BEST PRACTICE

17

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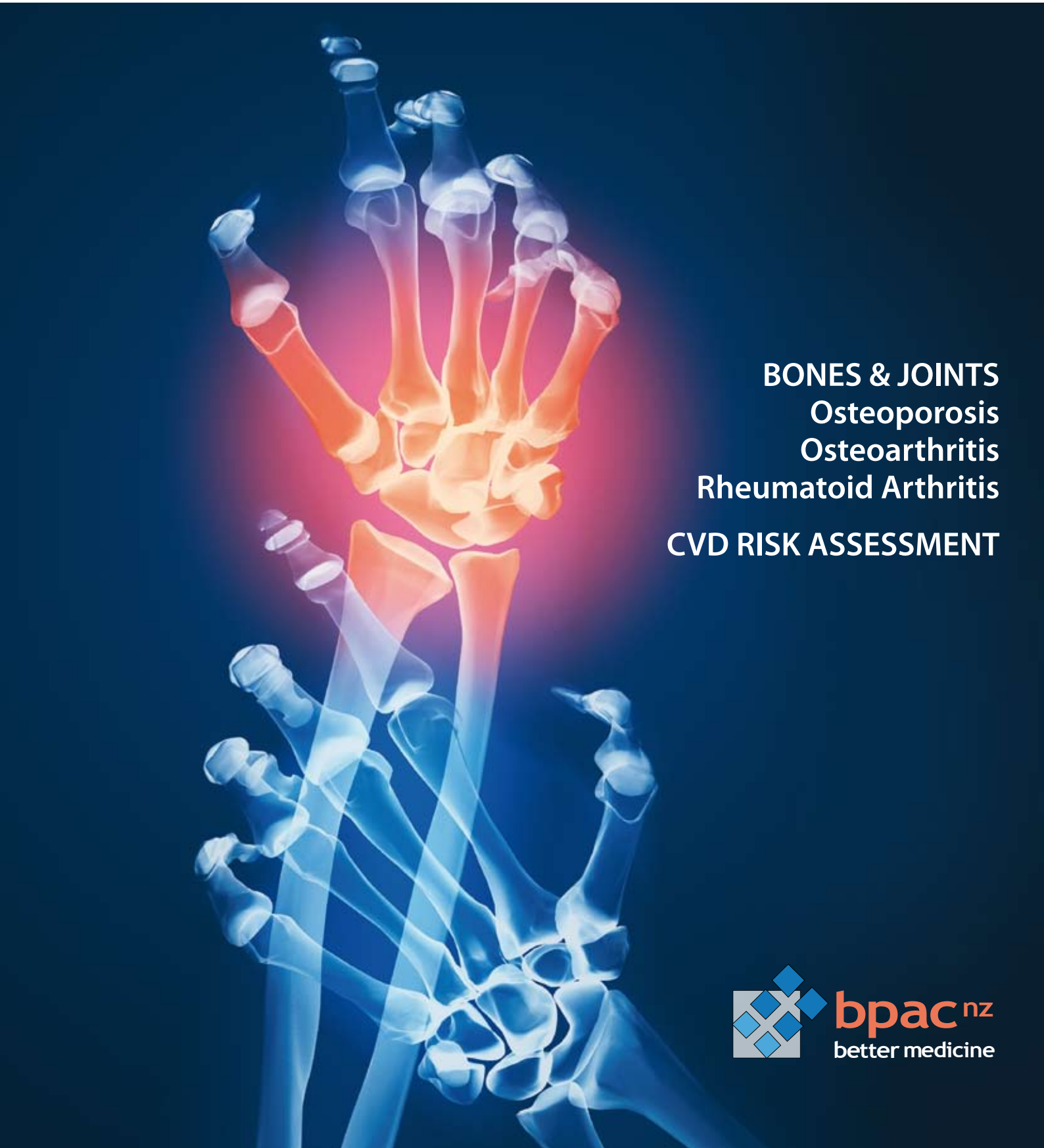

BONES & JOINTS
Osteoporosis
Osteoarthritis
Rheumatoid Arthritis

CVD RISK ASSESSMENT

bpac nz
better medicine

We would like to acknowledge the following people for their guidance and expertise in developing this edition:

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INTRODUCTION

WELCOME TO BPJ 17. We are now into our third year of publication and it has been satisfying seeing the BPJ continue to evolve guided by your feedback and support.

