# VII OVARIAN CANCER RESEARCH PROGRAM

Ovarian cancer ranks second among gynecological cancers in the number of new cases and first among gynecological cancers in the number of deaths each year. It was estimated that in 2005, approximately 22,220 women would be diagnosed with ovarian cancer in the United States, and an estimated 16,210 would die from the disease. Ovarian cancer often is without overt or specific symptoms until late in its development; therefore, most women are diagnosed with advanced stage disease. As a result, the 5-year survival rate for all stages of ovarian cancer is approximately 44%. However, local ovarian cancer has a 94% 5-year relative survival rate, thus emphasizing the need for early diagnosis.[1]

Vision: To eliminate ovarian cancer.

Mission: To support innovative, integrated, multidisciplinary research efforts that will lead to better understanding, detection, diagnosis, prevention, and control of ovarian cancer.

Congressional Appropriations for Peer Reviewed Research:

- $71.7M in FY97–03
- $10M in FY04
- $10M in FY05

Funding Summary:

- 80 awards from the FY97–03 appropriations
- 12 awards from the FY04 appropriation
- ~16 awards anticipated from the FY05 appropriation

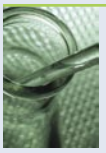
## PROGRAM BACKGROUND

The Department of Defense (DOD) Ovarian Cancer Research Program (OCRP) was established in fiscal year 1997 (FY97) by Appropriations Conference Committee Report No. 104-863, which provided $7.5 million (M) for research in ovarian cancer. As a major leader in extramural ovarian cancer research, the OCRP has managed $91.7M from FY97 to FY05 in an effort to eliminate ovarian cancer. A total of 92 awards have been made through FY04 across the categories of research, training/recruitment, and research resources. Key initiatives of the OCRP include building critical research resources, supporting innovative research, and bringing talented investigators into the ovarian cancer field. (See the box stories on pages VII-3 and VII-5 about the OCRP's investment in training and recruiting promising new investigators in the field of ovarian cancer.) Appendix B, Table B-4, summarizes congressional appropriations and the investment strategy executed by the OCRP for FY04 through FY05.

## THE FISCAL YEAR 2004 PROGRAM

Congress appropriated $10M in FY04 to continue the peer reviewed DOD OCRP, marking the eighth FY for this program. Two award mechanisms were supported: Idea Development Awards and New Investigator Research Awards. Both mechanisms encouraged innovative scientific ideas and technology applicable to tumor biology/etiology, preclinical development of targeted therapeutics, and molecular imaging/vital imaging in ovarian cancer research. However, New Investigator Research Awards did not require preliminary data and were directed at early-career investigators while Idea Development

[1] *American Cancer Society,* Cancer Facts and Figures, *2005*.



**"This continues to be the best of times and the worst of times in the field of ovarian cancer research. This year the Ovarian Cancer Research Program has seen a record number of applications, and the best quality of refreshingly innovative proposed scientific inquiry we've seen since the inception of this important program. Moreover, the potential of the research to truly impact the severe morbidity and mortality associated with this low incidence disease has never been greater. The frustration lies in our inability to support more of these novel ideas."**

**Nita Maihle, Ph.D., FY05 OCRP Integration Panel Member**

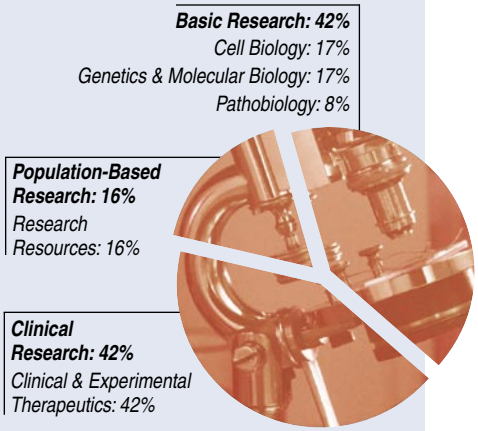



*Figure VII-1. FY04 OCRP Portfolio by Research Area*

Awards required preliminary data and were designed for investigators at all levels of experience. Of the 166 proposals received, 12 were funded. (See Table VII-1.) As illustrated in Figure VII-1, the FY04 OCRP has developed a research portfolio that encompasses basic, clinical, and population-based research.

