

Letters Correspondance

Introducing medical students to CAM

Verhoef et al¹ identify some important issues in their discussion of undergraduate medical education about complementary and alternative medicine (CAM) therapies.

Understanding how patients conceive of their health is important for effective communication. Just as it behooves compassionate physicians to be sensitive to the religious beliefs and cultural backgrounds of their patients, so it is also important that doctors be aware that their patients might have health beliefs, such as alternative medicine, that range from the somewhat-plausible-but-unproven to the fanciful.

One problem that arises, as noted by the interviewees, is how to introduce students to this topic “without seeming to endorse it.”

We believe that education about CAM is most appropriate in the context of teaching students about:

- how inert therapies can appear to be effective,
- what types of alternative therapies are popular and what are their principal claims,
- how desperate or fearful patients will seek hope regardless of evidence,
- the different ways that patients understand their health, and
- why the “evidence” behind CAM is not accepted by the scientific community.

Who, then, should be in charge of teaching students about these issues? Verhoef et al propose the use of CAM “champions.” But is this the best way to deliver objective information? If, for example, psychic healing were currently in vogue, one might propose a stand-alone course led by a faculty champion with experience in that area. To do otherwise might lead to the assumption that the appropriate “experts” had not been sought. After all, how can you really “know” about something unless you believe in it? Others, however, might be concerned that psychic healers (even the ones claiming to be

evidence based) might not be familiar with how to critically appraise the research pertinent to that field and that any course put forward by such proponents would simply be a promotional enterprise addressing none of the five points above.²

Our experience with such courses led by CAM champions has not been reassuring, and the examples of existing CAM programs (again, led by CAM champions) cited in the article³⁻⁵ do not take a non-promotional approach. One survey of existing CAM courses in US medical schools indicated that most were led by proponents clearly advocating these therapies.⁶⁻⁸

We propose that undergraduate CAM materials should be presented without promotion by faculty interested in critical and reflective discussions of the five points listed above. Course materials focusing on these points are being developed for students at the University of British Columbia in Vancouver, and comments and suggestions are welcome.

—Lloyd Oppel, MD, MHSC, CCFP(EM)

—Dale Hoshizaki, MD

—Richard Mathias, MD, FRCP(C)

—Morley Sutter, MD, PHD

—Barry Beyerstien, PHD

Vancouver, BC

by e-mail

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A career jump-start courtesy of the College

I am pleased and honoured to receive a Janus Scholarship and certainly thank the College of Family Physicians of Canada for this support.

I must admit that, after I had submitted my application for the scholarship, I put the decision about starting my Master's degree on hold. As family and golf competed with work and running over the summer, my enthusiasm for this new challenge was wavering. My wife has 1 year left on her second degree, and I thought that one adult university student per family was enough!

When I received your letter notifying me of the award, I was surprised to be successful. More gratifying, however, was the fact that I immediately became enthusiastic about the Master's degree. My application to the Master's program is accepted,

and I am registered for my first course. This is not the first time that the College has jump-started a new direction in my career!

—Preston Smith, MD, CCFP, FCFP
Moncton, NB
by e-mail

Must have been a spider

I am the physician camping in Algonquin Park who had the necrotizing skin lesion that you refer to in the introduction of your article on spider bites.¹ True, I did not see a spider bite me. Your list of differential diagnoses is complete, but none applied to my situation. The lesion appeared as I slept but took only 2 hours to reach maximal diameter and 6 hours to reach maximal necrosis depth, at which time the bulla burst, and I could see the depth of tissue loss. It did not worsen after that hyperacute onset; abrupt

cessation of progression rules out bacterial, fungal, or viral infections. There was only one huge bullous lesion on my arm—not typical of poison ivy or other contact reactions. It was clearly not a burn; I would have known if it were.

