Published in final edited form as:

JAMA Oncol. 2017 January 01; 3(1): 36–41. doi:10.1001/jamaoncol.2016.3022.

Subsequent Breast Cancer Risk Following Diagnosis of Atypical Ductal Hyperplasia on Needle Biopsy

Tehillah S Menes, MD¹, Karla Kerlikowske, MD², Jane Lange, PhD³, Shabnam Jaffer, MD⁴, Robert Rosenberg, MD⁵, and Diana L. Miglioretti, PhD^{6,7}

¹Department of Surgery, Tel Aviv-Sourasky Medical Center, Tel-Aviv, Israel and Sackler School of Medicine, Tel Aviv, Israel

²Departments of Medicine and Epidemiology and Biostatistics, University of California, San Francisco, CA, USA

³Fred Hutchinson Cancer Research Center, 1100 Fairview Ave N, Seattle, WA, USA

⁴Department of Pathology, The Mount Sinai Medical Center, New York, NY, USA

⁵Radiology Associates of Albuquerque, and Department of Radiology, University of New Mexico, Albuquerque, NM, USA

⁶Group Health Research Institute, Group Health Cooperative, Seattle, WA, USA

⁷Division of Biostatistics, Department of Public Health Sciences, UC Davis School of Medicine

Abstract

Background—Atypical ductal hyperplasia (ADH) is a known strong risk factor for breast cancer. Published risk estimates are based on cohorts that included women diagnosed prior to the widespread use of screening mammograms and do not differentiate between the methods used to diagnose ADH, which may be related to size of the ADH focus. These risks may overestimate the risk of women currently diagnosed with ADH. We sought to examine the risk of invasive cancer associated with ADH diagnosed on core needle biopsy versus excisional biopsy.

Design—Cohort study comparing ten-year cumulative risk of invasive breast cancer in women undergoing mammography with and without a diagnosis of ADH.

Setting—Five breast imaging registries that participate in the National Cancer Institute–funded Breast Cancer Surveillance Consortium (BCSC).

Participants—Women undergoing mammography in the BCSC.

Exposure—Diagnosis of ADH on core needle biopsy or excisional biopsy in women undergoing mammography.

Main outcome—Ten-year cumulative risk of invasive breast cancer risk.

Corresponding author: Tehillah S. Menes, Department of Surgery, Tel Aviv Sourasky Medical Center, 6 Weizmann St. Tel Aviv, Israel 64239, tehillahm@tlvmc.gov.il, Phone: 972-3-6974711, Fax: 972-3-6974635.

Potential conflict of interest: None.

Results—The sample included 955,331 women with 1,727 diagnoses of ADH. From 1996 to 2012, the proportion of ADH diagnosed by core needle biopsy increased from 21% to 77%. Ten years following a diagnosis of ADH, the cumulative risk of invasive breast cancer was 2.6 (95% CI 2.0, 3.4) times higher than risk in women with no ADH. ADH diagnosed via excisional biopsy was associated with an adjusted HR of 3.0 (95% CI 2.0, 4.5), and via core needle biopsy, with an HR of 2.2 (95% CI 1.5, 3.4). Ten years after an ADH diagnosis, an estimated 5.7% (95% CI 4.3, 10.1) of women were diagnosed with invasive cancer. Women with ADH diagnosed on excisional biopsy had a slightly higher risk (6.7 %; 95% CI 3.0, 12.8) compared to those with ADH diagnosed via core needle biopsy (5.0%; 95% CI 2.2, 8.9).

Conclusions—Current 10-year risks of invasive breast cancer after a diagnosis of ADH may be lower than previously reported. The risk associated with ADH is slightly lower for women diagnosed by needle core biopsy as compared to excisional biopsy.

Introduction

Atypical ductal hyperplasia (ADH) is a known risk factor for breast cancer¹. In a recent report the projected cumulative incidence of breast cancer (invasive and ductal carcinoma *in situ*) after ADH was 29% over 25 years². Most of the studies reporting on the increased risk of breast cancer in women diagnosed with ADH were done prior to the widespread use of screening mammograms and core needle biopsies to evaluate non-palpable suspicious lesions. Today ADH is more often detected by a needle biopsy of an imaging finding than by excisional biopsy. With the increased use of needle biopsies for imaging abnormalities and improvement in imaging technologies, very small foci of ADH are diagnosed. The significance of these small ADH foci is not clear; however, common practice is to view these women at increased risk of breast cancer and offer them increased surveillance and risk reducing strategies^{3–5}.