*Table VII-1. Funding Summary for the FY04 OCRP*

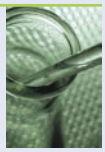
| Category and Award Mechanisms | Number of Proposals Received | Number of Awards | Investment |
|---|---|---|---|
| *Research* | | | |
| *Idea Development* | *85* | *7* | *$5.1M* |
| *New Investigator* | *81* | *5* | *$3.5M* |
| *Total* | *166* | *12* | *$8.6M* |

## THE VISION FOR THE FISCAL YEAR 2005 PROGRAM

Congress appropriated $10M to continue the OCRP in FY05. The program retained the Idea Development Awards and also introduced two new award mechanisms, the Historically Black Colleges and Universities/Minority Institutions (HBCU/MI) Collaborative Research Awards and Pilot Awards. All three award mechanisms placed emphasis on tumor biology/etiology, preclinical development of targeted therapeutics (excluding clinical trials), and early detection/diagnosis of ovarian cancer. As in previous years, the Idea Development Award continues to support innovative scientific ideas and approaches to disease eradication. The HBCU/MI Collaborative Research Award was designed to foster collaborations at an investigator level between an HBCU/MI and another institution. The Pilot Award was intended to support highly innovative, high-risk/high-reward ovarian cancer research from established investigators with less than 3 years of experience in the field of ovarian cancer or from junior faculty within 5 years of their last training experience. A total of 225 proposals were received across the three award mechanisms, as illustrated in Table VII-2, and approximately 16 awards are expected.

*Table VII-2. Award Mechanisms Offered and Proposals Received for the FY05 OCRP*

| Categories and Award Mechanisms | Number of Proposals Received |
|---|---|
| *Research* | |
| *Idea Development* | *135* |
| *Pilot* | *84* |
| *Research Resources* | |
| *HBCU/MI Collaborative Research* | *6* |
| *Total* | *225* |



## TRAINING OUR NATION'S FINEST RESEARCHERS

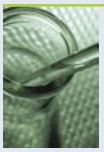
Dr. Michael Seiden, Chairman of the Research Committee of the Gynecologic Oncology Program at Dana Farber Cancer Institute/Harvard Cancer Center, was the recipient of an FY02 OCRP Institutional Training Grant (ITG). This mechanism was designed to encourage the initiation of a new postgraduate training program in ovarian cancer. The intent of the ITG was to focus on one or more program emphasis area(s) (i.e., etiology, prevention, early detection/diagnosis, and preclinical therapeutics) as related to epithelial ovarian carcinoma and/or primary peritoneal carcinoma. The ITG has been very competitive and successful in identifying four talented postdoctoral scientists committed to ovarian cancer research from a pool of 20 talented applicants. The postdoctoral scientists work with faculty at Dana Farber/Harvard Cancer Center in the fields of oncogenesis, signal transduction, pathology and mouse models, and cell biology. The following postdoctoral scientists have been funded with this training award:

- Dr. Ronny Drapkin has been developing new approaches to early detection using oligonucleotide microarray analysis for the identification of genes expressed in ovarian cancer. He demonstrated that a newly discovered human epididymis protein 4 (HE4) is overexpressed in primary tumors and ovarian cancer cell lines. Dr. Drapkin is trying to determine the utility of HE4 as a serum biomarker for early detection.
- Dr. John Miao is studying the role of the desmosomal junction protein hepsin in the progression of ovarian cancer.
- Dr. Sanja Sale has been focusing on the role of the serine threonine kinase mammalian target of Rapamycin (mTOR), in ovarian tumor progression. mTOR is involved in the regulation of cell growth through initiation of gene translation in response to nutrients, growth factors, insulin, and mitogens. Dr. Sale's main objective is to evaluate mTOR as a potential therapeutic target in epithelial ovarian cancer.
- Dr. Yong Zhan is focusing on the function of the Mullerian inhibitory substance (MIS) and its receptor in the development of the Mullerian epithelium. MIS, its receptor, and associated downstream signaling partners are targets for interventions for the treatment of epithelial ovarian carcinoma.

