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# Uptake of clinical genetic testing for ovarian cancer in Ontario: A population-based study

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## Abstract

**Background—**Approximately 13% of ovarian cancers in Canada are attributable to a mutation in BRCA1 or BRCA2. In 2001, genetic testing for BRCA1 and BRCA2 became freely available to all women in Ontario with a diagnosis of invasive ovarian cancer. It is unknown what proportion of women with ovarian cancer receive genetic testing as a result of this recommendation.

**Methods—**Patients in Ontario who had been diagnosed with epithelial ovarian cancer from 2002 to 2004 were identified using the Ontario Cancer Registry. Information was collected on demographic and risk factors, including information on previous testing for BRCA1 and BRCA2. Women were asked to provide a blood sample for genetic testing or to provide a genetic test result if clinical testing had been done. Genetic testing for BRCA1 and BRCA2 mutations was conducted on all blood samples.

**Results—**Of the 416 women, 80 women (19%) had undergone previous clinical genetic testing for BRCA1 and BRCA2. Of these 80 women, 30% had a positive genetic test result, compared to 5% of 336 women who had not had clinical genetic testing ($p$ <0.0001). Sixty percent of all mutations were identified within this group of 80 women.

**Conclusions—**Genetic testing is available in Ontario to all women with invasive ovarian cancer. However, only a small proportion of women are being referred for testing. This study suggests that increased public awareness directed at physicians and at women with cancer may expand the use of genetic testing.

### Keywords

BRCA1; BRCA2; Ovarian cancer; Genetic testing



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**Conflict of interest statement**
The authors declare that there are no conflicts of interest.

## Introduction

Mutations in BRCA1 and BRCA2 account for the majority of cases of hereditary ovarian cancer [1]. Approximately 13% of all invasive ovarian cancers are attributable to a BRCA1 or BRCA2 mutation. The prevalence of BRCA1 and BRCA2 mutations is much higher in Jewish women with ovarian cancer, ranging from 29 to 41% [2,3]. Previous research has shown that there are several predictors of a BRCA1 or BRCA2 mutation in women with ovarian cancer including age, histology, family history of cancer, and personal history of breast cancer [10].

Genetic testing for BRCA1 and BRCA2 mutations is offered to high-risk women (primarily based on family history of breast and/or ovarian cancer) in the context of the universal healthcare system in Ontario, Canada. Because of the high appreciable prevalence of BRCA1 and BRCA2 mutations in women with ovarian cancer, the Ontario Ministry of Health extended the criteria for genetic testing in 2001 to include all women with a diagnosis of invasive ovarian or fallopian tube cancer. It is not yet known what is the uptake of genetic testing in women diagnosed with invasive ovarian cancer in Ontario after the introduction of this policy.

The process for genetic testing for BRCA1 and BRCA2 begins with a referral from a treating physician to a cancer genetics centre, where genetic counselling is initiated. Individuals who qualify for genetic testing provide blood samples, which are sent for testing to an Ontario Ministry of Health laboratory. In general, patients rely on physician referral for genetic testing. The purpose of this study was to assess to what extent the expanded policy for genetic testing for BRCA1 and BRCA2 in Ontario, Canada is reflected in patient referral patterns. We sought to determine which factors predict the uptake of genetic testing amongst women who are newly diagnosed with ovarian or fallopian tube cancer in Ontario.

## Study population

The study protocol received full ethics approval from the participating institutions. All Ontario patients who had been diagnosed with epithelial ovarian or fallopian tube cancer, from 2002 to 2004, were identified using the Ontario Cancer Registry. For each case, the pathology report was reviewed to determine eligibility and histological type of the tumor. Eligible patients were 20 to 79 years of age and were residents of Ontario at the time of diagnosis. The attending physician of each eligible subject was contacted to obtain consent for patient contact. Upon receipt of physician consent, the subject was contacted and offered participation in the study.

