The Ribbon

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Invasive Breast Cancer: Different Histologic Types

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When assessing the needed therapy for and prognosis of an individual with breast cancer, there are a number of critical issues. The most important relate to the stage of the cancer (size, lymph node involvement and presence or absence of metastatic disease). Also important are characteristics related to the patient's health – including other medical problems, menopausal status, and the patient's own preference for types of therapy. In addition, the efficacy of the various therapies, their toxicity and the amount of evidence for improved outcomes – survival, lack of recurrence, etc. – are important.

There are other important factors which don't influence the stage of cancer which also have an impact. These include grade (how aggressive the cancer appears under the microscope), hormone receptor status (whether the tumor is likely to respond to hormonal therapy), other receptors, and the histology of the breast cancer. This article will focus on the histology.

Since the breast is a glandular structure, almost all malignancies of the breast are glandular cancers, also called adenocarcinomas. The current belief is that all or almost all invasive breast cancers start in a pre-invasive stage, also called *in situ*. These lesions don't have the ability to spread to lymph nodes or metastasize unless/until they become invasive. There are two very distinct types of *in situ* carcinomas of the breast – lobular and ductal.

Lobular-carcinoma-*in situ* (LCIS) is usually an incidental microscopic finding, as it lacks clinical or mammographic signs. It is not truly a malignancy but a marker for increased risk of breast cancer development in both breasts. There is approximately 10 -15% risk of ipsilateral (the same side of the body) and another 10% risk contralateral (opposite side of the body) invasive breast cancer occurring. Despite the fact that the initial lesion is lobular, most of the subsequent cancers are invasive ductal carcinomas (only about 30% are lobular cancers). The fact that most women with LCIS don't go on to develop breast cancer, that the risk is bilateral and that the usual cancers which occur are ductal prove that this is a marker – a risk for breast cancer and not an actual pre-invasive cancer. With LCIS, there is about a 1% chance per year of developing invasive cancer and it should be thought of as a risk factor, just like family history, age, or nulliparity. Occasionally a decision is made for bilateral mastectomy, especially if there is the additive risk factor of a strong family history. There is no role for unilateral mastectomy or wide local excision or radiation therapy.

The other non-invasive breast cancer, ductal-carcinoma-*in situ* (DCIS) is very different. It is an actual pre-

invasive malignancy with risk of invasive cancer at that site. Accordingly, therapy and risks are very different from LCIS. DCIS is almost always discovered as an abnormality on a mammogram, usually as clustered microcalcifications. In the days before the routine use of mammograms, it was a rare diagnosis. Now it accounts for 20% or more of all breast cancers. There has probably been more debate about the treatment of DCIS than any other type of breast cancer. Even after conservative therapy (less than mastectomy) became a standard option for invasive breast cancer, mastectomy remained standard for DCIS. Today, even though no randomized trial of mastectomy compared to lesser therapies exist, the vast majority of women with DCIS don't need and don't have a mastectomy.

DCIS can be cured with mastectomy 98 - 99% of the time. The lymph nodes are only involved about 1% of the time. With conservative therapy there is certainly a higher percentage of recurrences of both DCIS and invasive cancer, however in appropriately selected patients, survival can be 97% at ten years. A number of studies have looked at whether adding other therapies to lumpectomy improves the outcome. The National Surgical Adjuvant Breast Project (NSABP) has performed a number of studies. NSABP B17 showed that adding radiation to conservative surgery lessened the recurrence rates. More recently NSABP B24 has shown that adding Tamoxifen reduces recurrence in both breasts.

There are still times when mastectomy is needed: (1) inability to remove all the DCIS with conservative surgery; (2) very large areas of DCIS or (3) extensive DCIS that is multifocal and involving multiple quadrants of the breast. In addition, DCIS is a heterogeneous group of lesions varying by patterns of growth. Most commonly these are divided into comedo-type DCIS which has prominent necrosis and large cells, and non-comedo-type. The non-comedo-type DCIS includes cribriform, micropapillary, papillary and other less common types. Comedo type appears more aggressive histologically and also has markers of more aggressive disease. It is less commonly receptor-positive; more commonly it has abnormal amounts of DNA (aneuploid) and more often expresses "bad" oncogenes.

Treatment of DCIS needs to be individualized based on the tumor's characteristics, the family history and the patient's preferences.

