

osteoporosis update



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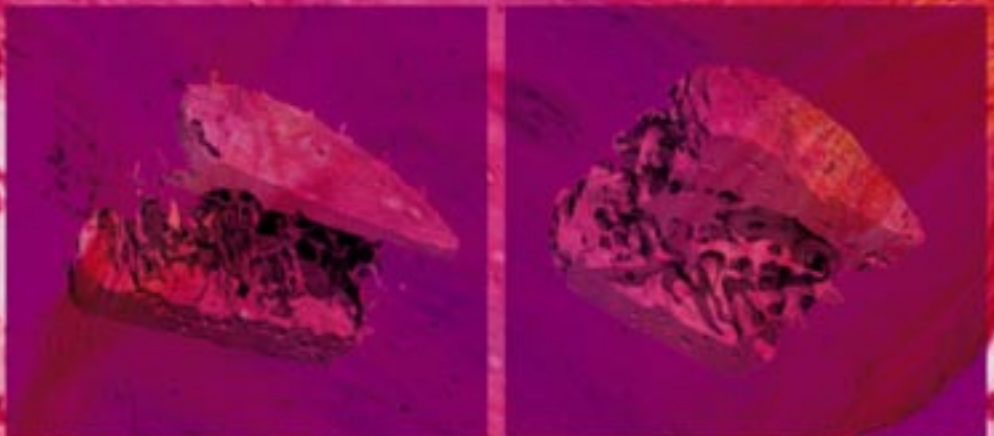
a practical guide for Canadian physicians

Fall 2004 vol. 8 no. 3

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case

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editorial comment

New kid on the block

Following soon after the recent approval of teriparatide (rhPTH[1-34]) by Health Canada, the Fall issue of *Osteoporosis Update* discusses this new, much-anticipated osteoporosis therapy. The first in a novel class of anabolic treatments, teriparatide acts to improve skeletal microarchitecture and increase bone mineral density (BMD) by stimulating new bone formation. Individuals who we hope will benefit from this option include postmenopausal women as well as men with severe osteoporosis, known to have already suffered a fragility fracture. Additional candidates for teriparatide therapy include patients not responding to antiresorptive agents currently in use, and those with established glucocorticoid-induced osteoporosis who require long-term steroid treatment.

The addition of teriparatide to our menu of available osteoporosis treatment

agents is exciting, and may serve to change the therapeutic approach of physicians.

Because of concerns about administration and monitoring, for now, family doctors may wish to refer potential candidates for teriparatide therapy to a specialist. Physicians can also direct their patients to programs

that provide information on use and reimbursement, together with education on self-injection.

Also special to this issue is the inclusion of a quick reference card on bone densitometry, which addresses issues such as who should be assessed and treated for osteoporosis, who is eligible for BMD testing, serial

monitoring, interpretation of results from the BMD report, etc. Below, Dr. Aliya Khan, Chair of the Canadian Panel of the International Society for Clinical Densitometry (ISCD) and leading force behind this project, introduces the pocket guide. We hope it will serve as a useful tool for physicians in their clinical practices.



Anthony Hodsmann, MB, BS, FRCPC

Professor, Department of Medicine, Division of Nephrology, University of Western Ontario
Medical Director, London Regional Osteoporosis Program.

Anthony B Hodsmann

The Canadian Panel of the ISCD, comprised of Canadian and international bone experts, has developed standards in order to establish the minimum level of acceptable performance for the practice of bone densitometry in Canada. These guidelines enable physicians to appropriately test and treat premenopausal and postmenopausal women, men and children. Recommendations are based on the use of dual energy x-ray absorptiometry (DXA) — the technology of choice for BMD testing — at central skeletal sites.

