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**DATA REPORT:**  
**OVARIAN CANCER IN MASSACHUSETTS**

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Bureau of Health Information, Statistics,  
Research, and Evaluation

Massachusetts Department of Public Health

January 2010

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# **DATA REPORT: OVARIAN CANCER IN MASSACHUSETTS**

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Massachusetts Department of Public Health

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# DATA REPORT: Ovarian Cancer in Massachusetts

## INTRODUCTION

Ovarian cancer was the eighth leading cancer diagnosed in females and the fifth leading cause of cancer death among females in Massachusetts between 2002 and 2006, whereas, uterine cancer was the fourth leading cancer diagnosed in females and the eighth leading cause of cancer deaths. For this period, the average annual age-adjusted incidence rate of ovarian cancer was 13.8 cases per 100,000 females and the age-adjusted mortality rate of ovarian cancer was 9.1 deaths per 100,000 females. The average number of ovarian cancer cases and deaths for this period would be 526 cases and 365 deaths per year (1). The national average annual age-adjusted incidence rate of ovarian cancer was 13.0 cases per 100,000 females for the period 2002-2006 (2). The age-adjusted mortality rate of ovarian cancer was 8.8 deaths per 100,000 females in the United States (2). The most recent data available from the National Cancer Institute reports an estimated 45.6% 5-year survival rate for ovarian cancer (2).

Ovarian cancer produces vague symptoms that, until recently, were believed to only occur in the later stages of the cancer (3). Researchers have found that 90% of women experience symptoms during the early stages of ovarian cancer; the key factors related to diagnosis are the frequency of these symptoms and the number of symptoms experienced that are associated with ovarian cancer (3). These symptoms include bloating, pelvic or abdominal pain, difficulty eating or feeling full quickly, and urinary symptoms (urgency or frequency) (3). Ovarian cancer is considered a rare cancer. The hereditary risk due to genetic mutations has become more relevant in both breast cancer and ovarian cancer, with 90% of hereditary ovarian cancers being associated with BRCA1 and BRCA2 tumor suppressor genes (4). There are very specific risk reduction recommendations given to women with BRCA1/BRCA2 genetic mutations, but there are no overall screening guidelines for the detection of ovarian cancer at this time (5).

There seems to be a relationship between the number of menstrual cycles in a woman's lifetime and her risk of developing ovarian cancer. Women have an increased risk of ovarian cancer if they started menstruating at an early age (before age 12), had no children or gave birth for the first time after age 30, and/or experienced menopause after age 50 (3). There are several identified ways to decrease the risk of ovarian cancer, including oral contraceptive use, tubal ligation and/or hysterectomy, pregnancy, breast feeding, and removal of ovaries (3). There is some speculation that the protective properties of these factors may be more strongly associated with ovarian cancer cases in premenopausal women (6).

Ovarian cancer can develop due to complex interactions between many types of factors, such as those discussed in this report (gynecological medications, gynecological procedures, genetics, hormonal cycles) or other unidentified factors that may influence the development of ovarian cancer. It is important to note that there is no way to completely eliminate the risk of ovarian cancer and all women are at risk of developing ovarian cancer. If there is a concern about the risk of ovarian cancer, a health care professional can help identify ways to reduce risk as well as decide if consultation with a genetic counselor would be appropriate (3).

## INFORMATION IN THIS REPORT

This report is based on data reported to the Massachusetts Cancer Registry (MCR) between 1982 and 2006. Ovarian cancer cases are presented by age, race/ethnicity, stage at diagnosis, and histology. *In situ* cases are only included in analyses of incidence by stage. All other analyses use only invasive ovarian cancer cases. Trends in age-adjusted incidence and mortality rates for 1982 through 2006 are compared to national rates from the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) Program. Age-specific incidence rates are compared using two sets of five-year time periods, 1990-1994 and 2002-2006. Age-adjusted rates by race/ethnicity, case distribution by stage, and case distribution by histology are also presented for Massachusetts for the period 2002-2006. Finally, the probability of diagnosis with or death from ovarian cancer is presented for Massachusetts females.