In total, 1326 cases were identified by pathology report. Of these, 334 were deceased, 199 were without physician consent, and 155 were ineligible or could not be included for other reasons (14 had a previous ovarian cancer diagnosis, 26 had a different histology, 13 were non-residents of Ontario, 44 were too ill, 34 had a language barrier, 9 physicians were unable to be located, and 15 patients were lost to contact). Of the 638 eligible cases with physician consent, 550 women were contacted, and of these, 491 agreed to participate (89.3%). Detailed information was obtained on family history of cancer and medical and reproductive histories. Each woman was asked if she had previously undergone genetic testing at a provincial testing laboratory and if so, what was the result. If the woman had not previously been tested she was offered testing in the context of the research study, and asked whether or not she wished to receive the test result. If she had previously been tested, a copy of the genetic test report was requested. 65 women provided neither a blood sample nor a genetic test report and were excluded. A blood sample was provided by 419 women (including seven with previous genetic testing) and a previous genetic test report was

provided by seven women. Ten women were excluded because the tumour was found to be primary peritoneal cancer. Therefore, complete data were available on 416 women.

## BRCA1 and BRCA2 analysis

Lymphocyte DNA was prepared from whole blood. All samples were screened for 11 common mutations (seven in BRCA1 and four in BRCA2), including the three mutations common to Ashkenazi Jews and others of Eastern European ancestry and six mutations previously identified in the French Canadian population [4]. Nine of these mutations were assayed using a rapid multiplex method [5]. We tested separately for the presence of the 6-kilobase (kb) duplication in exon 13 of BRCA1 [6] and for the mutation 546G>T in exon 7 of BRCA1. If no mutations were found, exon 11 of BRCA1 and exons 10 and 11 of BRCA1 were then screened with the protein truncation test. Primer sequences used to amplify overlapping fragments were obtained from the Breast Cancer Information Core (BIC). The protein truncation test, using [$^{35}$S]methionine and [$^{35}$S]cysteine (New England Nuclear, Wellsley, MA) for protein truncation detection, was performed with the TNT rabbit reticulocyte lysate system (Promega, Madison, WI).

Patients who were not found to carry mutations by the preceding methods were then screened for additional BRCA1 and BRCA2 mutations. For other BRCA1 mutations, fluorescent multiples denaturing gradient gel electrophoresis (DGGE) [7] was used. All the remaining codon exons, the exon–intron boundaries, and the beginning and end of exon 11 were included; noncoding exons 1a and 1b and the noncoding part of exon 24 were excluded. For the additional BRCA2 mutations, denaturing high-performance liquid chromatography (DHPLC) was used to screen the remaining coding exons and exon–intron boundaries [8].

All variants identified by protein truncation test, DGGE, and DHPLC were confirmed by direct DNA sequencing (Promega). All the observed mutations included in this report are highly likely to be deleterious. The various founder mutations are established to be deleterious, and the protein truncation test identifies mutations associated with shortened, nonfunctional proteins. The mutations found by DGGE or DHPLC are substitutions producing premature termination codons, which are also associated with nonfunctional truncated proteins, or are mutations that have been reported previously and as documented in the BIC databases or elsewhere [9], are deleterious. For women who wanted to receive the genetic test results, a referral was made to a local cancer genetics clinic for full genetic counselling.

## Statistical analysis

We determined the number of women who had undergone genetic testing prior to the study. We then compared the proportion of positive genetic tests results between women tested previously and those tested as a component of the research study. We compared the women who had and who had not received prior genetic testing for a number of demographic and other variables, including age, education, area of residence and treating hospital.

## Results

Four hundred and sixteen women participated in the study; 397 of the women had ovarian cancer and 19 had fallopian cancer. The mean age of the women at time of cancer diagnosis was 57.5 years (range 23–79) (Table 1). The majority of women were white and had an education beyond high school. Seven percent of the women had a family history of ovarian cancer, 17% had a family history of breast cancer, and 7% had a personal history of breast

cancer (mean age of diagnosis 49.2 years). Of the 416 women, 41 (9.9%) women had a mutation in BRCA1 or BRCA2. Ninety-six percent of the women elected to receive their genetic test result.

Eighty women (19.2%) had received clinical genetic testing for BRCA1 and BRCA2 in an Ontario Ministry laboratory prior to entry into our research study. Of those who had undergone previous genetic testing for BRCA1 and BRCA2, 30% (24 of 80 women) had a positive genetic test result, compared to 5% of women (17 of 336 women) who only had research testing ($p<0.0001$). Although only 19.2% of women with ovarian cancer received clinical genetic testing prior to the study, this group represented 58.5% of the women with mutations.

We studied factors that predicted the uptake of clinical genetic testing (Table 2). A higher frequency of testing was seen among women treated at teaching hospitals (21.3%) than at community hospital (15.4%) but the difference was not statistically significant ($p=0.1$). Uptake of testing did not depend on age at ovarian cancer diagnosis ($p=0.6$) or marital status ($p=0.3$).