This pocket card summarizes the key take-home messages for clinicians from the Standards I and Standards II documents in a convenient, easy-to-use format for handy reference. I would like to acknowledge and thank the Canadian Panel members (see back cover of guide for list) for the major time and effort they have put into preparing and reviewing these standards. We also gratefully acknowledge the unrestricted educational grants received from Eli Lilly Canada Inc., Merck Frosst Canada, Inc., Procter & Gamble Pharmaceuticals and Aventis Pharma Inc., which make the publication and distribution of this guide possible.

— **Dr. Aliya Khan**, Chair, Canadian Panel of the ISCD, McMaster University

Breast cancer treatment and osteoporosis risk



Laura, age 61, had a “lumpectomy” for an invasive ductal carcinoma just under 2 cm in diameter. There was no evidence of lymphatic or vascular invasion, and the lesion was strongly positive for estrogen and progesterone receptors. Five years earlier, she had a pulmonary embolus following a cholecystectomy. She and her physicians have decided upon adjuvant therapy with an aromatase inhibitor, rather than tamoxifen, for her breast cancer.

Key questions

- What is the effect of adjuvant therapy on the risk of pulmonary embolus?
- What is the impact on bone mineral density (BMD) and osteoporosis risk?
- How should Laura be assessed for osteoporosis risk?

Dr. David Hanley comments:

Although tamoxifen may have some protective effect against cardiovascular disease, there is a significant rise in the risk of deep vein thrombosis and pulmonary embolus with this agent. Aromatase inhibitors (AIs) are not known to increase this risk. In a study comparing tamoxifen with the AI anastrozole, the relative risk of thromboembolic events with anastrozole was 0.59.

Estrogen is an important hormone for building and maintaining bone mass and strength in women, even after menopause. The enzyme aromatase, found in other tissues of the body besides adrenals and gonads, most notably skin and fat, catalyzes the conversion of androgens to estrogens. After menopause, estrogen synthesis continues due to the conversion of adrenal androgens in peripheral tissues. Individuals with large amounts of fat make more estrogen. In the Study of Osteoporotic Fractures, postmenopausal women with the highest endogenous estrogen production had the lowest risk of osteoporosis (Cummings SR, *N Eng J Med* 1998). In the same cohort, unfortunately, higher estrogen production was also associated with a greater risk of breast cancer (Cauley JA, *Ann Intern Med* 1999).

Dr. David A. Hanley is a Professor in the Department of Medicine, Division of Endocrinology and Metabolism at the University of Calgary, and Medical Director of the Grace Osteoporosis Centre, Calgary, Alberta.

AIs have come to the forefront of adjuvant therapy for breast cancer but, by blocking estrogen synthesis, may also cause BMD loss. The ATAC (Arimidex, Tamoxifen, Alone or in Combination) trial comparing anastrozole, tamoxifen, and the combination, reported significantly more fractures in women receiving the AI. Whether this reflects the benefits on bone of tamoxifen, a selective estrogen receptor modulator (SERM), or a major detrimental effect of the AI, is not clear. Raloxifene, another SERM associated with reduced incidence of breast cancer, is a front-line therapy for prevention and treatment of postmenopausal osteoporosis (OSC Clinical Practice Guidelines, Brown J, Josse R, *CMAJ* 2002). Preliminary evidence suggests that the nonsteroidal AIs may cause loss of bone density, but it is not yet certain whether exemestane, a steroidal AI, can increase the risk of osteoporosis.

ADDRESSING BONE RISKS

In premenopausal women, both AIs and tamoxifen will induce a state of relative estrogen deficiency and cause bone loss. As physicians resort more often to the use of AIs, they will need to pay attention to the potential for osteoporosis and fractures associated with these drugs.

In this patient, it would be appropriate to obtain a BMD measurement and follow the OSC guidelines for therapeutic intervention. Attention to calcium and

vitamin D nutrition, as well as other lifestyle risk factors for osteoporosis, is important. Patients given AIs who do not meet criteria for preventive therapy with a bisphosphonate should have repeat BMD tests in one year to ensure they are not suffering significant bone loss.