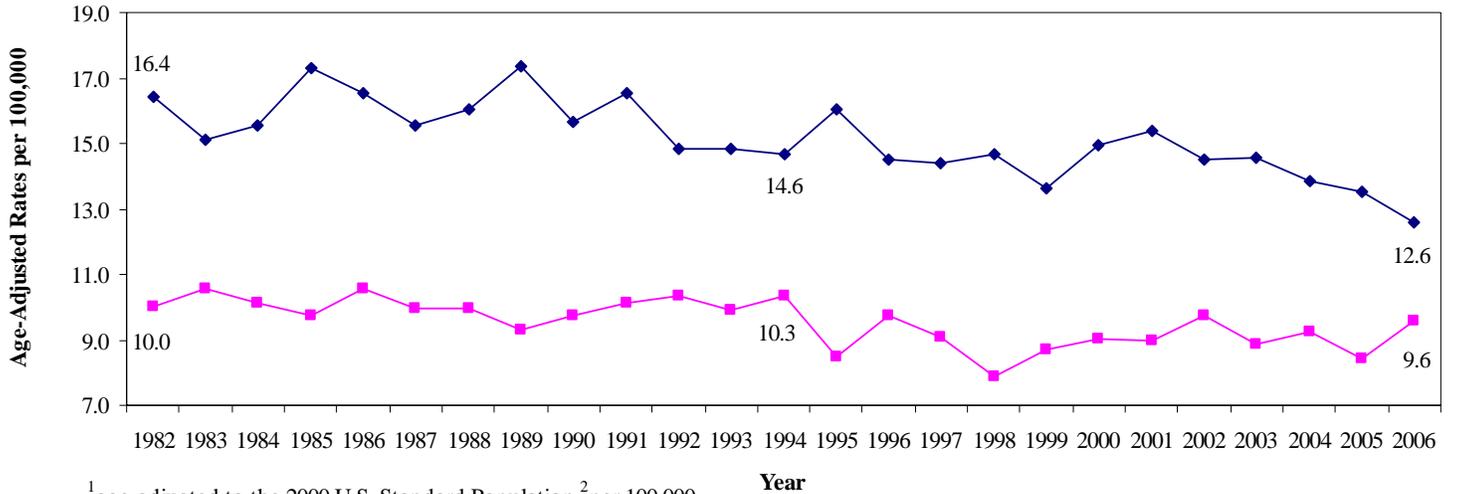
## DATA SECTION

### *Data Highlights*

- Age-adjusted incidence and mortality rates for ovarian cancer declined slowly, and at comparable rates, between 1982 and 2006. (Figure 1)
- The age-specific ovarian cancer incidence rates for 1990-1994 showed higher rates up to the 75-79 age group when compared to the rates for 2002-2006. (Figure 2)
- White, non-Hispanic females had the highest incidence and mortality rates of ovarian cancer among race/ethnicity groups. (Figure 3)
- 58.0% of ovarian cancer cases were detected at the distant stage. (Figure 4; Definitions, page 6)
- Epithelial tumors make up 86.5% of the ovarian cancer cases. (Figure 5; Definitions, page 7)
- The probability of developing ovarian cancer over the lifespan (0-85 years) was 1.5% for females. (Table 1)
- The probability of dying from ovarian cancer over the lifespan (0-85 years) was 1.2% for females. (Table 2)

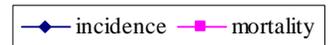
*Overall Incidence and Mortality Trends*

**FIGURE 1**  
**AGE-ADJUSTED<sup>1</sup> OVARIAN CANCER INCIDENCE AND MORTALITY RATES<sup>2</sup> FOR**  
**FEMALES**  
**Massachusetts, 1982-2006**



<sup>1</sup>age-adjusted to the 2000 U.S. Standard Population <sup>2</sup>per 100,000

Sources: Massachusetts Cancer Registry and Surveillance, Epidemiology, and End Results (SEER) Program



Age-adjusted incidence and mortality rates for Massachusetts females for the years 1982 to 2006 are presented in Figure 1. The incidence and mortality trends slowly declined over this time period.

The long-term incidence and mortality trends for females were analyzed using a Joinpoint regression model as described in the *Statistical Notes* section of this report. The results of the analyses are as follows:

- The incidence rate for females declined 0.8% per year for 1982-2006.
- The mortality rate for females declined 0.6% per year for 1982-2006.

The trends were statistically significant for both incidence and mortality rates for 1982-2006.

*Age-Specific Incidence Rates by Time Period*

**FIGURE 2**  
**AGE-SPECIFIC OVARIAN CANCER INCIDENCE RATES<sup>1</sup> FOR FEMALES**  
**Massachusetts, 1990-1994 and 2002-2006**

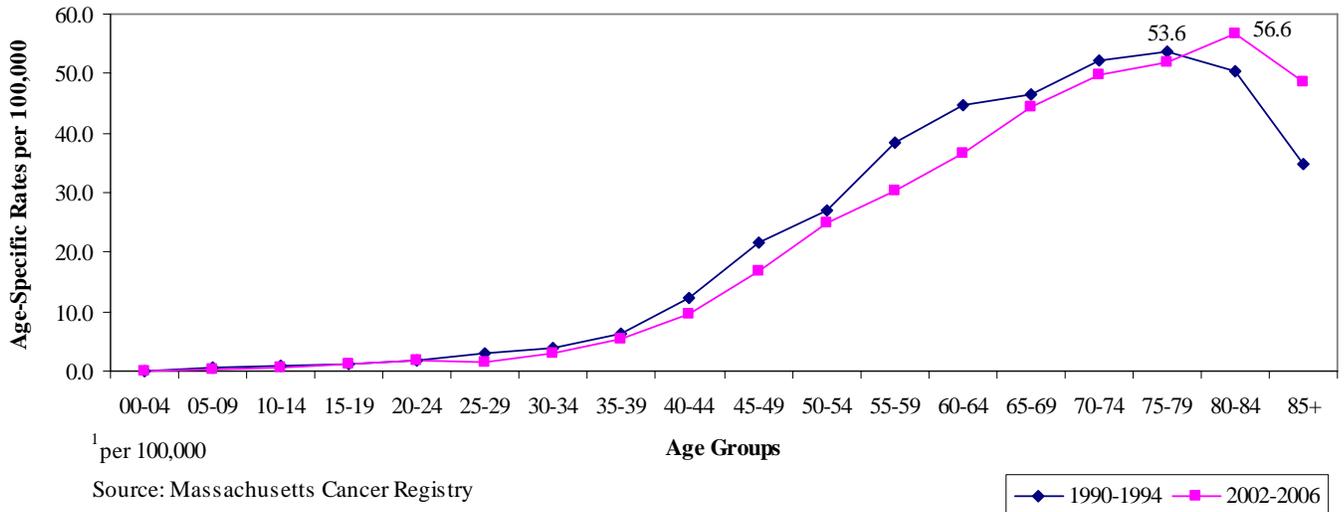


Figure 2 presents age-specific ovarian cancer incidence rates for two time periods: 1990-1994 and 2002-2006.