The mentoring program and training provided by the faculty at Dana Farber/Harvard Cancer Center have been especially successful at advancing the career of Dr. Drapkin. Dr. Drapkin recently left the training program to accept a faculty position in the Division of Molecular Pathology at Dana Farber Cancer Center. Additionally, he is the recipient of a transition grant from the Ovarian Cancer Research Foundation and a Mentored Clinical Scientist award from the National Cancer Institute, both of which are enabling him to launch his independent research career in ovarian cancer.

## SCIENTIFIC OUTCOMES AND ADVANCES

DOD OCRP awardees are making exciting contributions to the understanding, detection, diagnosis, prevention, and control of ovarian cancer. As evidenced in the highlighted projects in this section, the OCRP portfolio contains many innovative initiatives that hold promise toward eliminating this life-threatening disease. Additional examples of scientific outcomes, products, and technologies resulting from OCRP support can be found in Section III of this annual report.



## Signs and Symptoms

Ovarian cancer is often not associated with any obvious signs or symptoms until late in its development. However, some indicators of ovarian cancer may include the following:

- Enlargement of the abdomen
- General abdominal discomfort and/or pain (gas, pressure, indigestion, or distention)
- Abnormal bleeding from the vagina (although rare)
- Urinary symptoms

While these nonspecific symptoms are not always related to a serious condition, many women with advanced ovarian cancer recall experiencing these symptoms.

### Engineered Endostatin: Promising Antiangiogenic Therapeutic Agent for Ovarian Cancer

*Sundaram Ramakrishnan, Ph.D., University of Minnesota, St. Paul, Minnesota*

Dr. Sundaram Ramakrishnan, a FY98 OCRP Program Project awardee at the University of Minnesota, has been studying the role of angiogenesis in the etiology and prevention of ovarian cancer. Angiogenesis, the formation of new blood vessels, is a fundamental process in the growth of normal and cancerous tissues. Dr. Ramakrishnan and his team are developing an exciting therapeutic approach aimed at preventing angiogenesis in ovarian cancer by enhancing the biological activity and therapeutic efficacy of human endostatin, an important antiangiogenic protein. Dr. Ramakrishnan and his team engineered a new endostatin, called P125A-endostatin, by making a single amino acid substitution (a substitution of the amino acid proline with alanine) with further modifications to increase the bioavailability of P125A-endostatin. When delivered in a mouse model of ovarian cancer, the genetically engineered endostatin showed improved antiangiogenic biological activity compared to the native protein. Dr. Ramakrishnan's preclinical studies will help support human clinical trials using the mutant endostatin to inhibit the growth of ovarian cancer. Thus, mutant endostatin may prove to be a valuable antiangiogenic therapeutic agent for the treatment of ovarian cancer.

*This research has resulted in the following publications and patent:*

Subramanian IV, Ghebre R, and Ramakrishnan S. 2005. Adeno-associated virus-mediated delivery of a mutant endostatin suppresses ovarian carcinoma growth in mice. *Gene Ther* 12:30–38.

Yokoyama Y and Ramakrishnan S. 2004. Addition of integrin binding sequence to a mutant human endostatin improves inhibition of tumor growth. *Int J Cancer* 10;111(6):839–848.

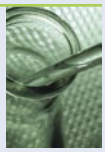
Yokoyama Y and Ramakrishnan S. 2004. Improved biological activity of a mutant endostatin containing a single amino-acid substitution. *Br J Cancer* 90:1627–1635.