The purpose of this study was to assess the cumulative invasive breast cancer risk in women diagnosed with ADH comparing detection by core needle biopsy versus excisional biopsy. As an association between number foci of ADH and subsequent risk of breast cancer was reported², we speculated that the risk associated with small foci of ADH detected on core needle biopsy may be lower than risks previously reported from general populations of women diagnosed with ADH.

We used data prospectively collected by the Breast Cancer Surveillance Consortium (BCSC) to compare the risk of invasive breast cancer in women diagnosed with ADH on core needle biopsy versus excisional biopsy and to compare these risks to risk in women undergoing mammography without an ADH diagnosis.

Methods

Data sources

Five breast imaging registries that participate in the National Cancer Institute–funded BCSC (<http://breastscreening.cancer.gov>) were included: the Carolina Mammography Registry, Group Health Cooperative in Washington, the New Hampshire Mammography Network, the New Mexico Mammography Project, and the Vermont Breast Cancer Surveillance System.

These registries collect information on mammographic examinations done in their defined catchment areas. Each mammography registry annually links women in its registry to a state tumor registry or regional Surveillance, Epidemiology and End Results program that collects population-based cancer data and pathology databases that collect information on both benign and malignant diagnoses. The BCSC Statistical Coordinating Center pooled and analyzed the data. Each BCSC registry and the Statistical Coordinating Center have received institutional review board approval for either active or passive consenting processes or a waiver of consent to enroll participants, link data, and perform analytic studies. All procedures comply with the Health Insurance Portability and Accountability Act, and all registries and the Statistical Coordinating Center have received federal certificates of confidentiality and other protection for the identities of women, physicians, and facilities studied by this research.

Data collection

Two datasets were analyzed from the BCSC. The first dataset was created to estimate rates of ADH per 10,000 screening mammograms by calendar year and included screening mammograms performed between 1994 and 2011 in women age 35 or older with at least one prior screening mammogram within 5 years and no personal history of breast cancer, LCIS, or breast augmentation. Screening mammograms were defined using the standard BCSC definition (http://breastscreening.cancer.gov/data/bcsc_data_definitions.pdf). Exams were excluded if the date through which biopsy or pathology records were considered to be at least 94% complete was less than a year following a screening mammogram. In the dataset, the most proximal screening mammogram within a year before a biopsy diagnosing ADH was associated with the ADH diagnosis.

The second dataset (1994–2012) was created to estimate the risk of invasive breast cancer in the ten years following an ADH diagnosis, compared to the risk in the general population of women undergoing screening mammography, with no diagnosis of ADH. Women were excluded if before entering the BCSC they had a biopsy or a diagnosis of breast cancer, ductal carcinoma *in situ* (DCIS), or lobular carcinoma *in situ* (LCIS). Each woman contributed one or two observations. The follow-up for the first record started six months after the first screening or diagnostic mammogram that occurred when she entered the BCSC to exclude breast cancers associated with the index mammogram. The follow-up for the second record started six months after a biopsy detecting ADH.

For each observation, follow-up continued for up to 10 years after the index date and ended at a diagnosis of invasive breast cancer (the event of interest) or a censoring event, including diagnosis of DCIS or LCIS; end of complete cancer capture; death, or exit from the cohort. Characteristics of the woman and mammogram modality (digital versus film) were collected at the preceding mammogram most proximal to study entry. The type of biopsy detecting ADH (core needle versus excisional) and all subsequent excisional biopsies in a six-month follow-up were recorded.

Statistical analysis

Crude rates of ADH diagnoses per 10,000 screening mammograms by screening mammogram calendar year were calculated using the first analysis dataset. Using the second dataset the fraction of ADH diagnosed by core needle versus excisional biopsies across study years was calculated.

Partly conditional Cox proportional hazards models were used to estimate relative rates of invasive breast cancer after a screening mammogram without a subsequent ADH diagnosis versus an ADH diagnosis in the 10-year follow-up period⁶. Since the same woman may contribute two observations, standard errors were based on a robust sandwich estimator to account for woman-level clustering⁷. Separate models were fit to examine the association between rates of invasive breast cancer and ADH diagnosis according to mode of ADH detection (core needle biopsy vs. excisional biopsy). Core needle biopsy refers to all types of percutaneous biopsies in which breast tissue is procured for pathology (including core and vacuum biopsies). Fine needle aspiration and biopsies of unknown type (N= 34) were excluded from this analysis.