- Rates were relatively low for young women and then rose gradually starting at the age of 30 for both time periods.
- The rates for 1990-1994 rose to a peak of 53.6 per 100,000 in the 75-79 age group, while the rates for 2002-2006 peaked at 56.6 per 100,000 in the 80-84 age group.
- The rates for 1990-1994 dropped after the age of 79, while the rates for 2002-2006 dropped after the age of 84.

*Incidence and Mortality Rates by Race/Ethnicity*

**FIGURE 3**  
**AVERAGE ANNUAL AGE-ADJUSTED<sup>1</sup> OVARIAN CANCER INCIDENCE**  
**AND MORTALITY RATES<sup>2</sup> FOR FEMALES BY RACE/ETHNICITY**  
**Massachusetts, 2002-2006**

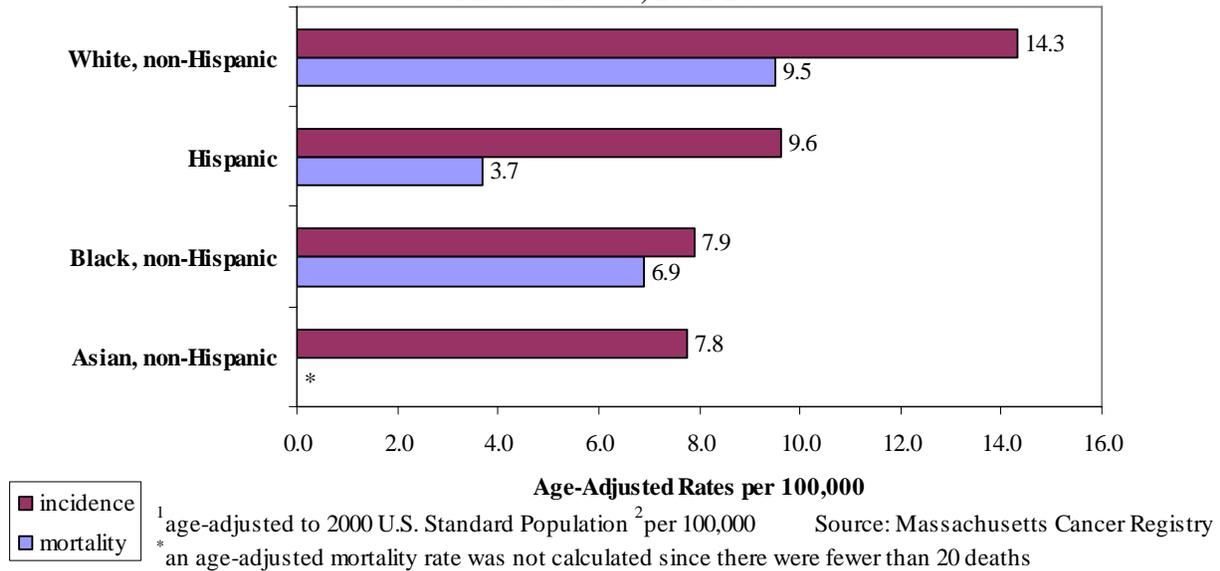


Figure 3 presents the average annual age-adjusted ovarian cancer incidence rates by race/ethnicity for Massachusetts females.

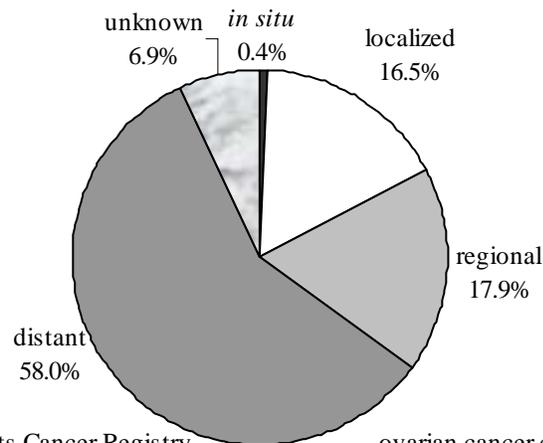
- White, non-Hispanic females had the highest ovarian cancer incidence and mortality rates.
- White, non-Hispanic females had an incidence rate 1.8 times higher than black, non-Hispanic females and Asian, non-Hispanic females, and 1.5 times higher than Hispanic females.
- White, non-Hispanic females had a mortality rate 1.4 times higher than black, non-Hispanic females and 2.6 times higher than Hispanic females. The mortality rate for Asian, non-Hispanic females was not calculated because there were fewer than 20 deaths.

### ***Incidence by Stage***

Ovarian cancer is classified into the following five stages, which help to determine treatment options and prognosis (7).

- *In Situ* (*early stage*): The earliest stage of cancer, before the cancer has spread, when it is limited to a small number of cells and has not invaded the organ itself.
- Localized (*early stage*): The cancer is found only in the body part (organ) where it began; it hasn't spread to any other parts.
- Regional (*late stage*): The cancer has spread beyond the original point where it started to the nearest surrounding part of the body (other tissues).
- Distant (*late stage*): The cancer has spread to parts of the body far away from the original point where it began. This is the most difficult stage to treat, since the cancer has spread throughout the body.
- Unstaged (*unknown*): There is not enough information about the cancer to assign a stage.