Patent: Genetic Modification of Endostatin (6,825,167).

### Antiangiogenic Role of Dendritic Cells in Ovarian Cancer

*Weiping Zou, Ph.D., Tulane University Health Science Center, New Orleans, Louisiana*

Dr. Weiping Zou of the Tulane University Health Science Center is a recipient of an FY02 OCRP Idea Development Award to study the potential angiogenic role of immune cells, particularly dendritic cells (DC), in ovarian tumors. Tumor angiogenesis, the generation of new blood vessels, is essential for the tumor growth and nourishment.



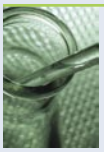
## THE NEW INVESTIGATOR AWARD PAYS OFF

During its first 2 years, the OCRP focused on building research resources through funding Program Projects. Beginning in FY99, the OCRP focused on human resources, instituting the New Investigator Award to prepare new, independent investigators (Assistant Professors or equivalents with no more than 6 years of experience in the field of ovarian cancer) for careers in ovarian cancer and to attract more senior investigators who are new to the ovarian cancer field. The intent was to encourage these investigators to stay in the ovarian cancer research field and seek additional funding. The OCRP funded 6 new investigators in FY99 and 10 in FY00. In 2004, the OCRP performed an analysis of the outcomes of these awards, seeking to define and determine the success of the mechanism in terms of leveraging CDMRP research dollars. This analysis showed that within the lifetime of their awards, the 16 FY99 through FY00 new investigators won 17 subsequent research awards, 6 of these in ovarian cancer. For each $1 OCRP spent, these investigators brought $2.65 in additional funds to cancer research. Jin Cheng, M.D., Ph.D., was an assistant professor at the University of South Florida (USF) when he received an FY99 New Investigator Award to study the role of the phosphatidylinositol 3-kinase (PI3K)- Akt (protein kinase B) pathway in ovarian cancer. Dr. Chen credits the OCRP award with focusing his research career toward understanding and treating ovarian cancer, research that now constitutes about 80% of his work. This OCRP award allowed him to develop support for the importance of the Akt pathway in breast, ovarian, and prostate cancers, research that he feels played a key role in his subsequently obtaining two R01 awards from the National Cancer Institute. The objective of his current NCI grant is to develop inhibitors of Akt proteins as therapeutics for ovarian, breast, and prostate cancers. An exciting outcome of Dr. Cheng's research has been the "resurrection" of the chemotherapeutic purine analogue triciribine, abandoned about a decade ago because its efficacy was erratic. Dr. Cheng and colleagues showed that triciribine is a specific inhibitor of the Akt signaling pathway and is likely to be effective in the 30% of ovarian cancers exhibiting overexpression of Akt; clinical trials for cancer patients with hyperactive Akt are planned for 2006. Since 1999, Dr. Cheng has published more than 30 articles on cancer research in peer-reviewed journals, including 13 on ovarian cancer. He is currently training seven postdoctoral scientists and three graduate students. Dr. Cheng is an example of how the OCRP New Investigator Award attracted an outstanding early-career scientist to ovarian cancer research, where he has established a research program that has had a significant impact on ovarian cancer.

DC, antigen-presenting cells that play an important role in the immune system, have not been studied for their role in tumor angiogenesis. The ultimate goal of this research is to determine the specific DC subsets that might differentially affect tumor angiogenesis. Dr. Zou's team isolated the two principal human DC subtypes, plasmacytoid dendritic cells (PDC) and myeloid dendritic cells (MDC), from peripheral blood mononuclear cells and ovarian tumor ascites and prepared two different subsets of Matrigel plugs bearing tumor-associated PDC and MDC. They tested these Matrigel plugs in healthy non-obese diabetic-severe combined immunodeficient mice and discovered that tumor-associated PDC induced angiogenesis through production of tumor necrosis factor-alpha and interleukin-8. In contrast, MDC suppressed angiogenesis in vivo through production of interleukin-12. Dr Zou's research demonstrates the novel role of DC in tumor neoangiogenesis. Furthermore, these results appear promising, as blocking