Before turning to invasive breast cancer I'd like to discuss benign breast conditions – especially hyperplasia or overgrowth of cells. With "mild hyperplasia of the usual type," i.e. without atypical features, there is <u>not</u> an increased risk of breast cancer development. In "moderate or florid hyperplasia of the usual type" there is a slight increased risk which rises in conjunction with a positive family history of breast cancer. Atypical hyperplasias have some of the characteristics of LCIS or DCIS but lack the complete pathologic criteria, either because of small size or involvement of just one duct or lobule. These lesions have a relatively high risk of developing into invasive breast cancer. The table below presents the relative

RELATIVE RISK CATEGORIES FOR INVASIVE BREAST CARCINOMA BASED ON HISTOPATHOLOGY, AGE AT PRESENTATION, AND PRESENCE OR ABSENCE OF A FAMILY HISTORY OF BREAST CANCER, COMPILED FROM STUDIES SINCE 1985

No Increased Risk
Mild Intraductal hyperplasia (RR 1)
Slightly Increased Risk
Moderate or florid intraductal hyperplasia (RR 1.1 – 2.5)
Mild To Moderately Increased Risk
Atypical hyperplasia, ductal or lobular (with no family history) (RR 2.6-4.3)
Atypical hyperplasia, ductal or lobular (in a postmenopausal woman) (RR 3.3 – 6.5)
High Risk
Atypical hyperplasia, ductal or lobular (with a family history) (RR 6.6-22)
Atypical hyperplasia, ductal or lobular (in a premenopausal woman) (RR 4.5 – 12)
Ductal carcinoma in situ (RR 8 – 10)
Lobular Carcinoma in situ (RR 8 – 10)

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risk of breast cancer for all of the categories discussed to this point.

NSABP recently completed its first prevention trial looking at Tamoxifen versus placebo for women at high risk. Those with atypical hyperplasia were among those at highest risk and who had the biggest benefit from Tamoxifen. Benefit has also been shown in LCIS and DCIS.

Among invasive breast cancers (those with the potential to metastasize) the majority are invasive ductal. This includes those tumors that have some characteristics of the more specific types discussed below, but which don't fully meet their criteria. About 30% of ductal carcinomas have some other features. Invasive ductal carcinomas form solid tumors and when large enough are usually palpable. Size and grade are the most important local determinants of prognosis. The more a cancer resembles normal breast tissue, the lower the grade and the better the prognosis. This is certainly true for lymph node (LN) negative tumors and less clear for LN positive tumors. Invasive ductal cancers are found in all age groups and are the typical cancers against which other histologic types are compared.

Other factors having prognostic significance for invasive ductal carcinoma include: blood vessel invasion, invasion of the nerves (perineural invasion), and presence of HER2/neu over expression.

There are several types of invasive cancer which are much less common, have distinctive histologic features and tend to have a better outcome than the invasive ductal carcinomas. Tubular, also called well-differentiated carcinoma, is as the name implies a cancer that closely resembles normal mammary ducts. They almost always have an *in situ* component. While they constitute only 1 - 2% of breast carcinomas, among small, mammographically detected tumors of 1 cm or less, they constitute 5 - 10%. Because they are smaller and slower growing, they are less likely to involve lymph nodes or to metastasize. Their prognosis is much better than the average breast cancer, even taking their small size into consideration.

Mucinous carcinomas also make up 1 - 2% of breast carcinomas, but more commonly occur in the elderly (over age 75), where more than 5% of carcinomas are mucinous. As the name implies, they secrete mucin and little glandular tissue is seen. They tend to have a better outcome than the typical invasive ductal carcinomas early on but have a high rate of metastases at 10 years or greater.

Medullary carcinoma comprises about 7% of breast carcinomas; it is more common in younger individuals, less than 50 years old. It tends to present with large tumors. Although they tend to grow rapidly and present as large lesions, their prognosis is also better than average. They are less likely to go to axillary lymph nodes and when the nodes aren't involved, the prognosis is especially favorable. This diagnosis has a very rigid set of pathological criteria and if not all the features are present it is called atypical medullary carcinoma. The outcome for this variant is not as good.

Papillary carcinomas make up another 1 - 2%. They are more commonly located near the nipple and frequently present with nipple discharge. They have a frond-like or papillary growth pattern. There are several other even rarer carcinomas which start in the ducts. Details on these can be found in the references.

The other common breast carcinoma is lobular. About 10 - 15% of breast carcinomas are infiltrating lobular. They present much more frequently as a mass and less commonly on mammograms than other types. They don't cause calcification on mammograms. Both grossly and microscopically these tumors don't have well-defined margins. They are more commonly bilateral than other types – either synchronously or metachronously. There are often components of other histological types mixed in. Microscopically these cells are often arranged in a linear fashion.