In summary, adjuvant therapy for breast cancer has been shown to significantly reduce the risk of recurrent disease and tamoxifen has long been the frontline agent. SERMs bind to estrogen receptors in cells and act primarily as estrogen antagonists in certain tissues such as breast. In other tissues, including bone, SERMs act as partial agonists and are linked to increased BMD in postmenopausal women. A large clinical trial comparing raloxifene to tamoxifen in breast cancer prevention is in progress. Other approaches include:

- agents that inhibit the final step in estrogen synthesis from androgens (e.g. the AIs anastrozole, letrozole and exemestane)
- drugs that inhibit pituitary stimulation of estrogen synthesis (e.g. the LHRH agonist goserelin)
- an estrogen receptor antagonist that has no agonist activities (e.g. fulvestrant)

Clinical trials are underway to test aromatase inhibition in combination with a SERM. This may have benefits in breast cancer management as well as in preventing bone loss. At present, however, approved therapy is with a single agent.

Because even low-level endogenous estrogen production after menopause enhances bone, agents like AIs might be expected to cause BMD loss. Given our present state of knowledge, if therapy with an AI is chosen, attention must be paid to bone health. 🌿

Complete references are available upon request from: mackinnon@parkpub.com

The promise of PTH

New treatment option for severe osteoporosis



Prefilled delivery device

Preventing fractures and avoiding their debilitating effect on functioning and quality of life is one of the major goals, and challenges, of osteoporosis management. For cases of severe osteoporosis — where bone mineral density (BMD) is low and patients have already experienced a fragility fracture — a new parathyroid hormone (PTH) treatment is now available that may provide additional benefits over current treatments for some patients.

The recent approval by Health Canada (June 2004) of a PTH analog, teriparatide (Forteo™), provides a promising new option for building bone strength and improving bone structure in people with severe osteoporosis. Parathyroid hormone is the first in a new class of osteoporosis treatments. An anabolic agent, it does not simply halt bone loss, but increases bone mass by stimulating new bone formation.

A NOVEL MECHANISM OF ACTION

Bone loss in osteoporosis is due to an imbalance in the remodelling process, i.e. the rate of bone resorption surpasses formation. Parathyroid hormone is crucial to maintaining calcium homeostasis. It acts directly to increase calcium resorption at renal tubules, and indirectly to enhance calcium absorption by the intestine through its renal effect to increase 1,25 (OH)₂ vitamin D synthesis. PTH is involved in regulating bone remodelling, though not necessarily in the overall determination of skeletal mass.

Teriparatide is the market-approved 34-residue amino-terminal fragment of recombinant human PTH (rhPTH [1–34]). Numerous studies have shown that daily teriparatide

injections lead to increased mineralization, total bone mineral content and cortical bone thickness, while decreasing the incidence of fractures in women and men. The therapy also improves trabecular connectivity within bone.

As an anabolic stimulant, teriparatide leads to the formation of new bone with an apparent dissociation between bone formation and resorption rates. This is in contrast to the action of current antiresorptive therapies — including bisphosphonates, calcitonin, estrogens and raloxifene (a selective estrogen receptor modulator) — which slow the remodelling and resorption processes, but have no direct effects on bone formation. These drugs act on osteoclasts to preserve bone mass, stabilize bone structure and quality and reduce fracture rates. By reducing the rate of bone remodelling, antiresorptive drugs increase the extent of secondary mineralization throughout the skeleton, producing a rise in BMD, but with no net change in bone architecture.

Table 1 compares the anabolic PTH-mediated therapy with the mode of action of antiresorptive therapies.

Does combining therapies work?

Because the mechanism of conventional osteoporosis medications differs from that of PTH, there was speculation as to whether combination therapy would result in additive bone-strengthening effects. Studies comparing PTH and alendronate — alone and in combination — concluded that concurrent administration may blunt the PTH-mediated BMD response. While no clinical information exists on interactions between other bisphosphonates and PTH, these drugs are not recommended for concomitant therapy with PTH. Research suggests, however, that, in combination with estrogen and possibly raloxifene, the anabolic response to PTH is not impaired, although there is no evidence for any additive benefit.