In response to recent feedback we have made some changes to this issue of the BPJ, expanding the range of content to incorporate more bpac material into the one publication.

What won't change is our focus on providing practical advice and concise clinical guidance, relevant to general practice.

Each year thousands of bpac quizzes and clinical audits are completed. The results of these provide interesting insights and are a great source of learning. From now on we will be publishing summaries of the results in the journal, accompanied by expert commentary.

The PHO performance programme is now well established and we want to play our part by promoting practical strategies to support you in achieving the goals of the programme.

In this issue we begin by looking at the new cardiovascular disease indicators, focusing in particular on GPs experiences with cardiovascular risk assessments, advice on effective strategies for communicating risk and SMART ways to engage patients in managing their risk.

One way of ensuring the BPJ remains relevant to primary care is by responding to your suggestions for topics. This issue includes a focus on "Bones and Joints" looking at the prevention of osteoporosis, management of osteoarthritis and the use of disease modifying drugs in rheumatoid arthritis.

We hope you'll continue to find the BPJ interesting and useful and we encourage you to keep on providing feedback, especially around the topics you'd like to see us cover in future editions.

Regards
The bpac^{nz} Team

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Cardiovascular risk screening: Targeting individuals or populations?

POPULATION SCREENING for identifying individuals at high risk of cardiovascular disease (CVD) is a key objective of public health policy. However some health professionals are questioning the benefits of this approach.

The issue of screening was highlighted in a recent "Head to Head" debate in the British Medical Journal. The question was "**Will screening individuals at high risk of cardiovascular events deliver large benefits?**"

Professor Rod Jackson (University of Auckland) and colleagues said "Yes", arguing that targeting high risk individuals is the most effective strategy. Professor Simon Capewell (University of Liverpool) answered "No", arguing that greater gains are achieved by population wide strategies.

In New Zealand, we use a combination of both methods to reduce CVD risk. However is this the right approach? Should we be targeting high risk people, high risk behaviour or both? The following article presents a summary of the two view points.

THE ARGUMENT – FOR[1]

Rod Jackson says interventions should be aimed at those at the greatest risk, arguing that this is both cost effective and maintains the long-term health of people better than population based interventions, such as reducing salt intake and managing obesity. He claims it is better to target people than risk factors.

Rod Jackson points out that approximately a third to a half of all cardiac events occur in people with a previous CVD event—approximately 6% of the population. By treating these people with aspirin, statins and antihypertensives (triple therapy), the number of events could be reduced by at least two-thirds. Even if only half of this group was adherent, this could achieve a 10% reduction in events over ten years.

On the other hand, to achieve a similar reduction with population based interventions the rest of the population would need to lower their personal risk by approximately 20%. Rod Jackson considers that this would be a "huge challenge now that much of the low hanging fruit receptive to population-wide strategies have been picked."

Identifying the small group of high risk people with a previous CVD event is relatively easy and they are usually

motivated to make changes. In addition Rod Jackson argues that there are still gains to be made in this group as most patients with established CVD are not receiving triple therapy.

Accurately identifying people at risk of CVD is becoming easier as new equations to estimate risk are being developed. These include factors such as social deprivation and ethnicity and provide the opportunity to more precisely target treatment. But, as risk thresholds for treatment are lowered, more people will be identified who may benefit from therapy. This would result in an increased workload and cost for primary care, and Rod Jackson says that simplified drug regimens such as the "single daily combination pill" may be the solution.

Rod Jackson believes that the key for preventing CVD is well targeted treatment with safe, inexpensive and effective drugs for patients at high risk.

THE ARGUMENT – AGAINST[2]

Simon Capewell says that the "high risk" approach to preventing CVD has been disappointing in its effectiveness. He says it is also associated with high cost, medicalisation and increasing inequalities. He argues that whole population approaches are more effective such as those introduced in Denmark (banning trans-fatty acids), Finland (halving dietary salt) and UK, Ireland and Italy (promoting smoke-free public spaces).