Factors that predicted clinical genetic testing included race, parity, histology, tumor site, and family history of breast and/or ovarian cancer. White women were more likely to have undergone genetic testing (20.6%) than were women of other races (3.1%; $p=0.05$). Women with children were more likely to have had testing (21.3%) than were women with no children (10.8%; $p=0.03$). Women with fallopian tube primaries were more likely to have undergone genetic testing (47.4%) than those with primary ovarian cancer (17.9%; $p=0.001$), and women who had cancer of serous histology were most likely to have had clinical genetic testing than were women with cancers of other types ($p<0.0001$). A positive family history of breast cancer or ovarian cancer, or a personal history of breast cancer was also predictive of clinical testing ($p<0.0001$ for each).

## Discussion

Ontario women with invasive ovarian cancer or fallopian tube cancer are now eligible for genetic testing for BRCA1 and BRCA2 mutations under the provincial health care plan. However, only 19% of the eligible women in our study had actually undergone genetic testing prior to study contact.

Risch et al. identified several predictors of a BRCA1 or BRCA2 mutation in women with ovarian cancer including age, histology, family history of cancer, and personal history of breast cancer [10]. Factors which predicted referral for clinical genetic testing in the current study included having a family history of breast cancer or ovarian cancer ($p<0.0001$), having a personal history of breast cancer ($p=0.0001$), and having a tumor with serous histology ($p<0.0001$). Although women diagnosed with ovarian cancer between the ages of 40 and 50 years have a higher risk of having a BRCA1 or BRCA2 mutation than women in other age groups, these age differences were not reflected in the testing frequencies.

Previous research has reported no differences in prevalence of BRCA1 or BRCA2 mutations between black and white women [11]. However, several reports have shown differences in uptake of genetic testing between the two groups [12–14]. We also observed that black women were less likely to have undergone genetic testing prior to our study than white women ($p=0.05$). It is unclear if this result is because few black women agreed to genetic testing when presented in the clinical setting, or if they were less likely to be offered genetic testing by their physicians. Our sample include very few black women, and future research is needed in this area. It is interesting that all of the women in this study agreed to undergo

genetic testing when it was presented to them in the research setting and almost all wished to know the result.

In total, 41 women were found to have a BRCA1 or BRCA2 mutation. Of these women, 24 (58.5%) had undergone clinical genetic testing at the time of ovarian cancer diagnosis or treatment. These 24 women were in a group of 80 women who had undergone genetic testing prior to our study. The proportion of women with a positive test is high (30%); this reflects efficiency from a laboratory perspective, but also implies that the referring physicians are being stringent in the referral patterns. Of the 336 women who had not undergone clinical testing, 17 were found to have a mutation. This is a low rate of positivity (5%), but also implies that an appreciable fraction of the women with a BRCA1 or BRCA2 mutations were not offered or had not availed themselves of testing.

To our knowledge, no other study has reported on the frequency of genetic testing for BRCA1 and BRCA2 mutations in a population-based sample of women with newly diagnosed ovarian cancer and where testing is freely available. Previous research on uptake of genetic testing has been done on individuals with a family history of cancer, including those from families with known BRCA1 or BRCA2 mutations [15–18], and on individuals attending high-risk cancer clinics [19,20]. However, for the great majority of ovarian cancer patients, there is no family history of ovarian cancer (93% in our study) or breast cancer (83% in our study). Therefore, genetic testing for BRCA1 and BRCA2 mutations might not be recommended for these patients. However, Risch et al. reported among women with ovarian cancer, 37% with BRCA1 or BRCA2 mutations had no family history [10]. Thus, omission of referral for genetic testing for such women will miss more than one-third with BRCA1 or BRCA2 mutations.

There are limitations to our study. It is possible that some women were offered genetic testing at the time of ovarian cancer diagnosis but refused. However, all of these women consented to take part in this study in which genetic testing was done, therefore, it seems unlikely that many women initially refused genetic testing and then elected for testing at the time of our study. In addition, more than 300 potentially eligible women were excluded because they had died prior to contact with our research personnel. It is unknown what proportion of these women had undergone genetic testing prior to death. It is also unclear from our analyses if practice changed after 2001 as we did not compare referral patterns pre- and post-2001.