BMD starts to fall again once PTH therapy is stopped. Though more large-scale trials are needed, there is some evidence that sequential therapy with antiresorptive agents after PTH treatment might lead to improved BMD.

MEASURE OF EFFICACY

Fracture reduction, a major treatment goal, can be used as a yardstick to measure the efficacy of PTH therapy. Neer et al compared daily injections of 20 µg or 40 µg of teriparatide with placebo (mean treatment duration approximately 18 months) in postmenopausal women who had prior vertebral fractures. The two doses were found to be similar with respect

to fracture reduction, but 40 µg PTH was more likely to have side effects. As well as causing a significant BMD increase, 20 µg of PTH was associated with a 65% risk reduction for new vertebral fractures and a 53% risk reduction for non-vertebral fractures (or 9.3% and 2.9% absolute risk reduction, respectively; $p = 0.05$).

SIDE EFFECTS AND LIMITATIONS

Adverse effects of PTH in clinical trials were generally mild and did not require discontinuation of therapy. Following injection, some patients experienced dizziness, nausea and leg cramps. The incidence of hypercalcemia is uncommon (about 3%), and mild when it does occur. Since PTH increases calcium absorption, total dietary and supplemental calcium should be limited to 1500 mg daily; it may be necessary to adjust supplements if serum calcium rises with PTH therapy. Adequate vitamin D intake (up to 1000 IU/day) is also advised.

Studies that found osteosarcoma occurring in rats after life-long daily administration of PTH (from infancy) have raised concerns. The clinical significance, however, is not likely to be relevant to adult humans. The recommended lifetime duration of teriparatide therapy is limited to 18 months in Canada (a relatively short span in adulthood) and the 20 µg daily dosage is far lower than the relative doses given to the rats. No other evidence links PTH or primary hyperparathyroidism to osteosarcoma and no cases occurred during the major teriparatide study (Neer RM et al, *N Eng J Med* 2001). In fact, this study

table 1 Anabolic vs antiresorptive therapy

(PTH) anabolic therapy	Antiresorptive therapy
<ul style="list-style-type: none"> Induces bone formation 	<ul style="list-style-type: none"> Halts or slows resorption
<ul style="list-style-type: none"> Stimulatory, increasing the rate of bone remodelling 	<ul style="list-style-type: none"> Inhibitory, both to resorption but also – tightly coupled to it – to bone formation
<ul style="list-style-type: none"> Increases number and activity of osteoblasts 	<ul style="list-style-type: none"> Decreases activity of osteoclasts
<ul style="list-style-type: none"> Increased bone formed in each remodelling unit, as assessed by osteon thickness 	<ul style="list-style-type: none"> Decreases size of remodelling space and number of resorptive cavities; increased BMD through secondary mineralization of preformed osteons
<ul style="list-style-type: none"> Increases cortical thickness Increases trabecular thickness and connectivity 	<ul style="list-style-type: none"> Preserves trabecular architecture

noted a significant reduction of other cancers following 20 µg of daily teriparatide therapy (relative to placebo, $p = 0.02$).

Since no data exist on its safety in adolescents or pregnant women, teriparatide should not be used in these groups. People under 50 years of age with low BMD are not recommended targets for this treatment. In addition, it is contraindicated in individuals with severe kidney impairment, primary hyperparathyroidism, hypercalcemia, and increased baseline risk for osteosarcoma (patients with Paget's disease, previous radiation therapy to the skeleton, and children or adolescents with open epiphyses).

