive behavioral therapy (CBT) for cessation addresses these concerns and has the potential to reduce/eliminate disparities. Our preliminary research found racial/ethnic differences in baseline perceived stress and depressive symptoms. Following CBT, these differences were no longer present. Moreover, compared to Whites, African Americans exhibited blunted hypothalamic-pituitary-adrenal (HPA) axis functioning. This RCT will be the first to test the impact of CBT on smoking cessation disparities. Our specific aims are to: (1) Examine the effects of CBT on perceived stress and depressive symptoms in a racially/ethnically diverse sample; (2) test the efficacy of CBT for eliminating smoking cessation disparities; and (3) examine physiological distress as an underlying mechanism for the effects of CBT on racial/ethnic minority smokers (exploratory). We expect that CBT will eliminate racial/ethnic differences in stress and depressive symptoms, and smoking cessation compared to the general health education (GHE) control group. We also hypothesize that HPA functioning will mediate the effect of CBT on smoking cessation, particularly among racial/ethnic minorities. We will randomly assign African American/Black, Hispanic, or White smokers to CBT or GHE, and provide transdermal nicotine patches (TNP) to both intervention groups. Assessments will occur at the end-of-therapy (EOT), and 3, 6, and 12-months. Our primary abstinence outcome will be smoking cessation over the previous 7 days. We will also examine the effect of CBT on TNP adherence. This study has implications for eliminating disparities in psychosocial factors related to smoking cessation, and disparities in quitting success. Addressing stress and depressive symptoms through CBT may facilitate cessation, particularly among racial/ethnic minorities.
University of Miami	$ 1,951,531.00	$ 97,576.55	Clinical Research	Adverse Airway Effects of Inhaled Nicotine from Tobacco and E-cigarettes	In healthy persons, the lungs and airways are cleared from dust, viruses and bacteria to prevent disease development. Cigarette smoke impairs these host defense systems, allowing mucus build up, which is revealed by cough productive of phlegm and associated with frequent infections. This leads to diseases called chronic bronchitis and COPD. From a public health perspective, smoking cessation is therefore an important goal. To try to decrease nicotine craving during smoking cessation, tobacco-free nicotine delivery devices such as electronic cigarettes (ECs) are used. However, the safety of inhaled nicotine via ECs is unknown. In the present study, we will first use human cells that represent the airway surface in a dish and expose them to smoke to study the mechanism by which smoke components, especially nicotine, cause changes leading to increased sputum production. Our preliminary results show that the inflammatory molecule TGF-β1 is responsible for many of these changes and that inhibition of this molecule's signaling can prevent mucus build up. We will test whether nicotine directly or delivery via ECs causes changes similar to tobacco smoke in vitro. Next, we will examine whether changes observed in vitro also occur in vivo in human beings. We will test whether subjects who quit smoking with ECs show toxic effects from nicotine delivered to their airways or whether such a strategy is safe. Therefore, this translational research project will examine treatments to reverse smoking effects on the airway epithelium and will comprehensibly examine whether the delivery of nicotine via ECs has detrimental effects as well. The outcome of this project will not only be important for subjects with smoke-induced lung diseases, but will also provide a decision making basis for subjects and policy makers how to use and regulate nicotine delivery devices such as ECs.