**FIGURE 4**  
**DISTRIBUTION OF *IN SITU* AND INVASIVE**  
**OVARIAN CANCER INCIDENT CASES BY STAGE**  
**Massachusetts 2002-2006**



Source: Massachusetts Cancer Registry

ovarian cancer cases N=2652

Figure 4 presents the distribution of *in situ* and invasive ovarian cancer incident cases by stage for Massachusetts females.

- The majority of ovarian cases (58.0%) are found when the cancer is at a distant stage.
- 34.4% of the cases are found when in a localized or regional stage.
- *In situ* cases account for less than one-half of one percent of cases.

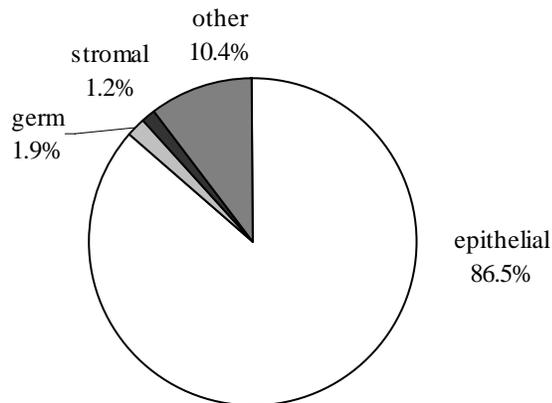
***Incidence by Histology Type***

*Epithelial tumors* start in the surface cells on the ovary.

*Germ cell carcinoma tumors* form in the cells that develop into the egg or ovum.

*Stromal carcinoma tumors* arise from the connective tissues that hold the ovary together and produce the hormones estrogen and progesterone.

**FIGURE 5**  
**DISTRIBUTION OF INVASIVE OVARIAN CANCER**  
**INCIDENT CASES BY HISTOLOGY TYPE**  
**Massachusetts, 2002-2006**



Source: Massachusetts Cancer Registry

ovarian cancer cases N=2636

Figure 5 presents the distribution of *in situ* and invasive ovarian cancer incident cases by histology type for Massachusetts females. Histology codes used are listed in the Technical Notes section of the report, under Disease Coding.

- 86.5% of ovarian cancers are of the epithelial cell type.
- Only 3.1% of ovarian cancers are of either the germ cell or stromal type.

### ***Probability of Developing or Dying from Ovarian Cancer***

To find the probability of developing ovarian cancer or the probability of dying from ovarian cancer for a female (F) of a certain age:

- Find the individual's age in the 'current age' column.
- Look across the row for the number that corresponds to the age of interest for the probability of developing ovarian cancer or the probability of dying from ovarian cancer.
- The percentage shown is the probability of developing or dying from ovarian cancer for an alive and cancer-free female by the age of interest.

Example: For a 50-year-old woman, the probability for developing ovarian cancer by the age of 70 is 0.9%.

**TABLE 1  
PROBABILITY OF DEVELOPING OVARIAN CANCER BY A SPECIFIC AGE FOR FEMALES  
Massachusetts, 1997-2006**

Percent Estimate of Developing Ovarian Cancer by a Certain Age

current age	35 F	40 F	45 F	50 F	55 F	60 F	65 F	70 F	75 F	80 F	85 F
0-85 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2	1.4	1.5
5 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2	1.4	1.5
10 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2	1.4	1.5
15 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2	1.4	1.5
20 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2	1.4	1.5
25 yrs	0.1	0.1	0.2	0.3	0.5	0.6	0.8	1.0	1.2	1.4	1.5
30 yrs	0.0	0.1	0.2	0.3	0.5	0.6	0.8	1.0	1.2	1.3	1.5
35 yrs	0.0	0.1	0.2	0.3	0.4	0.6	0.8	1.0	1.2	1.3	1.5
40 yrs		0.1	0.1	0.3	0.4	0.6	0.8	1.0	1.2	1.3	1.4
45 yrs			0.1	0.2	0.4	0.5	0.7	0.9	1.1	1.3	1.4
50 yrs				0.1	0.3	0.5	0.7	0.9	1.0	1.2	1.3
55 yrs					0.2	0.3	0.5	0.7	0.9	1.1	1.2
60 yrs						0.2	0.4	0.6	0.8	1.0	1.1
65 yrs							0.2	0.4	0.6	0.8	1.0
70 yrs								0.2	0.5	0.6	0.8
75 yrs									0.2	0.4	0.6
80 yrs										0.2	0.5
85 yrs											0.3

Source: Massachusetts Cancer Registry

Based on the 1997-2006 incidence data for ovarian cancer, there was a less than 0.1% chance of developing ovarian cancer in females before the age of 30. Therefore, those age segments are not included in this table.

The overall probability of developing ovarian cancer over the lifespan (0-85 years) was 1.5% for females.