Since teriparatide must be administered in 20 µg subcutaneous daily injections, patients are being offered special training when starting therapy. Family doctors will likely refer patients to specialty clinics to initiate therapy, in order that appropriate monitoring concerns can be addressed. Physicians who prescribe teriparatide can register their patients in the Forteo Customer Care Program. Nurses provide information on the program (including on reimbursement) and home care visits to train patients in administering self-injections (direct patients to the toll-free number: 1-877-436-7836).

ONGOING RESEARCH

At present, the clinically approved teriparatide is a 34-amino acid fragment of human parathyroid hormone. Research has been conducted on other analogs of PTH, and Phase III clinical trials are underway with full-length intact human parathyroid hormone, hPTH(1–84). Longer-term fracture and safety data, along with the great ability of PTH therapy to facilitate new bone formation, may provide clinicians with a whole new therapeutic approach for the treatment of osteoporosis. 🌟

References available on request from: mackinnon@parkpub.com

Key recommendations

- Candidates for teriparatide therapy may include:
 - Men and postmenopausal women with severe osteoporosis and a documented fragility fracture
 - Patients not responding to antiresorptive therapy
 - Patients with established glucocorticoid-induced osteoporosis, requiring long-term steroid treatment
 - Individuals at very high risk of fragility fractures (including those < 65 with very low BMD [T-score ≤ 3.5])
- Recommended duration of therapy in Canada is 18 months
- Discontinue alendronate when initiating treatment with teriparatide
- While no evidence to date supports concurrent antiresorptive therapy, these agents might be considered as maintenance following cessation of teriparatide
- Limit total calcium intake to 1500 mg/day and ensure adequate vitamin D intake (up to 1000 IU/day); it is advisable to measure pre-injection serum calcium levels after the first month of therapy (mild hypercalcemia may be treated by withdrawing dietary calcium supplement, reducing the dosing frequency of PTH or both), but routine serum calcium monitoring may not be necessary

Joint winners of 2004 Lindy Fraser Award

The Osteoporosis Society of Canada (OSC) was proud to present this year's Lindy Fraser Memorial Award to Dr. Jacques Brown and Dr. Robert Josse for their outstanding leadership role in spearheading the clinical practice guidelines revision project as well as for many years of service to the Society.

The OSC established the award in 1994 in honour of Lindy Fraser. After long years of battling osteoporosis alone, with no medical treatment until age 78, this courageous woman started the first support group for people with osteoporosis in Ottawa in 1981, at the age of 87. This initiative provided the driving force behind the founding of the OSC. Past recipients of the Lindy Fraser Award include Dr. Joan Harrison, Dr. Ed Yendt, Dr. George Jaworski, Dr. Tim Murray, Dr. Harold Copp, Dr. Jonathan (Rick) Adachi and Dr. Alan Tenenhouse.

ABOUT THE GUIDELINES

Beginning in 1999, the OSC undertook the task of updating the previous 1996 guidelines to reflect new research and clinical findings. Published in the *Canadian Medical Association Journal (CMAJ)* in 2002, the revised document represented the first evidence-based clinical practice guidelines for osteoporosis in the world. The approach consisted of retrieving 89,804 abstracts and reviewing, evaluating and grading 6,941 full citations based on the strength of the evidence.

Members of the Society's Scientific Advisory Council (SAC) and other osteoporosis experts from across the country devoted countless hours to the project. As Steering Committee Co-Chairs, Dr. Brown and Dr. Josse painstakingly oversaw the work of the Section Committee Chairs, who researched and developed

the guidelines, and members of the SAC, who reviewed and approved them.

The updated guidelines will help to translate the many scientific advances in osteoporosis research in recent years into clinical practice and alleviate the problem of underdiagnosis and under-treatment of this debilitating disease. They provide physicians with the best, most current evidence to ensure improved care — adapted to patient needs — and optimal health outcomes.

The 2002 guidelines focus on the prevention, diagnosis and treatment of osteoporosis and provide recommendations on bone mineral density testing, fracture risks, nutrition, physical activity and available treatment options.

