

erable support for a “patients’ bill of rights” that would give patients the right to sue the insurer as well as the doctor when performance is inadequate [see page 877]. This must also apply in Canada because Canadian government health insurers cannot be less responsible than their private, for-profit counterparts in the United States.

The major problem with Canadian medical care insurance is the lack of timely access to service, as witnessed by our long waiting lists. A patients’ bill of rights in Canada would give patients the right to sue the insurer — the insuring arm of government — for long delays in treatment. Adoption of this principle would do more to shorten waiting lists than all the reports from government commissions and inquiries, laid end to end.

This plan would in no way violate the Canada Health Act, nor would it lead to two-tier medicine or promote the privatization of medicine. It would simply compel the government to implement the act’s principles instead of paying them lip service.

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## Raloxifene and breast cancer

**W**e wish to acknowledge Kathleen Pritchard and colleagues for bringing to light a number of important issues regarding raloxifene and breast cancer.<sup>1</sup>

In a recent animal study, the effects of raloxifene on several breast cancer cell lines (which had been implanted into athymic mice) were investigated.<sup>2</sup> In the MCF7 (tamoxifen-sensitive) cell line, no significant growth was noted with either tamoxifen or raloxifene. In the MCF7TAMST cell line (a tamox-

ifen-resistant line exposed to tamoxifen for 5 years) “there was no significant difference between tamoxifen and raloxifene, in combination with E2 [estradiol] on tumor growth.” Neither the method by which study doses were chosen nor the use of a control group of mice was mentioned in the abstract. Although we agree with Pritchard and colleagues that raloxifene would not be the osteoporosis treatment of choice in women with tamoxifen-resistant breast cancer, it is premature to extrapolate the results of limited animal-model studies on the effects of raloxifene on tamoxifen-dependent breast cancer cell lines to disease-free humans.

We do not agree with Pritchard and colleagues that “raloxifene is very similar to tamoxifen.” There are differences in their respective tissue-specific effects that translate into distinct clinical profiles. For example, while tamoxifen (a triphenylethylene compound) has been shown to have stimulatory and carcinogenic effects on the human endometrium,<sup>3</sup> raloxifene (a benzothio- phene) has been proven to have no adverse effects on the endometrium.<sup>4,5</sup> The risk of venous thromboembolic events with raloxifene is similar to that seen with either tamoxifen or hormone replacement therapy.<sup>6,7</sup>

We agree that raloxifene is not currently indicated for breast cancer prevention and that it should not be used as a substitute for tamoxifen as adjuvant therapy for breast cancer.

Three large ongoing trials involving over 35 000 women, with reduction of breast cancer as the primary endpoint, will help to further clarify the role of raloxifene in the prevention of breast cancer.

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## References

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6. Grady D, Wenger NK, Herrington D, Khan S, Furberg C, Hunninghake D, et al. Postmenopausal hormone therapy increases risk for venous thromboembolic disease. The Heart and Estrogen/progestin Replacement Study. *Ann Intern Med* 2000;132:689-96.
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## Corrections

**D**r. Elmira Buxton was predeceased by her husband, Dr. Nigel Buxton. Incorrect information appeared in a recent death notice.<sup>1</sup>

## Reference

1. Deaths. *CMAJ* 2001;165(4):511.

**T**he last line of the final entry in the third column of Table 1 in a recent commentary by Ross Upshur and colleagues was cut off in error during production.<sup>1</sup> The entry should read as follows: “Unclear whether registrants are aware of the data and their uses.”

## Reference

1. Upshur REG, Morin B, Goel V. The privacy paradox: laying Orwell’s ghost to rest. *CMAJ* 2001;165(3):307-9.