**TABLE 2**  
**PROBABILITY OF DYING FROM OVARIAN CANCER BY A SPECIFIC AGE FOR FEMALES**  
**Massachusetts, 1997-2006**

Percent Estimate of Dying from Ovarian Cancer by a Certain Age

current age	45 F	50 F	55 F	60 F	65 F	70 F	75 F	80 F	85 F
0-85 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2
5 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2
10 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2
15 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2
20 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2
25 yrs	0.1	0.1	0.2	0.3	0.5	0.7	0.8	1.0	1.2
30 yrs	0.2	0.3	0.5	0.6	0.8	1.0	1.2	1.3	1.5
35 yrs	0.2	0.3	0.4	0.6	0.8	1.0	1.2	1.3	1.5
40 yrs	0.1	0.3	0.4	0.6	0.8	1.0	1.2	1.3	1.4
45 yrs	0.1	0.2	0.4	0.5	0.7	0.9	1.1	1.3	1.4
50 yrs		0.1	0.3	0.5	0.7	0.9	1.0	1.2	1.3
55 yrs			0.2	0.3	0.5	0.7	0.9	1.1	1.2
60 yrs				0.2	0.4	0.6	0.8	1.0	1.1
65 yrs					0.2	0.4	0.6	0.8	1.0
70 yrs						0.2	0.5	0.6	0.8
75 yrs							0.2	0.4	0.6
80 yrs								0.2	0.5
85 yrs									0.3

Source: Massachusetts Cancer Registry

Based on the 1997-2006 incidence data for ovarian cancer, there was a less than 0.1% chance of dying from ovarian cancer in females before the age of 40. Therefore, those age segments were not included in this table.

The overall probability of dying from ovarian cancer over the lifespan (0-85 years) was 1.2% for females.

## DISCUSSION

After reviewing the Massachusetts ovarian cancer data, there are no obvious reasons behind the decreasing rates found in the incidence and mortality trends (Figure 1). There are also no obvious explanations behind the decreasing incidence rates by age groups (Figure 2) or the disproportionate incidence and mortality rates among the race/ethnicity groups (Figure 3). The references that were assembled point to some gynecological procedures and medications that could help explain some of the trends noted, however. These trends are subject to change over time based on how gynecological procedures, medications, and other emerging medical research findings may affect ovarian cancer incidence and mortality.

The ovaries are organs that depend on hormonal signals to produce an egg or ovum and create the environment that is right for implantation of a fertilized egg into the uterus. There are several types of gynecological procedures and medications that can affect hormone levels: hysterectomies, surgical sterilization, and oral contraceptives. Increased awareness of the symptoms for ovarian cancer has become a greater priority in helping to identify ovarian cancers at earlier stages when treatment is the most effective.

Early detection of ovarian cancer can save lives. The national five-year relative survival are 94% localized, 73% regional, 28% distant, and 27% unstaged (2). The Massachusetts data demonstrate that ovarian cancer is usually diagnosed at a later stage (Figure 4). However, the survival rate of ovarian cancer detected in the localized stage is higher than the distant stage. There are two main barriers to early diagnosis – the lack of ovarian cancer awareness and a lack of an ovarian cancer screening test. Historically, ovarian cancer was considered a “silent killer” because symptoms were not thought to develop until the cancer was advanced and the chance of cure was poor. However, recent studies have shown this term is untrue and that certain symptoms are more likely to occur in women with ovarian cancer than in women in the general population. These symptoms include bloating, pelvic or abdominal pain, difficulty eating or feeling full quickly, and urinary symptoms (urgency or frequency) (3). Although these symptoms are non-specific and could be related to other conditions, women should report them to their doctor to be evaluated.

The Ovarian Cancer National Alliance (OCNA) is a non-profit foundation that has been educating women and health care practitioners about the risk factors for and early symptoms of ovarian cancer for more than a decade (3). The need for ovarian cancer awareness and early detection has also garnered attention from the National Cancer Institute (NCI), which organized the Ovarian Cancer Specialized Programs of Research Excellence (SPORE) program that was initiated in 1999. Other NCI programs include the Division of Cancer Prevention's Prostate, Lung, Colon, and Ovarian Cancer (PLCO) Screening Trial and the NCI Intramural Program, which enrolled participants from 1993-2001 and will follow up patients through 2016 (8,7). The OCNA developed a media campaign called “Turn UP the Volume!” to increase awareness about the symptoms for ovarian cancer (3).

Currently, no routine screening test exists for ovarian cancer. If a woman is experiencing ovarian cancer symptoms or has a strong family history or genetic predisposition (such as a BRCA1 or BRCA2 mutation), doctors may monitor her with one or a combination of tests, including the

CA125 blood test, transvaginal ultrasound, and pelvic exam (3). The need for a screening test has generated interest in finding a biomarker for ovarian cancer.

Researchers are making strides toward finding biomarkers that could be used to help detect ovarian cancer at an early stage. The biomarkers that are being tested are HE4, mesothelin, the kallikreins, and proteomic markers (9). The four Ovarian SPOREs in the U.S. have banded together to evaluate these novel markers using the PLCO's resources (9). Researchers would also like to be able to find ovarian lesions at a pre-cancerous stage that may only require the removal of the affected ovary as the main treatment (9). It is hoped that the research being done on ovarian cancer today will result in better early detection with ovary-specific biomarkers and fewer reoccurrence of this cancer.

The menstrual cycle involves a complex interaction of hormones. With each menstrual cycle, there is the potential for a single epithelial cell to develop into a cancer cell. A leading hypothesis that links ovarian cancer with ovum release suggests that this release, and rapid cell growth on the surface of the ovary, may lead to changes of the surface cells that can lead to cancer (6). The Massachusetts data on histology demonstrate that epithelial cancer represents the majority of the ovarian cancer cases (87.4%) (Figure 5).