In conclusion, this study suggests that the women at highest risk of having BRCA1 or BRCA2 mutations are adequately identified and offered genetic testing in Ontario. However, if testing was restricted to those referred by physicians because of a high risk of carrying a mutation, approximately 40% of the mutations would remain unidentified. This suggests that reliance on physicians for patient referrals for genetic testing is inadequate and that greater physician education and public awareness is needed. The medical community should seek to make women aware that genetic testing may be indicated for all women with invasive ovarian cancer. That is not to say that they should seek testing directly from the laboratory, but that they should bring this to the attention of their physicians and should request referrals to genetic counselling. In addition, future consideration should be given to pathology reports indicating that genetic referral is indicated with any serous tumor.

## References

1. Narod S, Ford D, Devilee P, Barkardottir RB, Eyfjord J, Lenoir G, et al. Genetic heterogeneity of breast-ovarian cancer revisited. Breast Cancer Linkage Consortium. Am J Hum Genet. 1995 Oct; 57(4):957–8. [PubMed: 7573057]

2. Moslehi R, Chu W, Karlan B, Fishman D, Risch H, Fields A, et al. BRCA1 and BRCA2 mutation analysis of 208 Ashkenazi Jewish women with ovarian cancer. Am J Hum Genet. 2000 Apr; 66(4): 1259–72. [PubMed: 10739756]

3. Modan B, Hartge P, Hirsh-Yechezkel G, Chetrit A, Lubin F, Beller U, et al. Parity, oral contraceptives, and the risk of ovarian cancer among carriers and noncarriers of a BRCA1 or BRCA2 mutation. N Engl J Med. 2001 Jul 26; 345 (4):235–40. [PubMed: 11474660]

4. Tonin PN, Mes-Masson AM, Futreal PA, Morgan K, Mahon M, Foulkes WD, et al. Founder BRCA1 and BRCA2 mutations in French Canadian breast and ovarian cancer families. Am J Hum Genet. 1998 Nov; 63(5):1341–51. [PubMed: 9792861]

5. Kuperstein G, Foulkes WD, Ghadirian P, Hakimi J, Narod SA. A rapid fluorescent multiplexed-PCR analysis (FMPA) for founder mutations in the BRCA1 and BRCA2 genes. Clin Genet. 2000 Mar; 57(3):213–20. [PubMed: 10782928]

6. Puget N, Sinilnikova OM, Stoppa-Lyonnet D, Audoynaud C, Pages S, Lynch HT, et al. An Alu-mediated 6-kb duplication in the BRCA1 gene: a new founder mutation? Am J Hum Genet. 1999 Jan; 64(1):300–2. [PubMed: 9915971]

7. Kuperstein G, Jack E, Narod SA. A fluorescent multiplex-DGGE screening test for mutations in the BRCA1 gene. Genet Test. 2006 Spring;10 (1):1–7. [PubMed: 16544996]

8. Gross E, Arnold N, Pfeifer K, Bandick K, Kiechle M. Identification of specific BRCA1 and BRCA2 variants by DHPLC. Hum Mutat. 2000 Oct; 16 (4):345–53. [PubMed: 11013445]

9. Peelen T, van Vliet M, Bosch A, Bignell G, Vasen HF, Klijn JG, et al. Screening for BRCA2 mutations in 81 Dutch breast–ovarian cancer families. Br J Cancer. 2000 Jan; 82(1):151–6. [PubMed: 10638982]

10. Risch HA, McLaughlin JR, Cole DE, Rosen B, Bradley L, Fan I, et al. Population BRCA1 and BRCA2 mutation frequencies and cancer penetrances: a kin-cohort study in Ontario, Canada. J Natl Cancer Inst. 2006 Dec 6; 98(23):1694–706. [PubMed: 17148771]

11. Malone KE, Daling JR, Doody DR, Hsu L, Bernstein L, Coates RJ, et al. Prevalence and predictors of BRCA1 and BRCA2 mutations in a population-based study of breast cancer in white and black American women ages 35 to 64 years. Cancer Res. 2006 Aug 15; 66(16):8297–308. [PubMed: 16912212]

12. Halbert CH, Kessler L, Stopfer JE, Domchek S, Wileyto EP. Low rates of acceptance of BRCA1 and BRCA2 test results among African American women at increased risk for hereditary breast–ovarian cancer. Genet Med. 2006 Sep; 8(9):576–82. [PubMed: 16980814]