James and Esther King Biomedical Research Program

Institution	Grant Amount	Life to Date Expenditures	Type of Grant	Project Title	Project Abstract
University of Florida	$ 1,464,750.00	$ 122,062.50	Discovery Science	Novel Small Molecules for Alpha-1 Antitrypsin Deficiency	The alpha-1 antitrypsin (AAT) deficiency is a common genetic disease with pulmonary emphysema and chronic obstructive pulmonary disease (COPD), for which there is no effective treatment. Smoking tobacco is the single most important risk factor to accelerate the lung disease. The fundamental pathological process is that the accumulation of mutant AAT in the form of polymers within hepatocytes causes low levels of AAT in the serum, resulting in lung tissue damage by proteinases. AAT is the second most abundant protein in the blood. A effective method to treat COPD is to stop AAT forming multiple chains in the liver and allow the protein coming out. Secretion of the protein may simultaneously alleviate both the liver and the lung diseases. Protein structural analysis have identified the site responsible for AAT polymerization (chains). This site is an attractive target for drug design. We think that specific small molecules that interfere with AAT polymerization can be identified by a molecular docking approach and these small molecules can be developed into novel therapeutic drugs. We have used computer-based molecular docking program and the NCI/Developmental Therapeutics Program (NCI/DTP) depository to identify promising compounds that demonstrate efficacy to enhance secretion of AAT protein. We have obtained US patent for these molecules. Our objective is to develop these small molecules into clinical useful drugs. In this proposed study, we will test and validate these compounds in cell and animal models. The preclinical study will be the scientific basis for subsequent clinical trials.
H. Lee Moffitt Cancer Research Center	$ 1,145,378.00	$ 95,448.16	Discovery Science	Proliferative Signatures to Predict the Benefit of Adjuvant Chemotherapy in Early-Stage Non-small Cell Lung Cancer	Stage 1 lung cancer patients have only a 50% chance of surviving for five years. We believe that many of these patients should be treated more aggressively than is currently recommended. Since 2010, these patients are treated surgically and are released, based on evidence that the group as a whole, does not benefit. However, since HALF of them will recur and die we can surmise that many were not cancer free after surgery. These might have benefitted from adjuvant (given after surgery) chemotherapy, but in the past there was no way to tell which patients would benefit. Recognizing this problem, we have identified a genetic signature that may identify early-staged tumors that have deadly potential. We have developed our signature into a relatively simple and inexpensive test based on Nano-String barcode technology. This test can be used on standard pathology sides (even if they are decades old).It would be very expensive to prove that our test works by conducting a clinical trial in which patients would be randomized into two arms. Fortunately, the trial has already been done, in two ways. First, a study was published in 2010 by the Spanish Lung Cancer Group that essentially performed the definitive clinical trial on treatment decisions for early staged lung cancer and they have provided the pathology slides from 223 of those patients. Second, we have utilized our access to samples from Floridian-based patients to identify a cohort of about 399 patients one-third of which were treated with ACT. We will also perform mutational analysis on these cohorts. We will use these two cohorts to further prove that our test works and validate how well it works in combination with other predictors such as mutation analysis.

Ed and Ethel Moore Alzheimer Research Program

Institution	Grant Amount	Life to Date Expenditures	Type of Grant	Project Title
University of South Fl	$ 112,500	$ 84,375	Diseases Related to Alzheimer's disease	Flavonoid-diosmin, a novel gamma-secretase modulator, for the treatment of Alzheimer's Disease
University of South Fl	$ 112,500	$ 84,375	Diseases Related to Alzheimer's disease	Modulation of Arginine Metabolism and Polyamines to Mitigate Alzheimer's Disease Pathology

PLEASE FILL OUT THE ENTIRE FORM AND SUBMIT TWO COPIES TO THE COMMITTEE/SUBCOMMITTEE ADMINISTRATIVE ASSISTANT AT THE MEETING

TYPE OR PRINT CLEARLY

COMMITTEE/SUBCOMMITTEE APPEARANCE RECORD

Bill Number N/A **Date** 11-17-15

Name Dr. Celeste Phillip

Title Deputy Secretary of Health

Address 4052 Bald Cypress Way

City Tallahassee **State/Zip** FL 32399

Phone Number 850-245-4006

Representing Department of Health

Lobbyist (registered) YES ☑ NO ☐

State Employee YES ☐ NO ☑

If you are testifying regarding an amendment, please indicate if your position as a proponent or an opponent is the same as on the bill as a whole.

			Amendment	Bill
I wish to speak	☐	Proponent	☐	☐
I have been requested to speak	☑	Opponent	☐	☐
		Information	☐	☐

Subject matter: Biomedical Research

Committee/Subcommittee: Health Care Appropriations

H-16 (2010)

PLEASE FILL OUT THE ENTIRE FORM AND SUBMIT TWO COPIES TO THE COMMITTEE/SUBCOMMITTEE ADMINISTRATIVE ASSISTANT AT THE MEETING

TYPE OR PRINT CLEARLY

COMMITTEE/SUBCOMMITTEE APPEARANCE RECORD

Bill Number N/A **Date** 11-17-15

Name Dr. Armstrong, Danny

Title Chairman, Biomedical Research Advisory Council

Address PO Box 016820

City Miami **State/Zip** FL 33101

Phone Number 305-243-6901

Representing BRAC

Lobbyist (registered) YES ☐ NO ☑

State Employee YES ☐ NO ☑

If you are testifying regarding an amendment, please indicate if your position as a proponent or an opponent is the same as on the bill as a whole.

			Amendment	Bill
I wish to speak	☐	**Proponent**	☐	☐
I have been requested to speak	☑	**Opponent**	☐	☐
		Information	☐	☐

Subject matter: BRAC

Committee/Subcommittee: Health Care Appropriations

H-16 (2010)