**"The DOD OCRP has initiated the best research that is leading to an understanding of the origins of ovarian cancer, which in turn may lead to early detection and cure of the now deadliest of gynecological cancers."**

**Patricia Goldman, FY05 OCRP Consumer Integration Panel Member**



**Fiscal Year 2005 Integration Panel Members**

**Stephen Rubin, M.D. (Chair)**
The University of Pennsylvania Medical Center

**Ronald Alvarez, M.D. (Chair Emeritus)**
University of Alabama at Birmingham

**Jeffrey Boyd, Ph.D. (Chair Elect)**
Memorial Sloan-Kettering Cancer Center

**Mary Scroggins, M.A. (Executive Committee Member-at-Large)**
Ovarian Cancer National Alliance

**James P. Basilion, Ph.D.**
Massachusetts General Hospital

**Kathleen R. Cho, M.D.**
University of Michigan Medical School

**Patricia Goldman**
Ovarian Cancer National Alliance

**Thomas Hamilton, Ph.D.**
Fox Chase Cancer Center

**Elise Kohn, M.D.**
National Cancer Institute

**Nita J. Maihle, Ph.D.**
Yale University School of Medicine

**Maurie Markman, M.D.**
University of Texas M.D. Anderson Cancer Center

**Nyrvah Richard**
In My Sister's Care

**Gustavo Rodriguez, M.D.**
Northwestern University Feinberg School of Medicine

**Nicole Urban, Sc.D.**
Fred Hutchinson Cancer Research Center

**S. Diane Yamada, M.D.**
University of Chicago Hospitals

PDC-mediated neovascularization in tumors may be a novel strategy to treat human ovarian cancer.

*For additional reading about this research, please refer to the following publication:*

Curiel TJ, Cheng P, Mottram P, et al. 2004. Dendritic cell subsets differentially regulate angiogenesis in human ovarian cancer. *Cancer Res* 64:5535–5538.

## *Novel Serum Biomarkers for the Detection of Ovarian Cancer*

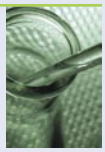
*Samuel Mok, Ph.D., Brigham and Women's Hospital, Boston, Massachusetts*

Most women are diagnosed with ovarian cancer at late stages of the disease. The 5-year survival rate for women diagnosed with early-stage localized disease is almost three times higher than that for women diagnosed with distant metastases. A new method for early-stage detection could save thousands of lives every year. Dr. Samuel Mok, a recipient of a FY98 OCRP Program Project Award, has been seeking new biomarkers to identify women with early-stage ovarian cancer. DNA microarray analysis shows that the epithelial cell adhesion molecule (Ep-CAM) is greatly overexpressed in ovarian cancers as compared to normal and benign ovarian epithelia. Ovarian tumors overexpressing Ep-CAM also produce corresponding levels of Ep-CAM autoantibodies, which can be measured easily in serum. Ep-CAM autoantibody expression is significantly higher in ovarian cancer patients than in patients with benign tumors or normal controls. Dr. Mok and colleagues also identified human kallikrein 6 (hK6, also known as protease M) as being overexpressed in ovarian cancer. Using a monoclonal antibody to hK6, they demonstrated that hK6 is highly expressed in ovarian tumors of various stages and subtypes, but not in normal ovarian epithelial cells. High levels of hK6 expression are found in many early-stage and low-grade tumors, and elevated hK6 proteins are found in benign epithelia adjacent to borderline and invasive tissues, suggesting that overexpression of hK6 is an early event in ovarian cancer development. Taken together, these findings suggest that Ep-CAM and hK6 may prove to be valuable serum biomarkers when screening women for early signs of ovarian cancer.

