Correspondance

We are concerned that the letter by Sana R. Sukkari ([Can Med Assoc J 1997;156:768-9]) misrepresents the relationship that Dr. Manson and I had with the pharmaceutical industry. Upon invitation, we wrote an editorial about pharmacotherapy for obesity.1 In the process, a series of miscommunications and misunderstandings occurred between the New England Journal of Medicine (NEJM) and us.

As stated in our subsequent letter to NEJM,2 we had briefly served as scientific consultants to Servier, the manufacturer of dexfenfluramine (Redux) and had submitted a proposed disclosure statement to NEJM. NEJM’s written disclosure policy statement had ambiguities, and our direct communications with their editorial staff were misinterpreted. This led to a series of misunderstandings.2

Most important, we had and have no financial interest in any manufacturer of anti-obesity drugs, nor do we stand to gain from the commercial success of any of their products. The opinions that we expressed were entirely our own and independent of industry. The editorial was carefully written and was in no way intended as an endorsement of appetite suppressants. We urged long-term studies and cautious prescribing to patients with medically significant obesity who had failed an exercise and diet program.

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References

Managing benign prostatic hyperplasia

As a very busy urologic surgeon in Toronto, I found that after reading the article “Efficacy and safety of finasteride therapy for benign prostatic hyperplasia: results of a 2-year randomized controlled trial (the PROSPECT Study)” ([Can Med Assoc J 1996;155:1251-9], by Dr. J. Curtis Nickel and colleagues, I was even more confused than before as to the appropriate management of benign prostatic hyperplasia (BPH).

Patients with symptomatic BPH usually require or request some treatment. To say that finasteride is a very effective, reliable and safe treatment. To say that finasteride is a viable and safe alternative to watchful waiting is confusing and inappropriate. If one has embarked on watchful waiting, then there is an understanding between the patient and the physician that no intervention is necessary because the symptoms or signs of BPH are not significant. There should be no therapy, not even a “safe, nonoffensive therapy,” that is not required.

If, however, one has determined that the symptoms (as defined by the symptom score), urinary flow or sequelae of BPH demand treatment, then one must prescribe the most effective, reliable and safe treatment.

There is no golden pill that works for everyone, even if patients have the same size of prostate. In my hands, terazosin has been very safe and reliably effective.

I find it hard to reconcile the fact that, in a recently published study of BPH in veterans,1 the investigators found no improvement in the patients taking finasteride compared with those taking a placebo. Even if we accept the retrospective analysis that finasteride, because of its mode of action, should be more effective in larger prostates, we still find significant discrepancies. In the subset of patients with prostate volumes greater than 50 mL, the urinary flow improved by 2.5 mL per second in the group taking finasteride v. 3.9 mL per second in the group taking terazosin. A similar trend was found in the symptom-score improvement. Another unexpected discovery in the study was that the prostate-specific antigen (PSA) level decreased in the group taking terazosin, but not in the group taking finasteride.

It seems logical that finasteride would work more effectively in larger prostates and that the patients’ PSA level would decrease. However, this was not corroborated in the 2 studies.

Logically, as well, α-blocking agents should be more effective in the smaller prostates usually seen in younger patients; in such patients, the impotence that is a side effect of finasteride would be more troublesome.

At the primary care level, once one has decided that therapy is indicated

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Drug to treat obesity: editorial writer responds

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