Kudos to Drs. Brown and Josse for outstanding contributions to the osteoporosis community

While these guidelines have set a precedent in establishing an effective process, they are still a work in progress. Work has already begun to review the new literature and more frequent updates can be expected.

A LIFETIME OF DEVOTION

Drs. Brown and Josse have been members of the SAC for over 20 years. As past Chairs, they have also served on the Society's national Board of Directors.

Among their various contributions, they have both provided years of expertise and invaluable support to the Society and the Canadian osteoporosis community as experts on the editorial board of *Osteoporosis Update*.

Many thanks and heartfelt congratulations to Dr. Brown and Dr. Josse! 🌟



Dr. Robert Josse (left) and Dr. Jacques Brown were surprised and honoured to receive the Lindy Fraser Memorial Award during a reception at a recent conference of the European Calcified Tissues Society (ECTS) in Nice, France. The award recognizes outstanding contributions in research and education in the field of osteoporosis.

Dr. Jacques Brown is a rheumatologist and reputed Canadian authority in metabolic bone diseases. He is Clinical Professor in the Department of Medicine at Laval University, and Head of the Division of Rheumatology at the Centre hospitalier de l'Université Laval (CHUL) in Québec City.

He has authored and co-authored 104 papers and abstracts. His main research interests include Paget's disease of bone and osteoporosis. He is the Principal Investigator of many clinical research trials and Centre Director (Québec) for the Canadian Multicentre Osteoporosis Study (CaMos). He is also Principal Investigator of the disease management program ROCQ (Recognizing Osteoporosis and its Consequences).

Dr. Brown sits on the medical and advisory panels of several associations, including the OSC, the Arthritis Society, the Canadian Rheumatology Association, the American College of Rheumatology, the American Society for Bone and Mineral Research, the International Bone and Mineral Society and the Paget Foundation.

Dr. Robert Josse, an endocrinologist, is Associate Physician-in-Chief and past Chief, Division of Endocrinology and Metabolism at St. Michael's Hospital. He is a Professor of Medicine at the University of Toronto. His major research interests include calcium metabolism and osteoporosis as well as diabetes, hyperlipidemia and various nutritional metabolic problems. He is the director of the Toronto site of CaMos. He has published over 125 articles in peer-reviewed journals and 21 book chapters. He is a well-known national and international lecturer.

Dr. Josse is a scientific advisor to the OSC and sits on the Scientific Advisory Council of the International Osteoporosis Foundation. He is President of the Canadian Society of Endocrinology and Metabolism.

Our panel of specialists answers your questions



“If bone loss after menopause is associated with increased periosteal apposition (partially preserving bone strength), could a bone size/strength index be used along with bone density to predict fracture risk?”
— a GP in St. Andrew’s, NB

Dr. Bill Leslie responds: This topical question gets at one of the most exciting developments in bone densitometry and fracture risk assessment. Before considering the clinical role for these new geometric measurements, it is important to make some preliminary observations:

- Many fractures, even those of the hip and spine, occur in individuals who are not osteoporotic by conventional bone mineral density (BMD) criteria (T-score ≤ 2.5 standard deviations below the young adult mean). While BMD measurement using dual x-ray absorptiometry (DXA) provides an excellent, rapid and well documented classification of fracture risk, other features, particularly age and clinical risk factors for falling, are also important independent fracture risk predictors. At a time when much medical science seems to undervalue the clinical assessment, it is encouraging to note that several large cohort series have reaffirmed its feasibility and power in fracture risk stratification.
- Besides BMD, bone geometry and quality are other key determinants of strength. In fact, bone densitometers do not actually measure BMD directly. BMD is derived as bone mineral content divided by projected bone area and expressed in units of g/cm^2 . This calculation partially adjusts for differences in bone size, but is not completely satisfactory when the dimension is significantly larger or smaller than average (such as in children). Larger bones of identical density (g/cm^3) will give a greater BMD measure due to an increase in the undetermined third dimension (depth). Although growth is most striking during childhood and adolescence, bones continue to enlarge slowly — even in the elderly — due to periosteal bone apposition (around the exterior of bone) but with overall thinning of the cortical thickness due to continuing endosteal bone resorption. Gender appears to affect this, with men showing greater periosteal expansion than women.