Ovarian cancer is still considered a rare cancer; the probability of developing ovarian cancer over an 85-year lifespan is only 1.6% (Table 1) and the probability of dying from ovarian cancer over that lifespan is only 1.1% (Table 2). The potential for developing ovarian cancer can be decreased by suppressing the hormonal cycle that the ovaries go through each month. Oral contraceptives and female sterilization have been the two leading forms of contraception in women since 1982 (10).

Oral contraception has been around for over 50 years. Although the FDA approved the first oral contraceptive in 1960, contraceptives were not available to married women in all states until 1965, and were not available to unmarried women in all states until 1972 (11). Today's oral contraceptives vary in formulation, but in general contain lower doses of estrogen and progestins (progesterone) than did earlier forms of the drug (11). Oral contraceptives are only considered protective for ovarian cancer if they are used continuously over at least a 5-year time period (5,6,10,15,16). The protective effects are due to the reduction in estrogen levels in the ovaries and prevention of ovulation (4). In 2002, among U.S. women aged 15-44, white, non-Hispanic women (87%) tended to have used oral contraception more often than black, non-Hispanic women (79%) (10). Both white, non-Hispanic women and black, non-Hispanic women have, over time, increased their use of oral contraceptives (10).

A hysterectomy with ovary removal reduces the risk of ovarian cancer. The majority of hysterectomies are done due to non-cancerous conditions (12,13,14). The average annual rates for hysterectomies in the Northeast region of the United States were 3.9 per 1,000 women for 1980-1993, 4.3 per 1,000 women for 1994-1999, and 4.3 per 1,000 women for 2000-2004 (12,13,14). The percentage of hysterectomies performed in which both ovaries were removed more than doubled over the past 34 years, from 25% in 1965 to 55% in 1999, according to national statistics (10). That study's authors conservatively estimate that, today, approximately half of all women have their ovaries removed during a hysterectomy (10). As more information becomes available

about how the absence of hormones can affect the heart and bone absorption, some doctors and researchers question the advisability of including ovary removal when hysterectomies are performed (15). The overall estimated hysterectomy rate was 6.2 per 1,000 for black, non-Hispanics, 5.3 per 1,000 for white, non-Hispanics, and 5.9 per 1,000 for other races, but the differences in the overall rates were not statistically significant. However, differences in hysterectomy rates among black, non-Hispanic and white, non-Hispanic women aged 35-39 (12.5 and 8.3, respectively) and 40-44 years (16.8 and 10.8, respectively) were statistically significant ( $p < 0.05$ ) (13). The higher prevalence of leiomyomas or fibroids among black, non-Hispanic women might contribute to the higher proportion of abdominal hysterectomies in this group (13).

Surgical sterilization is an alternative method of contraception that can decrease the risk of ovarian cancer. In a prospective study, a 33% decrease in the risk of ovarian cancer was observed among women who underwent surgical sterilization or tubal ligation [after adjusting the data for oral contraceptive use, parity (the number of children born alive that a woman has delivered), and other ovarian cancer risk factors] (5). This finding was supported by large prospective U.S. and Danish cohort studies (16,17). After rising from 16% to 42% between 1965 and 1988, the prevalence of surgical sterilization among married women 15–44 years old remained stable at 41% in 1995 according to the 1965 National Fertility Study and the 1973, 1982, 1988, and 1995 cycles of the National Survey of Family Growth (17). Approximately 50% of women between the ages of 40-44 used female sterilization as a form of contraception in the United States in 2002 (10). Over time, black, non-Hispanic women overall experienced the largest increase in surgical sterilization or tubal ligation, from 20% in 1973 to nearly 50% in 1995 (17). Tubal ligation accounted for the bulk of the increase in surgical sterilization from 1973 to 1995, with the proportion of black, non-Hispanic women with tubal ligations quadrupling from 10% to 41%. The increase in hysterectomies and surgical sterilization may have decreased the risk of ovarian cancer among black, non-Hispanic women.

The women who were 84 years of age in 2005 were 44 years of age in 1965. Their choices of birth control would have been limited during the time when they were choosing to have families. Women who were younger at that time had more choices of birth control, thereby allowing them to plan out their families. The trend of increased use of contraception in the form of oral contraceptive, surgical sterilization, and hysterectomies over the years may be reflected in the age-specific rates (Figure 2) and the average annual age-adjusted rates by race/ethnicity (Figure 3). As each method of contraception became more accepted by the health care providers and general population, there may be a cumulative effect of slight decreases in ovarian cancer among later generations of women.

Differences by race/ethnicity are also seen in the rates of oral contraceptive use, surgical sterilizations, and hysterectomies, with black, non-Hispanics having higher percentages of surgical procedures. Black, non-Hispanic women in 1995 were consistently more likely than Hispanics or white, non-Hispanics to have undergone a tubal ligation, regardless of age at first birth (17). These higher rates of hysterectomies and surgical sterilizations among black, non-Hispanic women may have also helped to decrease their risk of getting ovarian cancer in their lifetimes. A benign tumor in the uterus called a fibroid can be found in a higher percentage of black, non-Hispanic women compared to their white, non-Hispanic counterparts. These tumors can cause excessive bleeding and pain, which can lead women to undergo hysterectomies. The

use of these surgical procedures and oral contraceptives may have some cumulative effect that decreases ovarian cancer among this population of women. Since 1995, there has been another procedural option called fibroid embolization that cuts off the blood supply to the fibroid (18,19). As this procedure gains in popularity, it may decrease the number of hysterectomies among black, non-Hispanic women and could indirectly affect the number of women in this population who develop ovarian cancer in the future.