There was no surrounding redness. The margins were not undermined. I have no chronic medical conditions. The lesion was on my arm; that rules out pyoderma gangrenosum. The lesion healed with no treatment; that rules out cancer. There was no lymphadenopathy or systemic symptoms or contact with rabbits; that rules out tularemia. Pressure ulcer at the flexor crease of my elbow is not a credible diagnosis, either.

That leaves spider bite as a genuinely credible alternative. You hold too high a standard to prove spider bites as cause of such a lesion. For example, I often do not see mosquitoes bite me. That does not mean that the pruritic boggy papules that frequently appear on my skin in the summer are not mosquito

bites. I concede that *Loxosceles reclusus* might not have been the species that bit me. But I still have no doubt that it was a spider of some sort. There may be “myths” about spider bites. But that does not mean that lesions like mine are not due to spiders. It is the only credible diagnosis in this instance.

—John Nelson, MD
Fort Frances, Ont
by e-mail

Reference

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Response

Although we applaud your efforts to diagnose your necrotic skin ulcer, we remain convinced it was not the result of a spider bite. At this point, long after the event, likely the only realistic diagnosis is “idiopathic necrotic lesion.”

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The long list (referenced in our article) of necrotic conditions misdiagnosed as spider bites is far from exhaustive. You have ruled out only about a half dozen of the many listed conditions, and you still show no evidence to implicate a spider. Also, you would not be the first person to mistakenly rule out one of the many diagnoses more probable than spider bite. For example, we know of at least one person who unknowingly suffers repeated thermal burns and blames the subsequent lesions on spiders.

Your mosquito bite analogy is faulty. Mosquitoes (and other obligatorily hematophagous arthropods) actively seek out mammals and other vertebrates for the blood meals necessary for their survival. No spider does this. We are sure you have witnessed the actual bites of many individual mosquitoes representing a variety of genera and species. Therefore we are confident in your ability to diagnose certain types of lesions as *likely* resulting from the bite of a mosquito. No one has ever shown a causal relationship, however, between the bite of any Canadian spider and a necrotic lesion. You have no factual basis to blame a spider for your lesion.

In fact, apart from the rare cases of true loxoscelism, “necrotic arachnidism” is a myth. As Geoffrey Isbister states in his article¹:

This association [of necrotic ulcers and spiders] remains despite no significant evidence to support the involvement of spiders in necrotic ulcers. The medical community is by no means immune to the myth of necrotic arachnidism and is responsible for its persistence by not questioning the evidence or investigating necrotic ulcers in the same way as any other disorder.

Considering the current desire for evidence-based medicine as well as the medical community’s conservative nature and consequent reticence to accept new concepts, techniques, or remedies without proof, it astonishes us that spiders are so commonly and erroneously implicated as causative agents of idiopathic lesions. Apparently we have succeeded in convincing you

that “*Loxosceles reclusus* might [our emphasis on “might”] not have been the species that bit” you. We strongly urge you to accept that a *Loxosceles* spider *did not* bite you and that, furthermore, there is no evidence to suspect any spider in your case, or any of the other cases we report. To do otherwise contributes to the perpetuation of a lamentable decades-old medical myth.

—Robert G. Bennett, MSC, PHD
Saanichton, BC

—Richard S. Vetter
Riverside, Calif
by e-mail

Reference

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Good intentions, poor study design

Your article¹ “Caveat emptor. ‘Probiotics’ might not be what they seem” by Dr Brenda Huff caught our attention. As scientists working with the probiotic industry to improve standards and promote evidence-based efficacy substantiation, we can appreciate Dr Huff’s motivation for doing this project, especially because third-party verification of probiotic compliance with label claims is not available to consumers or health care professionals. We fully support recommendations for probiotic products to live up to label claims as per the recent FAO/WHO guidelines (see http://www.who.int/foodsafety/fs_management/en/probiotic_guidelines.pdf). Indeed, there are likely commercial probiotic products that do not comply with their label claims.

