



1/13/2016

To: Jin-Xin Kong  
Science Editor  
*World Journal of Obstetrics and Gynecology*

Dear Dr. Kong,

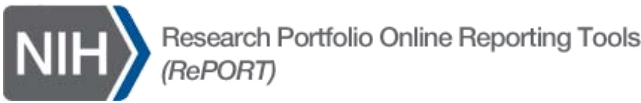
I am writing this letter to declare that this review work (manuscript No. 21039) is supported by my two NIH/NCI R01 grants, No. 5R01CA157779-03 and No. 5R01CA163820-04. The federal funding agency of my grant funds is the National Cancer Institute (NCI) of National Institutes of Health (NIH) in the United States of America.

I also provided the online accessible information (appended with this letter, pages 2-6) of my grants from the federal public grant search website "NIH Research Portfolio Online Reporting Tools" (NIH RePORTER; <https://projectreporter.nih.gov/reporter.cfm>). Please feel free to contact me if you have any questions in regard to the provided grant information.

Sincerely,

A handwritten signature in cursive script that reads "Qun Zhou".

Qun Zhou, M.D., Ph.D.  
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Greenebaum Cancer Center  
Department of Biochemistry and Molecular Biology  
University of Maryland School of Medicine  
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	T	Act	Project	Year	Sub #	Project Title	Contact PI/ Project Leader	Organization	FY	Admin IC	Funding IC	FY Total Cost by IC	Similar Projects
<input type="checkbox"/>	5	R01	CA157779	03		<a href="#">SHIKONIN AND NRF2 CHEMOPREVENTION</a>	<a href="#">ZHOU, QUN</a>	UNIVERSITY OF MARYLAND BALTIMORE	2015	NCI	NCI	\$286,661	
<input type="checkbox"/>	5	R01	CA163820	04		<a href="#">MIR-140 AND BREAST CANCER PREVENTION</a>	<a href="#">ZHOU, QUN</a>	UNIVERSITY OF MARYLAND BALTIMORE	2015	NCI	NCI	\$318,513	

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## Project Information ?

5R01CA157779-03

DESCRIPTION DETAILS RESULTS HISTORY SUBPROJECTS

Project Number: 5R01CA157779-03

Title: SHIKONIN AND NRF2 CHEMOPREVENTION

Contact PI / Project Leader: [ZHOU, QUN](#)Awardee Organization: UNIVERSITY OF MARYLAND  
BALTIMORE

### Abstract Text:

DESCRIPTION (provided by applicant): Estrogen is a causative factor for the development of breast cancer. It remains critically important to identify novel therapies that prevent estrogen carcinogenesis without significant toxicity. Induction of detoxifying enzymes is considered an important mechanism of protection against estrogen-associated carcinogenesis because they facilitate removal of toxic estrogens. The levels of detoxifying enzymes are determined by the key transcription factor Nrf2 factor that binds to the antioxidant response element in the promoters of genes encoding detoxification enzymes to increase transcription. Thus, a chemoprevention goal is to enhance Nrf2 function. Our preliminary studies show a novel role for shikonin (a bioactive agent extracted from the Shikon plant) in breast cancer prevention via a dual mechanism of action. Shikonin inhibits estrogen signaling and induces Nrf2 expression. This unique chemopreventive activity inhibits transformation from normal mammary epithelial to malignant states and prevents estrogen-dependent tumor formation. Based on these findings, we hypothesize that shikonin activates Nrf2 function leading to increased detoxifying enzyme expression. Consequently, these enzymes will remove genotoxic estrogen, and reduce DNA damage. We will test this hypothesis by determining 1) mechanisms of estrogen and shikonin in regulating expression of Nrf2 and detoxifying enzymes; 2) ability of shikonin to impact the Nrf2-dependent chemoprotection pathway; and 3) the role of Nrf2 in shikonin chemoprevention of mammary tumorigenesis using Nrf2 knockout mice. The results obtained from this proposal reveal that shikonin reverses the estrogen action in a novel way by reducing estrogen suppression of Nrf2 expression. These studies demonstrate that shikonin is a novel dietary agent with great potential as a breast cancer preventative.

### Public Health Relevance Statement:

PUBLIC HEALTH RELEVANCE: Our proposed studies identify that a novel dietary agent, shikonin (a bioactive component extracted from the Shikon plant), prevents breast cancer by activation of Nrf2-dependent detoxifying enzymes in vitro and in vivo.