Dr. Bill D. Leslie is a radiologist with the Departments of Medicine and Radiology at St. Boniface General Hospital, Winnipeg, and Chair of the Manitoba Bone Density Program Committee.

New software that can derive geometrical measurements (more sophisticated than the old-fashioned assessment of phalangeal and metacarpal cortical thickness or simple indices of femoral neck length) from conventional DXA scans has made us aware of the potential clinical implications of bone expansion. Structural analysis predicts that periosteal expansion should increase bone strength even when the total amount of bone calcium is the same (this would actually produce a decrease in the BMD measurement). But at a certain point, further expansion actually compromises strength. Numerous studies are now focusing on the alterations in bone geometry that occur with normal development, aging, disease and intervention. If this isn’t confusing enough, some anabolic agents may actually increase bone strength by preferentially acting on periosteal remodelling without affecting measured BMD.

Geometric measurements present an exciting new development in bone densitometry

Whether bone geometric parameters will improve fracture risk stratification remains to be seen. One large cohort study concluded that hip structure variables were strong predictors of fracture, but not significantly greater than BMD alone. Several practical issues prevent widespread clinical use of structural analysis at present:

- The specialized research software required is not widely available.
- Considerable reference data will be needed in order to adjust for the effects of age, gender and ethnicity.
- The absolute differences in the size variables are very small and, to date, the association with fracture risk has required large data sets. It is unclear whether these measurements are sufficiently precise for routine clinical use and follow-up.

In summary, this is an important area of research, but no recommendations can be made yet for clinical purposes. I look forward to the day when we can move beyond simple BMD measurements and integrate a variety of clinical and other geometric measurements into fracture risk assessment. 

References are available upon request from: mackinnon@parkpub.com



Osteoporosis
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La Société
de l'Ostéoporose
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about the OSC

The Osteoporosis Society of Canada (OSC) is a national, not-for-profit organization dedicated to educating, empowering and supporting individuals and communities in the prevention and treatment of osteoporosis. The OSC, guided by its Scientific Advisory Council (SAC) made up of osteoporosis experts from across the country, works with healthcare professionals to make the latest prevention, diagnostic and treatment options available to Canadians. The OSC has developed the *2002 Clinical Practice Guidelines* — accessible at www.osteoporosis.ca — for use by physicians in their daily practice.

ONLINE RESOURCES

The Society's website provides useful clinical and treatment information for physicians and patients.

For health professionals

- Quick reference guide, to assist doctors to determine risk levels, means of assessment and treatment options — downloadable to PC or Palm
- *Osteoporosis Update* online — archived since Winter 2003
- Link to, and highlights of, the Society's *2002 Clinical Practice Guidelines* as published in *CMAJ*
- Calendar of events for healthcare professionals... and more

For patients

- Information on diagnosis, drug treatments, physical activity and nutrition
- Calcium Calculator to help estimate daily dietary calcium intake
- Calcium-rich recipes
- Contacts for local support groups... and much more

Visit the website at www.osteoporosis.ca today. Encourage your patients to get reliable osteoporosis information online or by calling **1-800-463-6842**.

CARE GAPS IN OSTEOPOROSIS

The Osteoporosis Society is supporting an upcoming symposium for family physicians, chaired by Dr. Brent Kvern, as part of the College of Family Physicians of Canada (CFPC) Family Medicine Forum

Thursday November 25, 2004 • Sheraton Centre, Toronto, ON • 10:30 a.m.–12:30 p.m.

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For more information or to register online, please visit the CFPC website at www.cfpc.ca