Certain genes may cause a higher probability of getting breast cancer. BRCA1 and BRCA2 (breast cancer 1 and breast cancer 2) are inherited defects. Since the discovery of these defects, ovarian cancer has also been linked to them. Families with various combinations of breast cancer and ovarian cancer are possible candidates to have the gene defect (20). These genes are responsible for about 5% to 10% of all ovarian cancers (3). Women who have the BRCA1 gene mutation have a 39-60% lifetime risk of developing ovarian cancer and women with the BRCA2 gene mutation have an 11-25% lifetime risk of developing ovarian cancer (21). However, a family member with a positive test result for the BRCA1/BRCA2 gene defect may not get cancer. Women who are of Ashkenazi (Eastern European) Jewish descent and women of Norwegian, Dutch, or Icelandic ethnic or geographic origin—groups with higher rates of BRCA1/BRCA2 mutations—need to be aware of their increased genetic risk of developing ovarian cancer (22). Another genetic link to ovarian cancer is an inherited syndrome called hereditary nonpolyposis colorectal cancer (HNPCC). Women with HNPCC have about a 12% lifetime risk of ovarian cancer. It is important to note that these various genes can be associated with more than one type of cancer, but having these genes does not guarantee that a woman will develop ovarian cancer.

A diagnosis of ovarian cancer is made by looking at the cells to see what types of cells are present in the cancerous growth. There are generally three types of ovarian cancer tumors (3). *Epithelial tumors* start in the surface cells on the ovary and generally occur in postmenopausal women. *Germ cell carcinoma tumors* form in the cells that develop into the egg or ovum. These types of tumors tend to occur in younger women and there are several subtypes that can be benign. The final type of ovarian cancer, *stromal carcinoma tumors*, arises from the connective tissues that hold the ovary together and produces the hormones estrogen and progesterone (3). The majority of these types of stromal cancer are found in the earlier stages. Hormonal changes, or the interruption of menstruation, may allow germ cell and stromal carcinomas to be found in earlier stages. As noted earlier, the majority of ovarian cancer cases are epithelial in origin.

Treatment for ovarian cancer is generally a combination of surgery and chemotherapy. The histology, stage, size of tumor, age, patient's general health, and whether it is an initial diagnosis or a recurrence are used to determine the treatment (23). Histology and staging of the cancer can influence the type of chemotherapy that is used. Surgery, which is generally the first course of treatment, helps determine how much the disease has progressed and allows the surgeon to remove as much of the tumor (or as many visible tumors) as possible to make subsequent treatment on the patient as easy as possible. The outcome is generally better when the surgery is performed by a gynecologic oncologist (3). Chemotherapy is delivered intravenously and/or directly into the peritoneal (abdominal) cavity. The treatments may change depending on the clinical response. The side effects that chemotherapy may cause include hair loss, nausea, fatigue, peripheral neuropathy (numbing of hands and feet), mouth sores, and loss of concentration and forgetfulness during and after treatment (3). Ovarian cancer is very hard to treat in the later

stages. The cancer tends to reoccur and treatment options decline as the cancer becomes resistant to the types of therapy that are used. Estimated 5-year survival data by stage, which are available from the National Cancer Institute, are: localized 94%, regional 73%, distant 28%, and unstaged 27% (2).

The signs and symptoms of ovarian cancer are non-specific, but women should be educated to consult with their doctor if they develop any of them. There are many types of medical interventions and physical variations that can affect the risk of developing ovarian cancer. As medical research finds new ways to detect and treat ovarian cancer, the hope is to improve early detection and increase long-term survival.

## TECHNICAL NOTES

### *Statistical Notes*

**Age-Specific Rates** – Age-specific rates were calculated by dividing the number of people in an age group who were diagnosed with cancer or died of cancer in a given time frame by the number of people in that same age group overall in that time frame. They are presented as rates per 100,000 persons and are site- and sex-specific.

**Age-Adjusted Incidence/Mortality Rates** – An age-adjusted incidence or mortality rate is a weighted average of the age-specific rates, where the weights are the proportions of persons in the corresponding age groups of a standard 100,000 population. The potential confounding effect of age is reduced when comparing age-adjusted rates for populations with different age-structures. The 2000 U.S. Census Bureau population distribution was used as a standard. Rates were age-adjusted using eighteen 5-year age groups. Age-adjusted rates can only be compared if they are adjusted to the same standard population.

**Joinpoint Regression Analysis of Cancer Trends** – These trend analyses were calculated using the Joinpoint Regression software developed by SEER (24). The software calculates the number and location (in time) of points where trends change direction (*joinpoints*). At each joinpoint, the trend may change in different ways. The joinpoint regression model describes the trend as a sequence of linear segments between corresponding joinpoints, so that each segment has an associated annual percent change positive trend, negative trend, or no trend.

**Probability of Being Diagnosed with or Dying from Cancer** – These probabilities were calculated using the DevCan software developed by SEER (25). The results are presented in a table showing the probability (as a percentage) of a person of a specified age group and sex being diagnosed with a cancer within a specific number of years or within his or her remaining lifetime. The lifetime was restricted to age 85 in these analyses.

