Low-back pain

Buchbinder and Underwood are to be commended for providing useful information regarding back pain.¹ However, they may not have had problems in understanding heterogeneity between studies outlined in a related *CMAJ* article² had the studies taken physical examinations into account.

Chronic back pain may be due to either an ongoing nociception or neuropathy affecting spinal nerves, both of which have different causes and therefore present with completely dissimilar physical findings.³ A correct diagnosis and proper treatment would lead to a quicker resolution. Without a correct diagnosis, treatment would be empirical and haphazard.

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The authors respond

We thank Dr. Gunn for his letter¹ regarding our article.² We acknowledge that nonspecific low-back pain may have a variety of causes; however, current diagnostic techniques, including physical examination, are unable to reliably identify the source of pain in most patients.³ As well, although identifying treatment effect modifiers (characteristics that identify subgroups of patients who might respond better to a particular treatment) may yet be possible, evidence for their existence is not yet convincing.⁴ We would be interested in any robust research showing that the classification system proposed by Dr. Gunn is a treatment effect modifier. In the meantime, guidelines typically recommend triage of patients with low-back pain into 1 of 3

categories: nonspecific low-back pain (the vast majority), back pain potentially associated with radiculopathy or spinal stenosis, or back pain potentially associated with another specific spinal cause.⁵

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Data to support PSA screening for younger men lacking

Roobol and colleagues¹ claim that in the European Randomized Study of Screening for Prostate Cancer (ERSPC) “there was actually a small treatment advantage for men with high-risk prostate cancer who had been randomized to the control arm of the study.”² In fact, Wolters and colleagues² stated, “A control subject with high-risk PC [prostate cancer] was more likely than a screen subject to receive radiotherapy, expectant management or hormonal treatment instead of radical prostatectomy,” suggesting no treatment advantage to the control arm. Treatment data from the other ERSPC countries has not been published. In contrast, in the US Prostate, Lung, Colon

and Ovary trial (PLCO), treatment was equivalent in the 2 arms by stage.³

Roobol and colleagues¹ state there was “poor compliance with biopsy recommendations” in PLCO. It was the policy in PLCO not to recommend biopsies. Reports on screening test results were sent to the participant and his physician, and they determined subsequent investigation. Many decided against immediate biopsy; by 4 years 80% of the abnormal tests were resolved.⁴

Roobol and colleagues claim that “the risk–benefit ratio shifts dramatically for healthy men with a long life expectancy.”¹ They comment that in PLCO “older age at randomization was associated with increased risk of dying from prostate cancer, even in the screening arm.” Higher rates of death from prostate cancer at older ages are to be expected, but that does not indicate a benefit from screening at younger ages. Men given a diagnosis of prostate cancer following PSA (prostate-specific antigen) screening at younger ages will live longer with the adverse consequences of treatment. In the ERSPC, only those aged 65–69 at randomization showed a significant reduction in prostate cancer mortality.⁵ To infer or suggest that the ERSPC trial showed a better risk–benefit ratio for younger than older men is highly misleading.

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