*Please refer to the following publications for additional information about this research:*

Ni X, Zhang W, Huang KC, et al. 2004. Characterization of human kallikrein 6/protease M expression in ovarian cancer. *Br J Cancer* 91:725–731.

Kim JH, Herlyn D, Wong KK, et al. 2003. Identification of epithelial cell adhesion molecule autoantibody in patients with ovarian cancer. *Clin Cancer Res* 9:4782–4791.



### *Squalamine and Cisplatin: Potential Ovarian Cancer Therapeutic Agents*

*Richard J. Pietras, M.D., Ph.D., University of California, Los Angeles, California*

Squalamine, a naturally occurring antiangiogenic steroidal compound, is found in tissues of the dogfish shark. Squalamine works through the protein vascular endothelial growth factor (VEGF) signaling pathway to inhibit endothelial cell surface proliferation and new capillary formation (angiogenesis). Squalamine also interacts with many chemotherapeutic agents and has been shown to enhance their effectiveness. Richard Pietras, a scientist from the University of California, Los Angeles, is studying a potential angiogenic role of squalamine alone and in combination with other chemotherapeutic drugs such as cisplatin and carboplatin. Through research funded by a FY00 OCRP Idea Development Award, Dr. Pietras has shown that squalamine is antiangiogenic in ovarian cancer xenografts and is able to enhance the cytotoxic effects of cisplatin on ovarian cancer cells. Dr. Pietras' research also provides evidence that ovarian tumor growth can be slowed due to the decreases in microvessel formation with squalamine treatment. Additionally, apoptosis (killing) of ovarian cancer cells was increased in xenograft nude mice due to increases in the cytotoxic effects of cisplatin in combination with squalamine. Dr. Pietras' preclinical studies in xenograft nude mouse models and ovarian cancer cells helped promote the initiation of clinical-translational Phase 2 trials of squalamine, which shows great promise for the treatment of patients with ovarian cancer.

*This research resulted in the following publication:*

Li D, Williams JI, and Pietras RJ. 2002. Squalamine and cisplatin block angiogenesis and growth of human ovarian cancer cells with or without HER-2 gene overexpression. *Oncogene* 21:2805–2814.

## OCRP RESEARCH IN THE NEWS

**March 28, 2005**

By Lori Oliwenstein, University of Southern California (USC) News Room

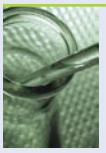
**Ovarian Cancer May Be Preventable**

A finding by USC researchers—which indicates that the disease is the result of a correctable biochemical problem—comes as good news for scientists and physicians.

Mutated BRCA1 genes cause ovarian cancer indirectly by interfering with the biochemical signals one ovarian cell sends to another, according to a team of researchers led by scientists at the USC/Norris Comprehensive Cancer Center and the Keck School of Medicine of USC.

**"Being a 13-year survivor of ovarian cancer with two recurrences, chemo, and radiation, I was honored and excited to participate on the DOD Peer Review committee for ovarian cancer. This helped me to fully understand the process of selecting worthy projects to possibly eradicate ovarian cancer and sharing this with other women in my community. It was a wonderful opportunity to meet others from major cancer institutions and see their dedication and impact on this work. As an ovarian cancer advocate, being on the Peer Review committee was interesting, challenging, and worthwhile and added to my advocacy growth. I would highly recommend this experience to others. Hopefully, one day, there will be a cure for ovarian cancer because of these efforts, and this type of work will not be necessary."**

**Linda Smith, FY05 OCRP Consumer Peer Review Panel Member**



**"In all of my years of serving on a variety of scientific review panels I have to say the OCRP uniquely drives home to me the urgency of the work to be done. I want to see this disease done with. One woman struck by ovarian cancer is too many for me."**

**Linda Malkas, Ph.D., FY05 OCRP Peer Review Panel Chair**

Their work is published in a recent issue of the journal *Current Biology*.