13. Armstrong K, Micco E, Carney A, Stopfer J, Putt M. Racial differences in the use of BRCA1/2 testing among women with a family history of breast or ovarian cancer. JAMA. 2005 Apr 13; 293(14):1729–36. [PubMed: 15827311]

14. Susswein LR, Skrzynia C, Lange LA, Booker JK, Graham ML III, Evans JP. Increased uptake of BRCA1/2 genetic testing among African American women with a recent diagnosis of breast cancer. J Clin Oncol. 2008 Jan 1; 26 (1):32–6. [PubMed: 18165638]

15. Biesecker BB, Ishibe N, Hadley DW, Giambarresi TR, Kase RG, Lerman C, et al. Psychosocial factors predicting BRCA1/BRCA2 testing decisions in members of hereditary breast and ovarian cancer families. Am J Med Genet. 2000 Aug 14; 93(4):257–63. [PubMed: 10946349]

16. Brooks L, Lennard F, Shenton A, Lalloo F, Ambus I, Ardern-Jones A, et al. BRCA1/2 predictive testing: a study of uptake in two centres. Eur J Hum Genet. 2004 Aug; 12(8):654–62. [PubMed: 15138457]

17. Julian-Reynier C, Sobol H, Sevilla C, Nogues C, Bourret P. Uptake of hereditary breast/ovarian cancer genetic testing in a French national sample of BRCA1 families. The French Cancer Genetic Network. Psychooncology. 2000 Nov–Dec; 9(6):504–10. [PubMed: 11180585]

18. Lerman C, Narod S, Schulman K, Hughes C, Gomez-Caminero A, Bonney G, et al. BRCA1 testing in families with hereditary breast–ovarian cancer: a prospective study of patient decision making and outcomes. JAMA. 1996; 275(24):1885–92. [PubMed: 8648868]

19. Armstrong K, Calzone K, Stopfer J, Fitzgerald G, Coyne J, Weber B. Factors associated with decisions about clinical BRCA1/2 testing. Cancer Epidemiol, Biomarkers Prev. 2000 Nov; 9(11): 1251–4. [PubMed: 11097234]

20. Lee SC, Bernhardt BA, Helzlsouer KJ. Utilization of BRCA1/2 genetic testing in the clinical setting: report from a single institution. Cancer. 2002 Mar 15; 94(6):1876–85. [PubMed: 11920551]

## Table 1

Characteristics of the study sample

| Variable | Description | Frequency (%) |
|---|---|---|
| Age at diagnosis | <=40 | 28 (6.7%) |
| | 41–50 | 93 (22.4%) |
| | 51–60 | 127 (30.5%) |
| | >=61 | 168 (40.4%) |
| | Mean age at diagnosis | 57.5 (23–79) |
| Race | White | 384 (92.3%) |
| | Black | 3 (0.7%) |
| | Asian | 29 (7.0%) |
| Histology | Clear cell | 39 (9.4%) |
| | Endometroid | 89 (21.4%) |
| | Mucinous | 21 (5.1%) |
| | Papillary serous | 222 (53.4%) |
| | Other | 45 (10.8%) |
| Tumour site | Fallopian | 19 (4.6%) |
| | Ovary | 397 (95.4%) |
| Number of daughters | 0 | 162 (38.9%) |
| | 1 | 135 (32.5%) |
| | 2 | 91 (21.9%) |
| | 3 | 20 (4.8%) |
| | 4 | 7 (1.7%) |
| | 5 | 1 (0.2%) |
| Number of sons | 0 | 166 (39.9%) |
| | 1 | 145 (34.9%) |
| | 2 | 74 (17.8%) |
| | 3 | 22 (5.3%) |
| | 4 | 8 (1.9%) |
| | 5 | 1 (0.2%) |
| Family history of ovarian cancer | No | 387 (93.0%) |
| | Yes | 29 (7.0%) |
| Family history of breast cancer | No | 344 (82.7%) |
| | Yes | 72 (17.3%) |
| Personal history of breast cancer | No | 387 (93.0%) |
| | Yes | 29 (7.0%) |
| Level of education | 11 years or less | 103 (24.8%) |
| | 12 years or completed high school | 114 (27.5%) |
| | Some or all community college or technical school | 123 (29.6%) |
| | Completed college or university | 57 (13.7%) |
| | Completed graduate or professional degree | 15 (3.6%) |
| | Don’t know | 3 (0.7%) |

| Variable | Description | Frequency (%) |
|---|---|---|
| | Missing | 1 |
| Marital status | Married or common law | 279 (67.2%) |
| | Divorced or separated or widowed | 97 (23.4%) |
| | Never married | 39 (9.4%) |
| | Missing | 1 |
| Genetic test results | Negative | 375 (90.1%) |
| | BRCA1 | 29 (7.0%) |
| | BRCA2 | 11 (2.6%) |
| | BRCA1+2 | 1 (0.2%) |