All models were adjusted for age at mammogram or ADH diagnosis (linear and quadratic terms), race/ethnicity, family history, menopausal status/hormone therapy (HT), mammogram modality (digital vs. film) and BI-RADS breast density. Using time-dependent partly conditional Cox models, the association between time since diagnosis (modeling categorical time as 0–2, 2–5 and 5+ years post diagnosis), and risk associated with ADH were examined.

Ten-year probability of an invasive breast cancer by ADH status, mode of ADH detection, and mammogram modality leading to ADH detection, was calculated using the estimated hazard ratios and the empirical estimate of baseline hazard, assuming the covariate distribution observed at baseline. Standard errors for these estimates were calculated via a parametric bootstrap, via resampling Cox regression coefficient estimates from a multivariate normal distribution, with mean and covariance set at the estimated values. All analyses were conducted in R 3.2.2 (R Core Team (2015) R: A language and environment for statistical computing. R foundation for Statistical Computing, Vienna, Austria. URL <http://www.R-project.org/>).

Results

The characteristics of the study population are summarized in Table 1. There are 956,508 observations in the sample corresponding to 955,331 women and 1,727 diagnoses of ADH. There were 1,058 ADH diagnoses made by core needle biopsy, 635 diagnosed by excision, and 34 of unknown type. ADH was associated with white race, first-degree family history of breast cancer, and high breast density on mammography.

Figure 1a shows rates of ADH diagnoses per 10,000 mammograms by study year. The rates range from a low of 2 in 1995 to a high of 6 per 10,000 mammograms in 2011. Figure 1b shows the proportion of ADH diagnosed via core needle vs. excisional biopsy. Detection by core needle biopsy increased over time while diagnosis via excisional biopsy decreased. In

the beginning of the study (1995) all ADH was diagnosed by surgery, and this percentage decreased to 23% by 2012.

After adjusting for age, race/ethnicity, family history, menopausal/HT status, mammogram modality and breast density, within ten years following a diagnosis of ADH, the rates of invasive breast cancer were 2.6 (95% CI 2.0, 3.4) times higher than those in women with no ADH diagnosis at baseline (Table 2). ADH diagnosed via excisional biopsy was associated with an adjusted HR of 3.0 (95% CI 2.0, 4.5), and via core needle biopsy, with an HR of 2.2 (95% CI 1.5, 3.4); however, a test for differences in HRs by biopsy type was non-significant ($p=0.28$).

The HR of invasive breast cancer after a diagnosis of ADH varied with time since diagnosis, with slightly higher rates seen in the first two years compared to subsequent years.

Ten years after an ADH diagnosis, 5.7% (95% CI 4.3, 10.1) of women were estimated to develop invasive cancer (Table 3). Women with ADH diagnosed on excisional biopsy had a slightly higher estimated risk (6.7 %; 95% CI 2.2, 8.9) compared to those with ADH diagnosed via core needle biopsy (5.0%; 95% CI 3.0, 12.8). In contrast, 2.2% (95% CI 1.7, 3.9) of those without ADH were estimated to develop invasive cancer.

Discussion

The risk of breast cancer associated with ADH was reported by studies that followed large cohorts of women undergoing breast biopsies. Most of these studies were done prior to the widespread use of screening mammography and image guided needle biopsies and do not report the indication for biopsy. Although not detailed, one can assume, based on the study years, that a palpable finding lead to many of these biopsies⁸⁻¹⁰, questioning the ability to generalize these results to women undergoing image-guided biopsies today.

The most cited study is the work of Dupont and Page⁸, which reported the outcome of women undergoing excisional biopsies between the years 1950–1968. In this study, women with ADH were at 4 times higher risk of subsequent invasive breast cancer when compared to the general population.

When communicating risk of disease, clinicians and patients were better able to interpret cumulative risks than relative risks¹¹. The Mayo cohort of women with benign breast biopsies includes women diagnosed mainly by excision (86%) between the years 1967–2001². In this cohort the projected cumulative risk of cancer for women with atypia (ADH or ALH) was 29% over 25 years. Lower risks were published from the Breast Cancer Detection Demonstration Project (BDDP)¹²; although this was an early study (women were enrolled between 1973 and 1978), all participants underwent screening mammography. Of 1,305 women with atypia, approximately 5% developed invasive breast cancer over 8 years. Tice reported 10-year risk of invasive breast cancer of 7% in women with atypia (either ductal or lobular) in a recent report from the BCSC¹³.