Simon Capewell argues that in reality interventions targeted at high risk individuals have low effectiveness due to issues such as accurate identification of these patients, uptake of screening and adherence to treatment.

CVD risk scoring systems have been shown to be inaccurate in estimating an individual patient's risk. Screening programmes require considerable effort, have high drop out rates and often those who experience the highest rates of disease and the most deprivation are not well engaged by these programmes. Studies show that long term adherence to both statins and antihypertensives is often less than 50%.

Furthermore, effectiveness is limited by the fact that medication does not remove the underlying pathology, it "merely puts a sticking plaster over the problem". This is the idea of residual risk. Interventions can never completely eliminate risk. At best, risk reductions are around 40% therefore significant risk remains.

Simon Capewell raises the issue of medicalisation associated with the high risk approach. "The implicit message for patients is that the doctor can fix it. This takes responsibility away from the individual and may encourage further risk taking behaviour." Studies show quality of life often decreases after starting treatment for CVD risk factors. Given this, he believes that most people would rather opt for behavioural change than lifelong medication.

Increased financial cost is a factor of the high risk approach as more people are prescribed medication due to reduced thresholds for intervention. Social costs are also increased, as targeting those at risk tends to benefit the affluent and educated, therefore contributing to increased disparity.

The answer, says Simon Capewell is small reductions in key modifiable cardiovascular risk factors which result in large reductions in cardiovascular events and deaths. These are best achieved through cheap policy interventions aimed at reducing risk factors across whole populations.

Finally, Simon Capewell argues that the greatest danger arising from the high risk approach, is that it is "misleading professionals, planners and politicians into thinking they can tick the mission accomplished box for preventing cardiovascular disease".

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Prevention of Osteoporosis

Key reviewers:

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Key concepts

For prevention of osteoporosis:

- Recommend adequate dietary intake of calcium and use supplements if necessary
- Advise on the role of vitamin D and consider sun exposure and the use of supplements if necessary
- Encourage regular weight bearing exercise
- Encourage smoking cessation
- Bisphosphonates can decrease the incidence of fracture in women with established osteoporosis

Osteoporosis is not just a result of ageing

Osteoporosis develops from a combination of the following factors:

- Age
- Genetics
- Lifestyle
- Hormones
- Medications
- Medical conditions

Age

Peak bone mass is achieved by around age 30–35 years and from then on starts to decline. The higher the peak bone mass achieved, the lower the impact of subsequent bone loss.

Genetics

Genes play a role in determining peak bone mass.

A person with a history of a hip fracture in a parent is at increased risk of osteoporosis. In addition, a study of hip fracture in New Zealand showed approximately 30% higher prevalence in people of European origin than for Māori, Pacific or Asian peoples. However, these may be due to differences in life expectancy and lifestyle factors relating to diet and body mass, as well as genetics.[2]

The relationship between body mass and osteoporosis is complex. Inherited muscular body mass appears to be protective whereas obesity may be a risk factor.[3]

Lifestyle

Lifestyle factors can have a direct effect on bone strength or alter calcium absorption. Factors associated with an increased risk of osteoporosis include, vitamin D deficiency, excess vitamin A, low calcium intake, smoking (both active and passive), high alcohol intake, lack of physical activity, immobilisation (paralysis, ill health), low body weight, exercise induced amenorrhoea and falls.

Hormones

Both men and women can develop osteoporosis but women are more at risk as their bones are smaller and there is an accelerated loss of bone density at menopause due to decreasing oestrogen. Early or surgical menopause or amenorrhoea increases the risk.

Any condition in men causing a decrease in testosterone can increase the risk.