Infiltrating lobular carcinoma has a different pattern of spread than other histologic types – more commonly going to the lining of the central nervous system (meninges) or to surfaces inside the abdomen. It can also invade many abdominal organs and mimic stomach, uterine or ovarian carcinomas. Overall the

Incidence of Histologic Types of Invasive Breast Cancer

Pure Tumor Types:

Infiltrating Ductal	53%
Medullary	6%
Invasive Lobular	5%
Mucinous	2%
Tubular	1%
Papillary	1%
Infiltrating Ductal & other(s)	28%
Pagets Disease	2%
Other Combinations	3%

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prognosis is similar to that of infiltrating ductal carcinoma.

An unusual but poor prognosis type of breast carcinoma is inflammatory carcinoma. The prognosis is poor enough that they are automatically classified as Stage III B (just one step below metastatic disease). Inflammatory carcinoma involves the skin and the lymphatics of the skin. It is usually large, often taking up more than one quadrant of the breast and is very rapidly growing. The skin often has a shriveled appearance called peau d'orange (skin of an orange). With multi-modality treatment in recent years, the prognosis has improved somewhat.

In summary, there are many different types of adenocarcinoma of the breast. Some of these types have clinical significance. The earlier proliferative breast lesions, both hyperplasia and *in situ* disease all present with increased risk of developing invasive breast cancer. The degree of risk and some of the potential therapies were discussed previously. For more detail on all of the above topics, please refer to the following references.

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Research Commentary

Hormone Replacement Therapy and Other Possible Risk Factors for Breast Cancer

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Previously accepted risk factors for breast cancer, such as gene defects (BrCa1, BrCa2, Her2Neu, etc.), early age at menarche, and late age at first pregnancy, account for only perhaps 15% of the annual incidence of breast cancer. There has therefore been a near feverish search for other breast cancer risk factors. Much of this search has focused on drugs, diet, and the environment as possible explanations for breast cancer risk.

Hormone Replacement Therapy

For many years the role of hormone replacement therapy (HRT), primarily Premarin in this country (although estradiol benzoate has been used in Scandinavia), has been the object of numerous studies with inconclusive results, both positive and negative. Cutting through this murkiness, a recent study by Gapstur and colleagues provided some surprising results. In this study, carried out on a large population base in Iowa, they analyzed breast cancer incidence *by type* in patients who used HRT and a control population who did not use HRT. In this study they found no significant increase in ductal carcinoma *in situ* (DCIS), or ductal or lobular carcinoma; they did find an increase in a rare (<5% of all tumors) type of invasive carcinoma, which has a relatively favorable histology and a low mortality. The reason for this slight shift in cancer incidence remains elusive. No such shift was found by Persson and colleagues in Sweden, who reported a relative risk of 1.4 in women using estrogen progestin combinations for 1-6 years but no increase in breast cancer risk in women using estrogen alone. However, in women using only estrogen the risk of endometrial cancer increased 4-fold but was non-significant in women using the combined drug. They did not analyze in terms of tumor type, preventing any direct comparison with the study of Gapstur and

colleagues. A more complicated risk issue is the safety of HRT in women with a history of breast cancer. In a recent review Seifert et al concluded that, despite the possible benefits in postmenopausal women, the potential risks remain uncertain and do not justify their use in such women, pending a suitable clinical trial.

The state of flux of research in this field is illustrated by the fact that two more recent papers have reported quite different findings. Stallard and colleagues in a recent paper in the British Medical Journal reported that HRT treatment neither generated tumors with a more favorable prognosis nor did it result in poorer prognosis tumors, in marked contrast to the results reported by Gapstur et al. The other paper by Schairer et al in JAMA reported still a different result, with a slight increase in risk with HRT treatment particularly in lean women with a Body Mass Index of less than 24.4. All of the studies had substantial study populations and appear to have been competently carried out. While the overall results suggest that the risk from HRT treatment is minimal, it is clear that additional studies are needed to sort out these widely varying results and arrive at a generally accepted assessment of the risk from HRT treatment.

Oral Contraceptives

A similar controversy still exists on oral contraceptives (OCs) as a risk for breast cancer. There have been innumerable studies on this topic, but the results are still inconclusive for the most part. Some studies have found no effect, while others have reported a modest increase in risk. There have been suggestive data for a slight increase in risk in teenagers taking OCs between the ages of 16 and 20. Compensating for any slight increases in breast cancer risk is the fact, reported by Henderson, Ross, and Pike that use of OCs for 4-5 years results in a permanent 50% decrease in ovarian and endometrial cancer. This is a real benefit which needs to be kept in mind in evaluating the medical benefits of OC use. The OC studies have not categorized cases in terms of cancer type as has been done for HRT. An exploration of cancer type in patients taking OCs would be of great interest and is likely to be undertaken in the near future.