"Before, we thought this gene was a classical tumor suppressor," said Louis Dubeau, professor of pathology in the Keck School and Principal Investigator on the paper.

If that were the case, it would mean that mutation of the gene would allow the cell it is in to grow out of control and create a tumor. Instead, Dubeau noted, "What we've shown is that the gene actually acts indirectly, that it disrupts interactions between different cell types."

BRCA1, the well-known breast cancer gene, not only gives carriers of its mutated form a four-in-five chance of developing breast cancer, it also confers a 40 percent risk of developing ovarian cancer by the age of 70.

How that risk is imparted, however, had been harder to pin down.

"We've known for a long time that ovarian cancer is associated with ovulation, in that women who have regular menstrual cycles through their life without interruption by pregnancy or oral contraceptive use are at highest risk for developing sporadic ovarian cancer," Dubeau said.

"So we had some clues that the cells that control the menstrual cycle —the ovarian granulosa cells—have an influence on ovarian cancer."

Dubeau eventually got a handle on the problem by looking at ovarian cancer rates in genetically modified mice created in collaboration with Robert Maxson, Keck School professor of biochemistry and molecular biology and director of the mouse core facility at the USC/Norris Cancer Center.

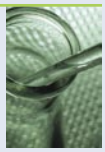
"The whole project was based on creating a mouse that lacks BRCA1 in only its granulosa cells," Dubeau said. "This collaboration was essential to the project's success."

What Dubeau and his colleagues found was that while mutating the BRCA1 gene in granulosa cells did indeed give rise to ovarian tumors, those tumors did not arise in granulosa cells.

Instead, when the tumor cells were analyzed, they were found to be epithelial cells very similar to those found in human ovarian cancers, with perfectly intact, functioning copies of the BRCA1 gene.

"What this says is that the cells that control the menstrual cycle, the ovarian granulosa cells, also control ovarian tumor development, but from a distance," Dubeau explained.

The most likely scenario, he said, is that the granulosa cells normally give off a chemical signal that stops the epithelial cells from growing out of control. When that chemical signal disappears or is muted by a



mutation in the BRCA1 gene, the epithelial cells don't get the message and keep on growing and dividing.

The result: ovarian cancer.

This finding is actually good news for scientists and physicians trying to figure out new ways to treat ovarian cancer.

If the cancer had arisen in the same cells that had the BRCA1 mutation, the only way to interfere would be to correct the mutation. In this case, however, there's a mediator—a biochemical of some sort—that scientists might be able to replace in people with identified BRCA1 mutations, making their risk of ovarian cancer drop.

In addition, once the chemical messenger that is affected has been identified, it will be much easier to diagnose a predisposition to ovarian cancer or pinpoint just who is at risk, simply by measuring the chemical's levels.

"The consequence of this finding," Dubeau said, "is that ovarian cancer is the result of some biochemical problem that may be correctable or preventable. That's what makes this finding so exciting."

Dubeau pointed out that women with BRCA1 mutations are also predisposed to cancers of the fallopian tubes and that the mice with mutated BRCA1 genes in their granulosa cells developed tumors there as well.

"This not only underscores the relevance of our mouse model to human cancer," Dubeau said, "but also strongly supports a theory we have formulated about the site of origin of ovarian cancers."

**"As a result of its two-tier review process, the OCRP has been successful at identifying and supporting some of the most innovative and important ovarian cancer research being done in the country today."**

**Stephen Rubin, M.D., FY05 OCRP IP Chair**

## BOTTOM LINE

Since 1997, the DOD OCRP has been responsible for managing $91.7M in congressional appropriations, which has resulted in 92 awards for FY97 through FY04. The OCRP's investment in building national ovarian cancer research resources, encouraging innovative research, and recruiting and training talented investigators new to the field has aided in the national health effort to improve the well-being of all women. Research highlights, award data, and abstracts of funded OCRP proposals can be viewed on the CDMRP website (http://cdmrp.army.mil).