tionships and career choice decisions.[2,4,6,7] Also, because of their sexual orientation, GLBT students and residents are often the targets of unprofessional behaviours, such as harassment and academic mistreatment, from their supervisors and faculty members.[9,10]

Although Canadian medical schools have been proactive in supporting other underrepresented groups in the profession, such as women and Aboriginal medical students, more work is needed to address the needs of GLBT medical students.

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I thank Louie Chan and Shelley Turner for their comments, in response to my recent *CMAJ* piece,[1] on a very important issue that faces GLBT medical students. I agree with them that medical schools across Canada need to be more proactive on this subject.

At the University of Alberta we are trying to address GLBT issues in several ways. The first was through an educational session on GLBT issues with our student advisors. We are hoping to integrate diversity modules into our medical curriculum and are developing a support group for staff, residents and medical students who are dealing with GLBT issues within our medical school. In 2004 I attended a conference of the Canadian Rainbow Health Coalition (a national organization that provides a means for people working on GLBT health and wellness issues to network and advocate together) and tried to network that organization with the Association of Faculties of Medicine of Canada. The Coalition is also developing educational materials that I hope can be used in the medical curriculum.

Although there is a long road ahead to completely change the attitudes of people within the medical field, I am hopeful that continued small advances will eventually lead to a safe and healthy environment for all minority medical student groups.

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An unusual crystal

We were most interested in the report by Joe Dylewski and colleagues on acute monoarticular arthritis caused by birefringent Maltese cross-like crystals composed of lipids.[1] Arthritis has been reported in patients with hyperlipidemias, especially type II.[2] Although Glueck and colleagues[3] reported synovitis in such patients, the body of evidence favours a periarticular site of inflammation.[4] Cholesterol crystals have been identified in some patients but do not appear to be particularly inflammatory.[5] To date we know of no other report of Maltese cross-like crystals in patients with arthritis associated with hyperlipoproteinemia.

Why arthritis is associated with hyperlipoproteinemia remains a mystery. Perhaps high levels of blood blood lipids of a certain type act as a source of lipid-bound macroenzymes.[6] It is perhaps germane that high concentrations of trypsin and lipase resulting from pancreatic disease cause synovial fat necrosis with either a mono- or polyarthritis and subcutaneous necrosis.[7,8]

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We appreciate the interest shown by Drs. Buchanan and Kean in our case.[1] As mentioned in the text, elevated levels of lipids in the serum and/or synovial fluid are unusual in Maltese-cross crystal-associated arthritis (only 2 of 13 reported cases). The references cited in our text suggest that the lipid-containing crystals are formed in the synovial fluid by various proposed but unproven mechanisms. They are not the product of an abnormal

lipid concentration in the serum. The rareness of this entity is corroborated by one of us (I.R.) who has been examining synovial fluid for crystals for over 15 years and has never seen this form of crystal before.

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Coxibs and cardiovascular risk

Jill Cotter and Eric Wooltorton[1] highlight the importance of differences in cyclooxygenase 2 (COX-2) selectivity as a potential explanation for the prothrombotic effects of coxibs. However, they neglect important evidence that celecoxib might not share the same cardiovascular (CV) risk as rofecoxib.

An increased thrombotic risk with celecoxib has been demonstrated only in the APC (Adenoma Prevention with Celecoxib) study.[2] On the other hand, data from the CLASS trial[3] and a meta-analysis of multiple controlled trials[4] have not uncovered any prothrombotic effect of celecoxib compared with either conventional NSAIDs or placebo. This is consistent with many observational studies that found increased CV risk with both high and low doses of rofecoxib but not with celecoxib.[5-7]

It is noteworthy that the APC trial demonstrated a trend toward increased CV risk of celecoxib among ASA users. Celecoxib might have caused this effect by interference with ASA-induced inactivation of platelet COX-1, which can occur at significantly lower concentrations of celecoxib than are necessary for inhibition of platelet COX-1 activity.[8] This notion does not contradict our previously stated view that ASA might mitigate CV risk associated with coxibs.[9] However, a recent case–control study showed that the risk-modifying effects of ASA were seen only at a low dose but not at a high dose of rofecoxib.[7] We have, therefore, suggested that higher doses of coxibs could also interfere with the anti-inflammatory effects of ASA mediated by COX-2.[10]

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Jill Cotter and Eric Wooltorton[1] indicated that COX-2 inhibitors appear to increase the risk of cardiovascular adverse events in a dose-related fashion. The CLASS and VIGOR clinical trials of COX-2 inhibitor(s) suggested that both celecoxib and rofecoxib can increase the risk of cardiovascular events.[2] The VIGOR trial compared rofecoxib with the NSAID naproxen in patients with rheumatoid arthritis[3] and indicated a 5-fold increase in the relative risk of developing serious cardiovascular events between the rofecoxib group and the naproxen group.

Clinical data have shown that expression of COX-2 is upregulated by inducible nitric oxide synthase (iNOS) that might play a protective role in the