Other Factors Impacting Body Estrogen

The role of obesity and diet is equally murky, partly because food intake is frequently confused with body composition, particularly in the popular press. There is clear evidence that the risk of obesity is age-related. Among premenopausal women the risk is higher for women of average weight than it is for very thin or — surprisingly — very obese women. Among post-menopausal women, however, obesity is clearly a risk factor. These paradoxes can be explained, but not in the limited space of this article.

Diet is important, but not as it is often understood. Although much attention has been paid to dietary fat consumption this concern is misplaced since total calories consumed, not total fat consumed, is the key to amount of body fat. There are ample data demonstrating that excess calories as carbohydrate result in increased fat deposition. It is certainly true that, because of the concentrated calorie content of fat, it is an easy cause of obesity. It is also true that, in the absence of parallel carbohydrate consumption, dietary fat does not end up as stored fat. This is because glycerol-phosphate required for resynthesis of body fat comes only from carbohydrate, not from fat. Dietary restriction, particularly of carbohydrate, and increased exercise, are sensible approaches to decreasing diet-related breast cancer risk. (This is not meant to dismiss the possible benefits of decreased fat intake in terms of heart disease and other metabolic problems.)

Extensive studies have shown that a variety of micronutrients can act to decrease breast cancer risk. These include a diverse group of compounds found primarily in vegetables. Examples include such compounds as indole-3-carbinol, its dimer diindolylmethane, sulfuraphane, all found in cruciferous vegetables; epigallocatechin gallate (EGCG), found in green tea; curcumin, found in tumeric; vitamin E and a host of other antioxidants. These compounds act in various ways. Thus the indoles and EGCG act in part by altering estrogen metabolism to increase the level of the protective estrogen metabolite, 2-hydroxyestrone. Other protective mechanisms include increasing phase II conjugating activity, increasing apoptosis (cell death), and inhibiting cyclin D.

Decreased risk for breast cancer in women who exercise regularly was first reported by Frisch, and more recently by Bernstein and colleagues, as well as in the Nurses Health Study. One of the actions of exercise is to increase the conversion of estradiol to 2-hydroxyestrone, the "good" estrogen, which is only weakly estrogenic. The action is indirect, with the intermediate step being a decrease in fat depots. This has been demonstrated by Frisch and Snow. This reduction in fat depots decreases the release of a protein that inhibits 2-hydroxylation.

The most confusing and speculative issue has been the risk-promoting effect of the environment. This includes the possible effects of a varied group of compounds and environmental parameters, including electromagnetic

fields (EMF), aryl hydrocarbons (benzpyrene, dimethylbenzanthracene and related compounds), pesticides, PCBs, etc. Recent studies have rendered EMF an unlikely risk factor for breast cancer. While aryl hydrocarbons are clearly tumor-promoting in rodent models, the evidence for a comparable role in human disease has not been strong and current case control studies have been negative. There has been considerable interest in the possible carcinogenic role of chlorinated pesticides. Cell culture studies showed that chlorinated pesticides promoted 16α-hydroxylation of estradiol, while no such effect was observed in the presence of phosphorus-based pesticides. Several earlier human studies were highly suggestive, while more recent studies have for the most part been negative, except for a Danish study using old blood samples, which showed a correlation between dieldrin levels and cancer risk. Because the widespread use of chlorinated pesticides has been markedly curtailed in this country and in much of Europe, circulating levels of these compounds have declined — creating the illusion that these compounds are harmless and decreasing the chance of seeing significant differences between cases and controls. The same is true for PCBs, whose circulating levels have also dropped significantly as the use of these compounds has been curtailed.

Despite the enormous amount of effort that has gone into looking for risk factors we still have no certainty about the risk factors causing the majority of breast cancers. At best we have some working hypotheses and a number of well-publicized speculations. We may have to make decisions and take action, on both the personal and governmental levels, in the face of incomplete knowledge; but we should not confuse speculation with fact.

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Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State (BCERF)

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__*FS # 3*—Understanding Breast Cancer Rates

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Critical Evaluations are available on the BCERF web page as portable document files (pdf), and can be accessed on the BCERF web site (see address below).

If you would like to order a hard copy please indicate below and send your check payable to Cornell University for **$3.00 each**, to cover the cost of reproduction and mailing.