## Table 2

Demographic characteristics of women with and without previous genetic testing

| Variable | Those not tested previously *N*=336 (80.8%) | Those tested previously *N*=80 (19.2%) | *P* |
|---|---|---|---|
| *Date of ovarian cancer diagnosis* | 2003.3 (2002–04) | 2003.4 (2002–04) | 0.75 |
| *Hospital type* | | | |
| Community | 121 (84.6%) | 22 (15.4%) | |
| Teaching | 215 (78.8%) | 58 (21.3%) | 0.15 |
| *Age at diagnosis* | | | |
| <=40 | 23 (82.1%) | 5 (17.9%) | |
| 41–50 | 73 (78.5%) | 20 (21.5%) | |
| 51–60 | 99 (78.0%) | 28 (22.1%) | |
| >=61 | 141 (83.9%) | 27 (16.1%) | 0.56 |
| Mean age | 57.8 (23–79) | 56.4 (23–79) | 0.32 |
| *Race* | | | |
| White | 305 (79.4%) | 79 (20.6%) | |
| Black | 3 (100%) | 0 (0.0%) | 0.05 |
| Asian | 28 (96.6%) | 1 (3.5%) | |
| *Parity* | | | |
| No | 74 (89.2%) | 9 (10.8%) | |
| Yes | 262 (78.7%) | 71 (21.3%) | 0.03 |
| Mean number of children (if parous) | 2.4 (1–7) | 2.4 (1–6) | 0.99 |
| *Education* | | | |
| 11 years or less | 91 (88.4%) | 12 (11.7%) | |
| 12 years or completed high school | 91 (79.8%) | 23 (20.2%) | |
| Community college/technical school | 100 (81.3%) | 23 (18.7%) | |
| Completed college or university | 40 (70.2%) | 17 (29.8%) | |
| Completed graduate/prof. degree | 13 (86.7%) | 2 (13.3%) | |
| Don’t know | 1 (33.3%) | 2 (66.7%) | |
| Missing | 0 | 1 | 0.03 |
| *Marital status* | | | |
| Married or common law | 220 (78.9%) | 59 (21.2%) | |
| Divorced or separated or widowed | 83 (85.6%) | 14 (14.4%) | |
| Never married | 33 (84.6%) | 6 (15.4%) | |
| Missing | 0 | 1 | 0.29 |
| *Histology* | | | |
| Clear cell | 34 (87.2%) | 5 (12.8%) | |
| Endometroid | 79 (88.8%) | 10 (11.2%) | |
| Mucinous | 20 (95.7%) | 1 (4.8%) | |
| Papillary serous | 172 (77.5%) | 50 (22.5%) | |
| Other | 31 (68.9%) | 14 (31.1%) | 0.01 |
| *Tumour site* | | | |
| Fallopian | 10 (52.6%) | 9 (47.4%) | |

| Variable | Those not tested previously *N*=336 (80.8%) | Those tested previously *N*=80 (19.2%) | *P* |
|---|---|---|---|
| Ovary | 326 (82.1%) | 71 (17.9%) | 0.001 |
| *Family history of ovarian cancer* | | | |
| No | 323 (83.5%) | 64 (16.5%) | |
| Yes | 13 (44.8%) | 16 (55.2%) | <0.0001 |
| *Family history of breast cancer* | | | |
| No | 298 (86.6%) | 46 (13.4%) | |
| Yes | 38 (52.8%) | 34 (47.2%) | <0.0001 |
| *Personal history of breast cancer* | | | |
| No | 325 (84.0%) | 62 (16.0%) | |
| Yes | 11 (37.9%) | 18 (62.1%) | <0.0001 |
| *Mean age at breast cancer* | 53.0 (34–73) | 46.9 (24–75) | 0.22 |