We report here the largest study on outcome of women diagnosed with ADH using contemporary imaging and biopsy technology. Using data from the BCSC, we found that

rates of ADH in present day practice range between 4 and 6 per 10,000 mammograms. Over the study years the proportion of ADH diagnosed via excisional biopsy has decreased substantially but remains above 20% in 2012. The 10-year cumulative risk of invasive breast cancer after a diagnosis of ADH was 5.7% with a higher rate seen in women diagnosed with excisional biopsies vs. those diagnosed by core needle biopsies (6.7% vs. 5.0%). These results are consistent with our hypothesis that over time with the advancements in imaging and biopsy technology, smaller foci of ADH are being diagnosed, which may carry a lower risk for invasive breast cancer.

There may be other explanations for the lower rates of breast cancer found in our study. We did not include DCIS as an outcome, which was counted in other reports^{2, 14, 15}. In these studies the proportion of DCIS from subsequent cancers ranged between 19 and 50%. We limited our study to women with ADH, whereas other studies examined the risk of breast cancer in women with either ADH or ALH^{2, 13}. The risk of breast cancer appears to be higher after a diagnosis of ALH when compared to ADH^{9,16}, which can explain the lower 10-year risk of invasive cancer we found even when compared to contemporary reports¹³. Use of chemoprevention such as Tamoxifen after a diagnosis of ADH can reduce the risk of subsequent breast cancer; however, low acceptance of chemoprevention has been documented in the US population¹⁷. Tamoxifen use was documented in 7% (96 of 1308) of women with an ADH diagnosis for whom subsequent self-report data was available in our study, regardless of type of biopsy. Therefore the lower risk found in our study cannot be attributed to Tamoxifen use.

There are several modifiers to the risk associated with ADH. Risk of breast cancer is inversely associated with age at diagnosis of ADH¹⁸. Family history of breast cancer has been shown to increase the risk associated with ADH by some (1) but not by others^{18,19}. Reproductive risk factors²⁰ and alcohol²¹ were reported to modify this risk as well. The risk associated with ADH is increased in women with dense breasts compared to women with fatty breasts²¹. While in the Mayo clinic cohort the number of foci of atypia correlated with the risk of cancer, a recent report from the Nurses' Health Study did not demonstrate such an association in the subgroup of women with ADH⁹. Calculators using several risk factors can better stratify women and help in consulting them on their risk and risk reduction options^{13, 22}.

Our study has several limitations, including no central pathological review. Pathological review could reclassify ADH either to DCIS or to hyperplasia with no atypia. However as most women do not get a second opinion after a diagnosis of ADH, this study can be generalized to women receiving such a diagnosis in community practice.

In summary, women diagnosed with ADH by core needle and excisional biopsy had a slightly lower risk of invasive breast cancer than previously reported. As the risk associated with ADH is modified in the presence of other risk factors, one should not recommend increased surveillance and risk reducing strategies without accounting for other risk factors. An assessment of an individual's risk based on multiple risk factors should be preferred^{13,22} before deciding on prevention strategies.

Acknowledgments

Jane Lange had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

This work was supported by the National Cancer Institute-funded Breast Cancer Surveillance Consortium (HHSN261201100031C). Data collection for this work was additionally supported, in part, by funding from the National Cancer Institute (P01CA154292, U54CA163303). The collection of cancer and vital status data used in this study was supported in part by several state public health departments and cancer registries throughout the US. For a full description of these sources, please see: <http://breastscreening.cancer.gov/work/acknowledgement.html>. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Cancer Institute or the National Institutes of Health. We thank the participating women, mammography facilities, and radiologists for the data they have provided for this study. A list of the BCSC investigators is provided at: <http://breastscreening.cancer.gov>.