Medications

Medications that can increase the risk of osteoporosis include:

- Steroids (>5mg/day for more than three months)
- Lithium
- Anticonvulsants
- Cancer chemotherapy drugs
- Depo-medroxyprogesterone (see BPJ 12)
- Proton pump inhibitors (see ETC, page 55)

Medical conditions

Many medical conditions are associated with osteoporosis either as a risk factor or consequence of (see Box 1).[1]

Prevention of osteoporosis

Prevention of osteoporosis in the whole population focuses on nutritional and lifestyle changes. The goals include:

- Acquiring maximal peak skeletal bone mass
- Maintaining this bone mass for as long as possible

Increasing awareness of the modifiable risk factors for osteoporosis through patient education is an important primary care role. A recent study of attitudes and knowledge about osteoporosis in a group of well-educated New Zealand women did not show a high level of knowledge. Although most demonstrated high levels of health motivation and most considered osteoporosis to be a serious disease, the women had low perceptions of personal susceptibility.[4]

Box 1: Examples of medical conditions associated with osteoporosis[1]

Endocrine e.g. diabetes mellitus, Cushing's syndrome, hyperparathyroidism, thyrotoxicosis

Gastrointestinal e.g. coeliac disease, inflammatory bowel disease, gastric bypass, pancreatic disease, malabsorption

Genetic e.g. cystic fibrosis, haemochromatosis, Marfan syndrome

Hypogonadal states e.g. premature ovarian failure

Haematologic e.g. multiple myeloma, haemophilia, leukaemia and lymphomas, thalassaemia

Rheumatic/autoimmune e.g rheumatoid arthritis, ankylosing spondylitis, lupus

Other e.g. alcoholism, emphysema, end stage renal disease, prior fracture, anorexia nervosa, bulimia

