comedo type, pose no special problem. The problems arise with low-grade DCIS, as described earlier, and the borderline cases will continue to pose a problem, even for experienced pathologists with an interest in this area.

Finally, the statement that the pathology assessment is critical not only to the diagnosis of DCIS but also to prognosis and choice of treatment definitely applies to high-grade, comedo-type DCIS. There is good evidence that such lesions occur frequently and will progress to infiltrating carcinoma if treated inadequately. Although we may not know as much about the natural history of low-grade DCIS, there is evidence that its clinical behaviour is less aggressive, as there is less recurrrence after excisional biopsy.[4] Even less is known about the natural history of limited foci of low-grade DCIS and ADH, although we do know that women who have these lesions are at increased risk of subsequent carcinoma. Pathologists must still strive to classify these lesions to the best of our abilities, so that clinical trials can determine their biological potential and the most appropriate management.

Wayne R. Ramsay, MD
Pathologist
St. Catharines General Hospital
St. Catharines, Ont.

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We were pleased to see the publication of this supplement. However, we were disappointed that although the guideline "The palpable breast lump: information and recommendations to assist decision-making when a breast lump is detected" (*CMAJ* 1998;158[3 Suppl]:S3-8) mentioned strong family history among the factors that increase the likelihood of breast cancer (level III evidence), nowhere else in the document was there any discussion of the recently discovered breast cancer susceptibility genes. It is now known that mutations in 2 recently identified genes, *BRCA1* and *BRCA2*, confer a risk of breast cancer. Mutations in these genes appear to account for 5% to 10% of all cases of breast cancer. Identification of such mutations provides important information about the risk of additional neoplasms in the affected individual and other family members. This risk includes the association of breast cancer with ovarian cancer in predisposed families and the risk of breast cancer among male members of these families. Furthermore, in some families with familial breast and ovarian cancer, there could be increased predisposition to colerectal cancer.[1]

The guidelines document also indicates that the risk of breast cancer increases with age. In 1997 in Canada the cumulative risk of breast cancer was approximately 11% by age 70 years.[2] This risk is much higher in families known to carry one of the mutant alleles. The cumulative risk for women carrying *BRCA1* mutations may be as high as 85% by age 70 years.[3]

The Cancer Genetics Studies Con-sortium recently published its recommendations for follow-up care of people with an inherited predisposition to breast cancer because of mutant genes.[4] The consortium concluded that identifying people with the relevant mutations is a necessary first step in improving prevention and treatment. Early breast and ovarian cancer screening was recommended for people with *BRCA1* mutations and early breast cancer screening for those with *BRCA2* mutations.

The management of breast cancer should surely include its prevention among high-risk individuals. We suggest that the steering committee seek the advice and involvement of the genetic community for the next version of these guidelines.

Bassam A. Nassar, PhD, MB, BCh
Mark D. Ludman, MD
M. Teresa Costa, MD
J. Philip Welch, MB, ChB, PhD
Charles A. Butts, MD
Jonathan R. Love, MD
Heather Hogg, BSc, RN
M. Jill Beis, MSc
Co-participants
The Maritime Hereditary Cancer Programme
Dalhousie University
Halifax, NS

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[The chair of the steering committee responds:]

On behalf of the Steering Committee on Clinical Practice Guidelines for the Care and Treatment of Breast Cancer I thank these contributors for their suggestions. The following comments are my own.

In reply to Drs. Mahoney, Brown and Godfrey, I would point out that breast reconstruction and lymphedema were high on the approximately 20 topics first considered by