A proper intention does not, however, justify poor study design and use of improper media and poorly described methods. The choice of media for detection and enumeration are inconsistent with those optimal for detecting probiotic lactobacilli. The lack of clarity in defining abbreviations left us to make some assumptions (did BAP stand for bacterial alkaline phosphatase as stated or more common

blood agar plates? Did CNA stand for calcium nutrient agar or the more common colistin nalidixic acid agar?). Assuming that blood agar was the medium used, it is not well suited to the growth of commercial lactobacilli or bifidobacteria. It is a better choice for enterococci and pathogenic microbes. The media used are more suited to fecal analysis and are not specific for lactobacilli nor bifidobacteria. The preferred media for evaluation and enumeration of probiotic lactobacilli are de Man, Rogosa, Sharpe (MRS) agar or tomato juice agar. Also, the use of a "1:1000 loop" is not adequate for enumeration. To achieve a quantitative result, a defined quantity of powder (1 to 10 g) should have been weighed, reconstituted, and serially diluted.

We suspect that examination of a Gram stain would have revealed a high number of Gram-positive rods (comprising lactobacilli and bifidobacteria) in most of these samples. Although a Gram stain will not differentiate between live and dead cells, a dominance of these microbes would have called into question growth methods that did not determine their presence even at the lowest level of recovery. Finally, the method used to determine the genera of the microbes isolated was not described. It is therefore impossible for readers to assess the likelihood that correct identifications were reported. Molecular techniques are preferred for identification of probiotic microbes.

The author concludes from this study that probiotics should not be recommended at this time. This is clearly an irresponsible and damaging conclusion, indicting an entire industry on the basis of 10 samples evaluated using poor and outdated methods. This paper will likely discourage health care professionals from using perfectly good products that could provide clinical benefit to their patients. Further, the author's statement that "No current government regulations apply to over-the-counter probiotic products" is simply untrue. In Canada, these products fall under the jurisdiction of Health Canada's Natural Health Products Directorate, and some previously registered drug identification number products fall under the Therapeutic Products Directorate.

—Thomas A. Tompkins, PHD
Institut Rosell Inc,
Montreal, Que

—Mary Ellen Sanders, PHD
Dairy and Food Culture Technologies

Centennial, Colo
President, International Scientific Association for
Probiotics and Prebiotics
by e-mail

Reference

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Probiotics

I was surprised at the results obtained in the article "Caveat emptor. 'Probiotics' might not be what they seem."¹ My understanding is that manufacturers must follow good manufacturing practice (GMP) as outlined in the Natural Health Products regulations defined by Health Canada.

According to Health Canada, as of January 1, 2004, probiotics (and all other natural health products) are subject to the requirements of the Natural Health Products Regulations, which include GMP, site licensing, and product licensing requirements. Quality control must be built into *each batch* of the product during all stages of the manufacturing process, and constant testing is required to monitor this quality. All raw materials are required to conform to a standard and are tested to their specifications to ensure compliance. Suppliers must provide a Certificate of Analysis for each batch of raw material. In addition, a qualified quality assurance person should be checking throughout the manufacturing, packaging, labeling, testing, and releasing steps (personal communication from Health Canada, Natural Health Products Division; June 2004).

It is not entirely clear from Dr Huff's article whether she followed GMP guidelines when conducting her study. My understanding is that the culture and counting of bacterial flora in this situation needs to be quite specific and standardized. If the author used a different culture media and counting techniques, then these results are clearly neither valid nor comparable with GMP guidelines.

Could Dr Huff please clarify her methods? If her methods are different from the standardized GMP guidelines, I must wonder why this variance was not dealt with in peer review. Arbitrary methods would cast doubt on the results and therefore the conclusions.

—Edward Leyton, MD, CCFP
Kingston, Ont
by e-mail

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Early screening for diabetes mellitus: has it been overstated?