### Project Terms:

AKT1 gene; Antioxidants; base; Binding (Molecular Function); Breast Cancer Cell; Breast Cancer Prevention; Breast Cancer Risk Factor; Breast Carcinoma; Breast Epithelial Cells; breast tumorigenesis; carcinogenesis; Carcinogens; Chemoprevention; Chemopreventive Agent; Chemoprotection; chromatin modification; Development; Diet; DNA Damage; Drug Metabolic Detoxication; Enzymes; Epithelial; Epithelial Cell Proliferation; Estrogen Receptor alpha; Estrogens; Excision; Exhibits; Exposure to; Gene Targeting; Genes; Genetic Transcription; Goals; Health; Human; In Vitro; in vivo; Knock-out; Knockout Mice; Malignant - descriptor; malignant breast neoplasm; malignant state; Mammary gland; Mammary Tumorigenesis; MAPK3 gene; Mediating; Mediator of activation protein; Monitor; Mus; NAD(P)H dehydrogenase (quinone) 1, human; Nature; NF-E2-related factor 2; novel; nucleocytoplasmic transport; oxidative DNA damage; Pathway interactions; Plants; prevent; Production; Promoter Regions (Genetics); Promotor (Genetics); Proteins; Reactive Oxygen Species; response; Response Elements; Role; screening; Signal Pathway; Signal Transduction; Staging; Testing; Toxic effect; transcription factor; tumor; tumor growth; Xenograft procedure

### Contact PI Information:

Name: ZHOU, QUN

Email: [Click to view contact PI email address](#)

Title: M.D PH.D

### Program Official Information:

Name: MALONE, WINFRED F

Email: [Click to view PO email address](#)

### Other PI Information:

Not Applicable

### Organization:

Name: UNIVERSITY OF MARYLAND BALTIMORE

City: BALTIMORE Country: UNITED STATES (US)

### Department / Educational Institution Type:

BIOCHEMISTRY

SCHOOLS OF MEDICINE

### Congressional District:

State Code: MD

District: 07

### Other Information:

FOA: [PA-11-260](#)

Study Section: Special Emphasis Panel (ZRG1-OTC-K (03))

Fiscal Year: 2015 Award Notice Date: 6-MAR-2015

DUNS Number: 188435911

Project Start Date: 1-JUL-2013

Budget Start Date: 1-MAY-2015

CFDA Code: 393

Project End Date: 30-APR-2018

Budget End Date: 30-APR-2016

### Administering Institutes or Centers:

NATIONAL CANCER INSTITUTE

### Project Funding Information for 2015:

Total Funding: \$286,661

Direct Costs: \$186,750

Indirect Costs: \$99,911

Year

Funding IC

FY Total Cost by IC

2015

NATIONAL CANCER INSTITUTE

\$286,661

History:								
Project Number	Sub #	Project Title	Contact Principal Investigator	Organization	FY	Admin IC	Funding IC	FY Total Cost by IC
5R01CA157779-03		SHIKONIN AND NRF2 CHEMOPREVENTION	ZHOU, QUN	UNIVERSITY OF MARYLAND BALTIMORE	2015	NCI	NCI	\$286,661
5R01CA157779-02		SHIKONIN AND NRF2 CHEMOPREVENTION	ZHOU, QUN	UNIVERSITY OF MARYLAND BALTIMORE	2014	NCI	NCI	\$278,062
1R01CA157779-01A1		SHIKONIN AND NRF2 CHEMOPREVENTION	ZHOU, QUN	UNIVERSITY OF MARYLAND BALTIMORE	2013	NCI	NCI	\$286,661
Subprojects:								
Project Number	Sub #	Project Title	Contact Principal Investigator	Organization	FY	Admin IC	Funding IC	FY Total Cost by IC
No Subprojects information available for 5R01CA157779-03								

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## Project Information ?

5R01CA163820-04

DESCRIPTION DETAILS RESULTS HISTORY SUBPROJECTS

Project Number: 5R01CA163820-04

Title: MIR-140 AND BREAST CANCER PREVENTION

Contact PI / Project Leader: [ZHOU, QUN](#)Awardee Organization: UNIVERSITY OF MARYLAND  
BALTIMORE

### Abstract Text:

DESCRIPTION (provided by applicant): Patients with ductal carcinoma in situ (DCIS) have significant risks of developing recurrence or invasive breast cancer even after they receive breast surgery. Thus, women stand to benefit from chemoprevention strategies that reduce the incidence of DCIS recurrence. However, the molecular mechanisms that underlie DCIS development remain unclear and so it is important to identify pathways that could be targeted for prevention. Our preliminary studies showed that loss of microRNA-140 (miR-140) expression is associated with the development of DCIS and that sulforaphane (a key bioactive ingredient of cruciferous vegetables) can restore miR-140 expression in primary DCIS cells. We further observed that reduced miR-140 expression is associated with increased expression of the SIRT1 histone deacetylase that is associated with enhanced cancer stem cell survival. Finally, miR-140 knockout mice spontaneously developed DCIS at 11 months of age. Our preliminary data suggest that miR-140 and SIRT1 have roles in DCIS development. Based on these results, we hypothesize that miR-140 loss leads to increased SIRT1 expression which drives DCIS development and increased accumulation of breast cancer stem cells. We further propose that sulforaphane treatment can restore miR-140 level which then targets and suppresses SIRT1 level to prevent DCIS development. Specific Aim 1 will define the mechanism of miR-140 inactivation in DCIS transformation. Specific Aim 2 is designed to determine the impact of miR-140 on cancer stem cell survival in DCIS transformation. Specific Aim 3 is designed to characterize the role of miR-140 in sulforaphane chemoprevention of DCIS in vivo. We believe that these studies are innovative and "high impact" because findings from our studies will identify a new mechanism of DCIS development and a new route of sulforaphane-dependent breast cancer prevention. We have developed all of the cell-based and animal models necessary to complete these studies.