References

1. Page DL, Dupont WD, Rogers LW, Rados MS. Atypical hyperplastic lesions of the female breast. A long-term follow-up study. *Cancer*. 1985; 55:2698–708. [PubMed: 2986821]
2. Hartmann LC, Degnim AC, Santen RJ, Dupont WD, Ghosh K. Atypical hyperplasia of the breast—risk assessment and management options. *N Engl J Med*. 2015 Jan 1;372:78–89. [PubMed: 25551530]
3. Fisher B, Constantino JP, Wickerham DL, et al. Tamoxifen for prevention of breast cancer: report of the National Surgical Adjuvant Breast and Bowel project-1 study. *J Natl Cancer Inst*. 1998; 90:1371–88. [PubMed: 9747868]
4. Coopey SB, Mazzola E, Buckley JM, et al. The role of chemoprevention in modifying the risk of breast cancer in women with atypical breast lesions. *Breast Cancer Res Treat*. 2012; 136:627–33. [PubMed: 23117858]
5. Goss PE, for the NCIC CTG MAP.3 Study Investigators. Exemestan for breast-cancer prevention in postmenopausal women. *N Engl J Med*. 2011; 364:2381–2391. [PubMed: 21639806]
6. Zheng Y, Heagerty PJ. Partly conditional survival models for longitudinal data. *Biometrics*. 2005; 61:379–91. [PubMed: 16011684]
7. Lee, E., Wei, L., Amato, D. Cox-type regression analysis for large numbers of small groups of correlated failure time observations. Netherlands: Kluwer Academic Publishers; 1992.
8. Dupont WD, Page DL. Risk factors for breast cancer in women with proliferative disease. *NEJM*. 1985; 312:146–51. [PubMed: 3965932]
9. Collins LC, Aroner SA, Connolly JL, Colditz GA, Schnitt SJ, Tamimi RM. Breast cancer risk by extent and type of atypical hyperplasia: an update from the Nurses' Health Studies. *Cancer*. 2016; 122(4):515–20. [PubMed: 26565738]
10. Hartmann LC, Sellers TA, Frost MH, et al. Benign breast disease and the risk of breast cancer. *N Engl J Med*. 2005; 353:229–37. [PubMed: 16034008]
11. Elmore JG, Gigerenzer G. benign breast disease - the risks of communicating risk. *N Engl J Med*. 2005; 353:297–9. [PubMed: 16034016]
12. Carter CL, Corle DK, Micozzi MS, Schatzkin A, Taylor PR. A prospective study of the development of breast cancer in 16,692 women with benign breast disease. *Am J Epidemiol*. 1988; 128(3):467–77. [PubMed: 3414655]
13. Tice JA, Miglioretti DL, Li CS, Vachon CM, Gard CC, Kerlikowske K. Breast Density and Benign Breast Disease: Risk Assessment to Identify Women at High Risk of Breast Cancer. *J Clin Oncol*. 2015; 33(28):3137–43. [PubMed: 26282663]
14. Worsham MJ, Raju U, Lu M, et al. Risk factors for breast cancer from benign breast disease in a diverse population. *Breast Cancer Res Treat*. 2009; 118(1):1–7. [PubMed: 18836828]
15. Zhou WB, Xue DQ, Liu XA, Ding Q, Wang S. The influence of family history and histological stratification on breast cancer risk in women with benign breast disease: a Meta-analysis. *J Cancer Res Clin Oncol*. 2011; 137(7):1053–60. [PubMed: 21499874]

16. Collins LC, Baer HJ, Tamimi RM, Connolly JL, Colditz GA, Schnitt SJ. The influence of family history on breast cancer risk in women with biopsy-confirmed benign breast disease: results from the Nurses' Health Study. *Cancer*. 2006; 107(6):1240–7. [PubMed: 16902983]
17. Waters EA, McNeel TS, Stevens WM, Freedman AN. Use of tamoxifen and raloxifene for breast cancer chemoprevention in 2010. *Breast Cancer Res Treat*. 2012; 134(2):875–80. [PubMed: 22622807]
18. Hartmann LC, Radisky DC, Frost MH, et al. Understanding the premalignant potential of atypical hyperplasia through its natural history: a longitudinal cohort study. *Cancer Prev Res (Phila)*. 2014; 7(2):211–7. [PubMed: 24480577]
19. Whiffen A, El-Tamer M, Taback B, et al. Predictors of breast cancer development in women with atypical ductal hyperplasia and atypical lobular hyperplasia. *Ann Surg Oncol*. 2011; 18:463–7. [PubMed: 20878246]
20. Kabat GC, Jones JG, Olson N, et al. Risk factors for breast cancer in women biopsied for benign breast disease: a nested case-control study. *Cancer Epidemiol*. 2010; 34(1):34–9. [PubMed: 20079704]
21. Tice JA, O'Meara ES, Weaver DL, Vachon C, Ballard-Barbash R, Kerlikowske K. Benign breast disease, mammographic breast density, and the risk of breast cancer. *J Natl Cancer Inst*. 2013; 105(14):1043–9. [PubMed: 23744877]
22. Pankratz VS, Hartmann LC, Degnim AC, et al. Assessment of the accuracy of the Gail model in women with atypical hyperplasia. *J Clin Oncol*. 2008; 26(33):5374–9. [PubMed: 18854574]

Figure 1.