### *Race/Ethnicity Classification*

The MCR uses an algorithm developed by the North American Association of Central Cancer Registries (NAACCR) called the NAACCR Hispanic Identification Algorithm (NHIA) to help classify Hispanic ethnicity. A modified algorithm for Massachusetts is only applied to cases with an unknown Spanish/Hispanic origin or cases that have been classified as Hispanic based on a Spanish surname only. The algorithm uses last name, maiden name, birthplace, race, and sex to determine the ethnicity of these cases.

The race/ethnicity categories presented in this report are mutually exclusive. Cases and deaths are only included in one race/ethnicity category. The race/ethnicity tables include the categories white, non-Hispanic; black, non-Hispanic; Asian, non-Hispanic; and Hispanic. The total population in Massachusetts also includes persons of unknown race/ethnicity and American Indians. As a result, the number of cases for the total population is not the sum of cases by race/ethnicity.

## *Population Estimates*

All of the population estimates used for this report were generated using the Massachusetts Community Health Information Profile (MassCHIP). The Research and Epidemiology division of the Bureau of Health Information, Statistics, Research, and Evaluation provides these data to MassCHIP using the Modified Age-Race-Sex (MARS) file from the National Center for Health Statistics (NCHS) in collaboration with the Census Bureau's Population Estimation Program. The NCHS reallocates the multiple race categories from the Census Bureau population estimates file to create four mutually exclusive race categories that are consistent with the race categories used to collect cancer incidence and cancer mortality data.

## *Data Sources*

**Massachusetts Cancer Registry:** The MCR collects reports of newly diagnosed cancer cases. Facilities reporting to the MCR in 2005 included 74 Massachusetts acute care hospitals, one medical practice association, pathology laboratories, one radiation/oncology facility, endoscopy centers, dermatologists, and urologists. Reports from dermatologists' offices and dermatopathology laboratories, particularly on cases of melanoma, have been collected by the MCR since 2001. Reports from urologists' offices have been collected by the MCR since 2002. Currently, the MCR collects information on *in situ* and invasive cancers and benign tumors of the brain and associated tissues. The MCR does not collect information on basal and squamous cell carcinomas of the skin.

**Surveillance, Epidemiology and End Results (SEER) Program:** National data on cancer incidence and mortality are from the National Cancer Institute's SEER Program, an authoritative source of cancer incidence data in the United States. The 24-year comparison was done using data from nine areas (Atlanta, Connecticut, Detroit, Hawaii, Iowa, New Mexico, San Francisco-Oakland, Seattle-Puget Sound, and Utah) that have been collecting collaborative data since 1975 and cover about 14% of the United States population. SEER rates are presented per 100,000 persons and are age-adjusted to the 2000 United States standard population. SEER does not report rates with fewer than 25 cases for a time interval. SEER rates were available to the year 2005 at the time of this publication.

**Massachusetts Registry of Vital Records and Statistics:** Massachusetts death data were obtained from the Massachusetts Registry of Vital Records and Statistics, which has legal responsibility for collecting reports of deaths in this state. Death reports from 2002 to 2006 were coded using the International Classification of Diseases, Tenth Revision (ICD-10) (26). The cancer site/type groups for deaths in this report were based on cancer site/type categories from the SEER Program. These SEER cancer site/type definitions are the standard categories commonly used by cancer registries.

## *Disease Coding*

**International Classification of Disease for Oncology, Third Edition (ICD-O-3) Implementation (27):** The International Classification of Diseases for Oncology, Third Edition (ICD-O-3) (28), was implemented in North America with cases diagnosed as of January 1, 2001. Cancer cases diagnosed before this date were classified according to the International Classification of Diseases

for Oncology, Second Edition (ICD-O-2). With the advances in diagnostic techniques over the past decade, the International Agency for Research on Cancer (IARC) and the cancer division of the World Health Organization (WHO) found it necessary to revise and update the ICD-O-2. As a result, the new edition (ICD-O-3) contains more specific information about certain cancers. The most important changes between the second and the third editions include:

- Certain hematopoietic diseases are now considered to be “malignant,” whereas previously they were classified as “uncertain whether benign or malignant.”
- Some neoplasms (mainly ovarian tumors) previously coded as “malignant” now revert to “uncertain whether benign or malignant.”

All cancer cases in the Massachusetts Cancer Registry database that were diagnosed prior to January 1, 2001 and coded using ICD-O-2 were converted to ICD-O-3, following SEER conversion rules.

The following ICD-O-3 histologies were used in preparing Figure 5:

Epithelial: 8010-8046, 8441-8442, 8460-8462, 8470-8472, 8480-8482, 8380-8382, 8140-8260, 8050-8074, 8562, 8120, 8130, 9014, 8313, 9015, 8800, 8801, 9000, 8310, 8323, 8440, 8450, 8490, 8570, 8574

Germ: 9060-9091, 8340, 8981, 9100

Stromal: 8600-8670, 8810, 8935, 8930

Other: 8890-8901, 9050, 9110, 9120, 9580, 8000, 8001, 8504, 8560, 8720, 8806, 8933, 8950, 8951, 8980

**Any comparison of annual incidence rates between the year 2000 and 2001 (or comparison of the rates presented in this report with previous reports) should take into the account the changes described above.**

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