### Public Health Relevance Statement:

Our studies strongly suggest that sulforaphane may be an important chemopreventive agent to suppress DCIS by correcting the epigenetic regulation to restore miR-140 expression.

### Project Terms:

3' Untranslated Regions; Accounting; Age-Months; Animal Model; base; Binding (Molecular Function); Biological Assay; Breast Cancer Cell; Breast Cancer Prevention; Breast Carcinoma; Breast Epithelial Cells; breast surgery; Breast-Conserving Surgery; Broccoli - dietary; Cancer stem cell; Cell Culture Techniques; Cell Survival; cell transformation; Cells; Chemoprevention; Chemopreventive Agent; chromatin modification; cruciferous vegetable; Data; design; Development; Diet; DNA Methylation; DNA Modification Process; Epigenetic Process; epigenetic regulation; Frequencies (time pattern); Genes; Goals; high risk; Histone Deacetylase; Histone Deacetylase Inhibitor; Histones; Human; In Vitro; in vitro Model; in vivo; Incidence; innovation; Knock-out; Knockout Mice; Lead; Malignant - descriptor; malignant breast neoplasm; Mammary Neoplasms; Messenger RNA; MicroRNAs; Modeling; Molecular; Mus; neoplastic; neoplastic cell; Noninfiltrating Intraductal Carcinoma; overexpression; Pathway interactions; Patients; prevent; Prevention; Promotor (Genetics); Proteins; Radiation therapy; Recurrence; Regulation; Repression; research study; response; Risk; Role; Route; Sampling; self-renewal; Staging; stem cell population; Stem cells; Sulforaphane; System; Testing; Tissues; Translations; tumor; tumor growth; tumorigenesis; Woman; Xenograft Model

### Contact PI Information:

Name: ZHOU, QUN

Email: [Click to view contact PI email address](#)

Title: M.D PH.D

### Program Official Information:

Name: ROSS, SHARON A.

Email: [Click to view PO email address](#)

### Other PI Information:

Not Applicable

### Organization:

Name: UNIVERSITY OF MARYLAND BALTIMORE  
City: BALTIMORE Country: UNITED STATES (US)

### Department / Educational Institution Type:

BIOCHEMISTRY  
SCHOOLS OF MEDICINE

### Congressional District:

State Code: MD  
District: 07

### Other Information:

FOA: [PA-10-067](#)

Study Section: Special Emphasis Panel (ZRG1-OTC-B (02))

Fiscal Year: 2015 Award Notice Date: 18-MAR-2015

DUNS Number: 188435911

Project Start Date: 1-JUN-2012

Budget Start Date: 1-APR-2015

CFDA Code: 393

Project End Date: 31-MAR-2017

Budget End Date: 31-MAR-2016

### Administering Institutes or Centers:

NATIONAL CANCER INSTITUTE

### Project Funding Information for 2015:

Total Funding: \$318,513

Direct Costs: \$207,500

Indirect Costs: \$111,013

Year

Funding IC

FY Total Cost by IC

2015

NATIONAL CANCER INSTITUTE

\$318,513

2015										NATIONAL CANCER INSTITUTE										\$318,513									
History:																													
Project Number		Sub #	Project Title				Contact Principal Investigator				Organization				FY		Admin IC		Funding IC		FY Total Cost by IC								
5R01CA163820-04			MIR-140 AND BREAST CANCER PREVENTION				ZHOU, QUN				UNIVERSITY OF MARYLAND BALTIMORE				2015		NCI		NCI		\$318,513								
5R01CA163820-03			MIR-140 AND BREAST CANCER PREVENTION				ZHOU, QUN				UNIVERSITY OF MARYLAND BALTIMORE				2014		NCI		NCI		\$308,958								
5R01CA163820-02			MIR-140 AND BREAST CANCER PREVENTION				ZHOU, QUN				UNIVERSITY OF MARYLAND BALTIMORE				2013		NCI		NCI		\$299,402								
1R01CA163820-01A1			MIR-140 AND BREAST CANCER PREVENTION				ZHOU, QUN				UNIVERSITY OF MARYLAND BALTIMORE				2012		NCI		NCI		\$318,513								
Subprojects:																													
Project Number		Sub #	Project Title				Contact Principal Investigator				Organization				FY		Admin IC				FY Total Cost by IC								
No Subprojects information available for 5R01CA163820-04																													

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