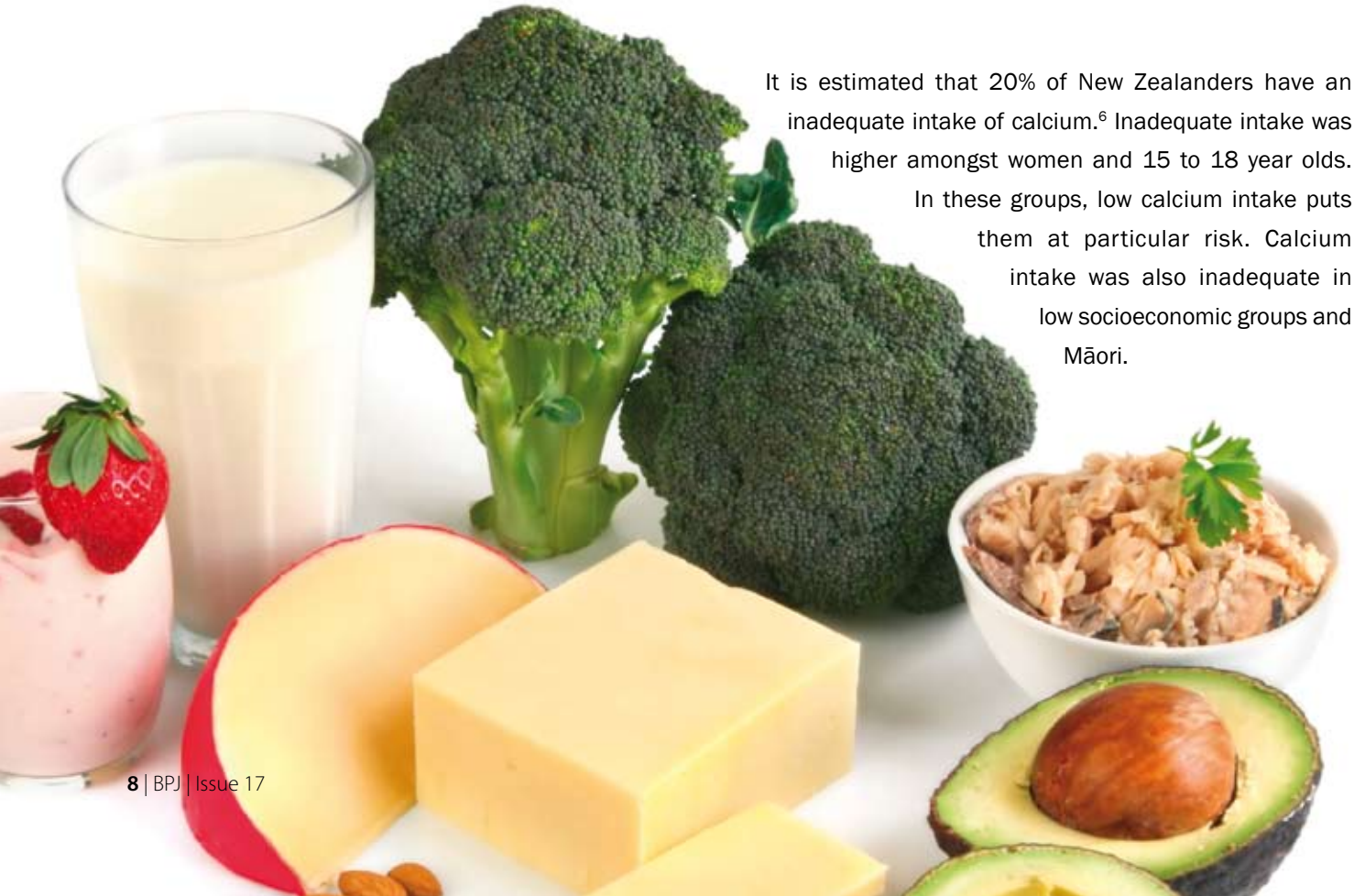
Adequate calcium intake

Adequate calcium intake is necessary for the acquisition of peak bone mass by the age of 35 and its subsequent maintenance. When exogenous supply is inadequate, bone tissue is resorbed to maintain serum calcium at a constant level. Calcium needs vary throughout life and between genders (see Table 1).

Table 1: New Zealand recommended daily intake of calcium (mg/day)[5]

Children age 1–3	500
Children age 4–8	700
Children age 9–11	1000
Adolescents age 12–18	1300
Men 19–70	1000
Men 70+	1300
Women 19–54	1000
Pregnancy and Lactation	1000–1300
Post menopausal	1300

It is estimated that 20% of New Zealanders have an inadequate intake of calcium.[6] Inadequate intake was higher amongst women and 15 to 18 year olds. In these groups, low calcium intake puts them at particular risk. Calcium intake was also inadequate in low socioeconomic groups and Māori.



Practical food suggestions for maximising dietary calcium

Calcium is more easily absorbed from dairy products so these are the best source. In addition, some brands of orange juice, bread, cereals and soy based drinks are calcium-fortified. See Box 2 for examples of calcium rich foods.

Some ideas for adding calcium rich food to the diet could include:

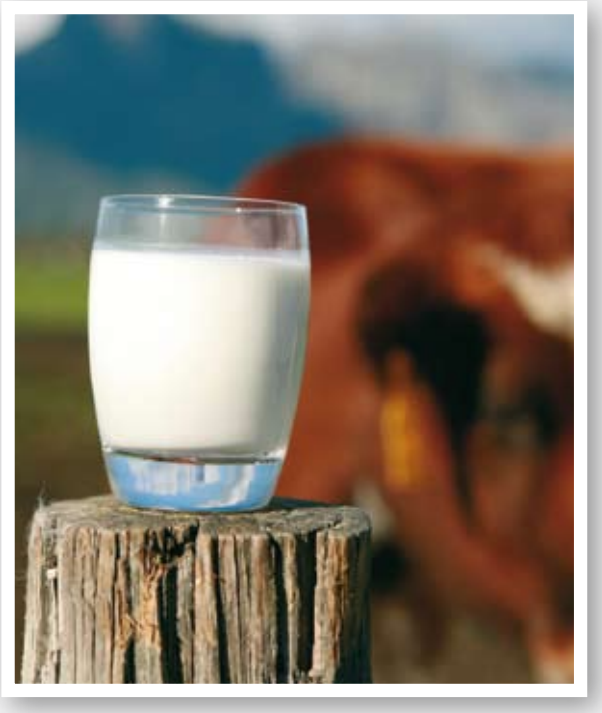
- Choosing low fat milk instead of carbonated soft drinks
- Sprinkling grated low fat cheese on salad, soup or pasta
- Making fruit smoothies with low fat yoghurt
- Making toasties with wholemeal bread, sardines and low fat cheese
- Serving raw fruits and vegetables with a low fat yoghurt based dip
- Making a vegetable stir-fry and including diced tofu or low fat cheese
- Making desserts such as instant puddings or custard with low fat milk
- Serving fruit for dessert with low fat yoghurt or ice-cream
- Adding skim milk powder to mashed vegetables, baking, puddings and soups