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What's New on the Web
www.cfe.cornell.edu/bcerf/

Spring is coming and with it BCERF has several new offerings on our website. Among our new features is an interactive glossary, the G.dot. The glossary is designed to help readers better understand vocabulary and ideas presented in the fact sheets.

Also added recently is a section introducing the Rural Initiative interactive computer display. The pages give the readers a glimpse at BCERF's newest outreach efforts. The pages feature photos of the display and a press release. It also offers contact information for educators who may be interested in using the interactive computer display at their event.

Other efforts include the February eUpdate. This eUpdate alerted our electronic readers to the selection of the BCERF Critical Evaluation on Atrazine as a background material for the EPA's review of the pesticide. The EPA was not the only federal agency to highlight BCERF's accomplishments. In January the USDA featured the BCERF webpage on their own homepage under Special Topics, providing a brief description of our program and a link to the site.

Lots of other projects are in the works. We hope you will visit our web site, read, learn and give us your opinions on how we can continue to improve.

Marie Stewart, BCERF Webmaster

NOTICE OF ADDRESS CHANGE:

BCERF staff is now consolidated on the first floor in Rice Hall. With this consolidation comes a different room address for the main office for BCERF. We are now located in **112 Rice Hall**. The phone number, fax, and e-mail address remain the same.

Below is a complete listing of BCERF campus-based personnel:

- June Fessenden MacDonald, PhD, Director, BCERF and Associate Professor, Biochemistry and Biology & Society
- Carol Devine, PhD, RD, Education Project Leader, BCERF and Associate Professor, Nutritional Sciences
- Renu Gandhi, PhD, Research Associate
- Ann T. Lemley, PhD, Associate Director, BCERF and Chair, TXA, Human Ecology
- Mary Maley, BS, Health Educator
- Carmi Orenstein, MPH, Assistant Director and Extension Project Leader
- Jodi Paar, PhD, MEd, Environmental Health Educator
- Carin Rundle, Administrative and Outreach Coordinator
- Neil Rotach, Administrative Assistant
- Suzanne Snedeker, PhD, Research Project Leader, BCERF and Assistant Professor, Environmental Toxicology and Health
- Sandra Steingraber, PhD, Visiting Assistant Professor
- Marie Stewart, MS, Webmaster
- Barbour Warren, PhD, Research Associate

Status of the Pesticide Sales and Use Reporting Database

William G. Smith, Senior Extension Associate, PMEP, Cornell University

The Pesticide Management Education Program (PMEP) at Cornell University continues to develop a pesticide sales and use reporting database in conjunction with the 1996 Pesticide Reporting Law and the Pesticide Reporting Section within the New York State Department of Environmental Conservation (NYSDEC). The PMEP database team is in the second year of the development phase of the database and has currently finished processing the 1998 sales and use data. Summaries of the 1997/1998 use and sales reports can be found at the PMEP website, http://pmep.cce.cornell.edu/regulation/psur/, and at the NYSDEC website, http://www.dec.state.ny.us/website/dshm/pesticid/prl.htm. Working with PMEP and the NYSDEC are the data entry vendor Lason and the electronic reporting vendor, Compaq.

There are six (6) phases that compose the PSUR Production Cycle:

1) Media Administration
2) File Verification
3) Audit Check
4) Data Validation
5) Report Generation
6) Web-based Report Preparation

All six phases are unique, highly complex, and require continual modification as the reporting entities and information become more stabilized. For instance, we processed approximately 11 million records for the 1998 reporting year which was twice as many as what was processed for 1997.

The Memorandum of Agreement (MOA) that Cornell has with the NYSDEC contains objectives other than the development of a pesticide sales and use reporting database. Cornell is assisting the NYSDEC in redesigning other databases (certification, commercial business, commercial permits, and product registration), as the data from these 'feed' directly into the sales and use reporting database.

To date, we have completed a prototype of an auxiliary database that will provide pesticide product information for people accessing the pesticide reporting data on the web. Among other things, this database will provide active ingredient and registrant information for the products listed in the report. We will also maintain a second copy of the database containing only pesticides that are currently registered in New York State. Both copies of the new database are maintained primarily with data received from the DEC Product Registration Section. Also, a recertification course calendar database has been developed that tracks recertification courses (dates, places categories, credits and contacts) for certified applicators. This database can be assessed from the PMEP website listed previously. Finally, the PMEP database team is currently working with the Product Registration Section to assess the technology for imaging labels and other documents that can eventually be retrieved/quarried by the public from a menu-driven (database) platform.

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