Rates of ADH per 10,000 screening mammograms (a); and mode of detection by study year (b).

Table 1

Description of study population

	All N =954,789 (%)	ADH-all N=1,727 (%)	ADH-Core needle biopsy ¹ N=1,058 (%)	ADH-Surgical biopsy ² N=635 (%)
BI-RADS density				
a. Breasts are almost entirely fatty	79195 (8)	60 (4)	44 (4)	16 (3)
b. Scattered areas of fibroglandular density	413261 (43)	635 (37)	390 (37)	236 (37)
c. Breasts are heterogeneously dense	373597 (39)	838 (49)	517 (49)	307 (48)
d. Breasts are extremely dense	88728 (9)	194 (11)	107 (10)	76 (12)
Menopause/Hormone treatment (HT) status				
Premenopause	399800 (47)	649 (43)	413 (45)	219 (39)
Postmenopause, no HT ³	217356(26)	301(20)	183(20)	113(20)
Postmenopause, HT	158440(19)	261 (17)	135(15)	121(21)
Postmenopause, HT unknown	36000(4)	244(16)	162(18)	80(14)
Surgical menopause, no HT	32754(4)	46(3)	21(2)	25(4)
Surgical menopause, HT unknown	2679(0.3)	20(1)	11(1)	9(2)
Year of study entry				
1994–1999	247288 (26)	258 (15)	103 (10)	153 (24)
2000–2004	409226 (43)	595 (35)	323 (31)	260 (41)
2005–2012	298267 (31)	874 (51)	632 (60)	222 (35)
Race/ethnicity				
White, non-Hispanic	642560 (76)	1244 (82)	763 (83)	457 (80)
Black, non-Hispanic	70575 (8)	63 (4)	40 (4)	23 (4)
Asian	47258 (6)	93 (6)	62 (7)	27 (5)
Other	19591 (2)	31 (2)	14 (2)	17 (3)
Hispanic	71043 (8)	93 (6)	41 (5)	51 (9)
First degree family history of breast cancer				
No	848360 (89)	1431 (83)	874 (83)	529 (83)
Yes	106421 (11)	296 (17)	184 (17)	106 (17)
Mammography modality				
Film	776188 (83)	1041(67)	558(58)	468 (81)
Digital	160602(17)	525(34)	401(42)	111(19)
Age, median, [IQR³]	49.7 [43.0,58.2]	52.6 [46.9,60.4]	52.7 [47.3,60.0]	52.4 [46.5,60.8]

¹ for ADH diagnosis, refers to most proximal screening mammogram

² N=34 ADH diagnosis with missing biopsy type were excluded

³ IQR- interquartile range

Table 2

Hazard ratios from Cox models adjusting for age and age-squared at baseline, ethnicity, BI-RADS density, family history, and mammography type.

	HR (95% CI)	p-value
No ADH at baseline mammogram	ref	
ADH	2.6 (2.0, 3.4)	<0.001
No ADH at baseline mammogram	Ref	
Core needle biopsy	2.2 (1.5, 3.4)	<0.001
Surgical biopsy	3.0 (2.0, 4.5)	<0.001
No ADH at baseline mammogram	Ref	
ADH 0–2 years	3.2 (1.9, 5.3)	<0.001
ADH 2.01–4.99 years	2.4 (1.4, 4.0)	<0.001
ADH 5–10 years	2.7 (1.7, 4.3)	<0.001

Table 3

Estimated 10 year probability of invasive breast cancer after ADH diagnosis

	Ten year probability of invasive breast cancer % (95% CI)
Mammogram at baseline	2.2% (1.7, 3.9)
ADH at baseline	5.7% (4.3, 10.1)
Core needle biopsy-ADH at baseline	5.0% (2.2, 8.9)
Surgical biopsy-ADH at baseline	6.7% (3.0, 12.8)