For children who do not like drinking milk suggest:

- Grilled cheese on wholemeal toast fingers with marmite or vegemite
- Calcium fortified cereal with milk
- Baked beans sprinkled with grated cheese
- Cheese sauce over vegetables
- Mashed salmon and potato fish cakes

Box 2: Examples of calcium rich foods

Food type	Approximate calcium content (mg)
250mL milk	300
125g pottle yoghurt	200
40g (2–3 slices) cheddar cheese (N.B. softer cheeses have less calcium)	300
½ cup tofu	300
1 tin sardines (with bones)	300
100g canned pink salmon (with bones)	280
1 cup mussels	300
1 medium bowl fortified muesli	200
1 cup cooked broccoli	80
½ cup raw whole almonds	200
5 dried figs	190
1 cup baked beans	100



Adequate Vitamin D

For more information see Best Tests – Vitamin D Testing in Primary Care, January 2007, www.bpac.org.nz keyword: vitaminD.

Vitamin D, which is produced in the skin, is essential for the acquisition and maintenance of bone mass.

Adequate exposure to sunlight is required to maintain vitamin D levels. This means about 15–20 minutes of sun exposure to the face and arms every day, avoiding sun exposure around midday in the summer. People with dark skin require approximately three to four times more exposure to gain the same benefit.

Although vitamin D is contained in some foods in small amounts such as oily fish (e.g. salmon, sardines, herring), adequate intake is not usually attained through diet alone.

Vitamin D deficiency is more prevalent in the following groups:

- Older people in residential care
- Older people admitted to hospital
- People with hip fracture
- People with dark skin
- People unable to obtain regular sun exposure

For these people, consider supplementation without testing. An appropriate dose is a single tablet of cholecalciferol 1.25mg monthly.

Regular exercise increases and maintains bone density

Regular weight bearing exercise should be recommended at all ages. This type of exercise can increase bone density and strength, particularly during childhood and adolescence. Exercise should be regular and ongoing as the beneficial effects on bone strength are lost when exercise is stopped.

Weight bearing exercise and muscle strengthening exercises may help prevent falls and fractures by improving agility, strength, co-ordination, posture and balance especially in older adults. Water exercise and cycling are regarded as non-weight bearing exercises but are still useful as muscle strength and fitness is maintained.

A person with established osteoporosis is at higher risk of fractures from high impact, jarring or twisting exercises such as running, jumping and aerobics.

Smoking is a significant risk factor for osteoporosis

There is some evidence that the significant risk of osteoporosis associated with smoking is a direct effect that is independent of confounding factors.[7] Smoking cessation is recommended.

Avoid excessive alcohol

Current evidence about the role of alcohol and bone density is conflicting.[8] Most publications report that an alcohol intake of three or more drinks per day is detrimental to bone density.[1] However, the exact mechanism of action remains unclear.

Fracture prevention in people with osteoporosis

The most common osteoporotic fracture sites are the spine, hip and wrist. They can have major consequences, for example hip fracture causes 10–20% excess mortality within one year and two and a half times increased risk of future fractures. In addition, after a hip fracture 20% of patients require long term residential care and only 40% fully regain their pre-fracture independence.[1]

The consequences of spinal fracture can also be significant with chronic back pain, kyphosis, loss of height and limitation of activities that require bending, reaching and lifting. Multiple compression fractures result in an increasing curvature of the spine and

compression of thoracic and abdominal organs. This may then cause shortness of breath, stress incontinence, and gastrointestinal symptoms such as anorexia, constipation, distension and abdominal pain.