The article¹ by Dr Stewart Harris and Ms Cynthia Lank in the March issue and the subsequent letter to the editor by Dr Jayabarathan² raise some interesting concerns about the Canadian Diabetes Association's clinical practice guidelines for preventing and managing diabetes in Canada.

The Expert Committee recommends screening all Canadians older than 40; this recommendation appears largely based on a Canadian study completed in 1998.³ This study arbitrarily chose to test glucose levels in patients older than 40; the study demonstrated a prevalence of 1.4% undiagnosed diabetes and 1.7% undiagnosed glucose intolerance in the 40 to 45 years age group. These numbers are smaller than in the older age groups and do not support the recommendation to push the screening age back 5 years. The position of the Expert Committee is certainly not shared by other groups: the American Diabetes Association in January 2004 maintains its recommendation to screen adults older than 45⁴; the US Preventive Services Task Force in 2003 concluded that there was insufficient evidence for screening asymptomatic adults at any age.⁵

In his response⁶ to Dr Jayabarathan's letter, Dr Harris questions her interpretation of the UKPDS study; Dr Harris reaffirms his interpretation that this study confirmed the protective effects of intensive glycemic control. He fails to note that the results of the UKPDS have been questioned in a number of articles.⁷⁻¹⁰ The UKPDS demonstrated that intensive glycemic control using various hypoglycemic agents did significantly reduce microvascular outcomes (chiefly retinopathy requiring photocoagulation) and, to a lesser degree, progression of microalbuminuria; there was no significant reduction in the incidence of blindness, of renal failure, or of macrovascular events. An isolated finding that metformin therapy in obese diabetic patients did significantly reduce

cardiovascular events and overall mortality appears to have been generalized to the broader topic of glycemic control by any means. An observational study as part of the UKPDS demonstrated that patients with higher glycosylated hemoglobin (A_{1c}) have a greater risk of microvascular and macrovascular events but did not demonstrate that lowering the levels altered the risk. The UKPDS did demonstrate that tight blood pressure control was of great importance in modifying outcomes.¹¹

Dr Harris indicates that early detection and treatment of the prediabetic state will prevent development of overt diabetes and delay onset of target-organ damage. Two recent clinical trials have confirmed the effectiveness of lifestyle changes^{12,13}; unfortunately, the intensive interventions (multiple diet education sessions, personal physical training supervision, regular follow-up visits and prompts) do not translate into a practical general population strategy, and the sad reality is that attempts to modify lifestyles in a family physician's office are frustrating and generally unsuccessful.¹⁴ Three clinical trials have shown normalization of glycemic levels using metformin, acarbose, or troglitazone (which has since been removed from the market); one might question the wisdom of instituting pharmacotherapy at such an early stage, thereby increasing the cumulative risk of side effects and drug-related complications, without any evidence to support the hypothesis that this will alter anything but the glycemic level.

Finally, the Expert Committee overlooks the social, emotional, and economic impact of labeling patients. Attaching a "sick" label to patients is not without consequences. The question of false-positive results has also not been addressed: between 12.5% and 42% of men diagnosed with diabetes reverted to normoglycemia after 2.5 to 8 years.^{15,16}

Dr Harris underplays the significance of clinical practice guidelines; they most certainly affect practice and standards of care; otherwise Expert Committees would not be expending such energy to develop them. Unfortunately, the Expert Committee of the Canadian Diabetes Association might have overstated the effectiveness of early

detection and intensive treatment of diabetes mellitus.

—François-Gilles Boucher, MD, CCFP, FCFP
Toronto, Ont
by e-mail

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on the College's website at www.cfpc.ca
by fax to the Scientific Editor at
(905) 629-0893 or by mail to
Canadian Family Physician
College of Family Physicians of Canada
2630 Skymark Avenue, Mississauga,
ON L4W 5A4

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(905) 629-0893 ou par la poste
Le Médecin de famille canadien
Collège des médecins de famille du Canada
2630 avenue Skymark, Mississauga,
ON L4W 5A4