The focus for fracture prevention for people with osteoporosis is:

- Preventing falls
- Detection of osteoporosis
- Pharmaceutical interventions

Preventing falls

Preventing falls prevents fractures. Lowering the risk of falls may include checks on vision, hearing, adverse effects from medications, safety at home and promotion of exercise programmes.

DEXA scanning

Bone densitometry (dual energy x-ray absorptiometry, DEXA) measures the bone mineral density usually at the hip and spine. Results may be given as a T score which is the result compared to "young normal" adults of the same sex or a Z score which is the result compared to that expected for the patient's age and sex. The T score is most predictive of future fracture.

Detection of osteoporosis

A clinical diagnosis of osteoporosis can be made if there is a low trauma fracture in an at-risk individual and may be suggested when an x-ray indicates low bone density. However the gold standard for diagnosis is bone densitometry (DEXA). Access to and funding of bone densitometry scanning varies throughout New Zealand.

Osteoporosis New Zealand Inc. recommends only measuring bone density when the result will impact on decision making.[9]

People who have had an osteoporotic fracture

A DEXA scan is not required for everyone with an osteoporotic fracture. Bisphosphonate treatment without the need for a prior DEXA scan can be considered for:

- Women over the age of 75 years who have had an osteoporotic fracture demonstrated on x-ray
- People who have had two or more demonstrated osteoporotic fractures
- People who have had systemic glucocorticoid steroid therapy (over 5mg per day prednisone equivalent for at least three months)

A DEXA scan is indicated for other people who have had an osteoporotic fracture.

People at high risk of osteoporosis

The contribution of individual risk factors towards the development of osteoporosis has not yet been quantified. Clinicians must make pragmatic decisions on who to refer for a DEXA scan based on major risk factors such as:[10]

- Age
- Female gender
- Low BMI
- Untreated premature menopause
- Family history of maternal hip fracture before the age of 75 years
- Conditions affecting bone metabolism (primarily inflammatory conditions, hyperthyroidism and prolonged immobility)
- Chronic steroid use

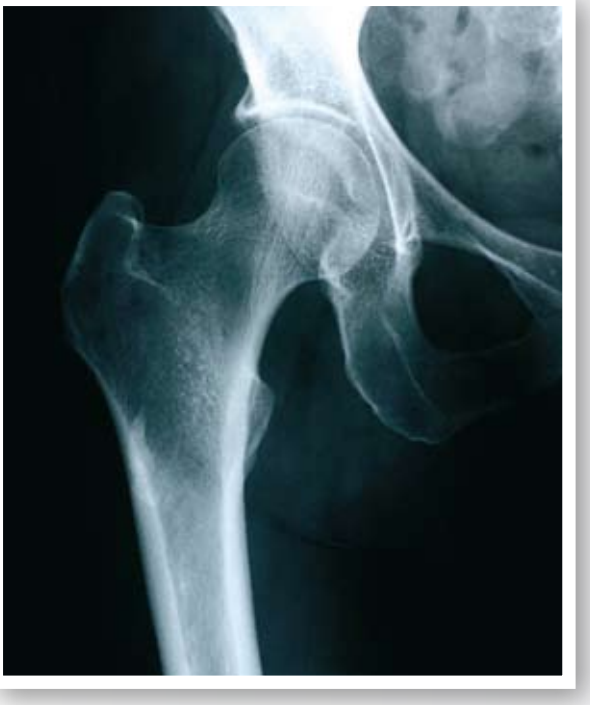
Criteria for alendronate

The current criteria for use of alendronate in osteoporosis are:

- History of one significant osteoporotic fracture demonstrated on x-ray and a documented T score ≤ –2.5
- History of one significant osteoporotic fracture demonstrated on x-ray, and either the patient is elderly, or DEXA scanning cannot be performed because of major logistical, technical or pathophysiological reasons
- History of two significant osteoporotic fractures demonstrated on x-ray
- Documented T score ≤ –3.0

The current criteria for use with glucocorticosteroid therapy are:

- Patient on systemic glucocorticosteroid therapy (≥ 5mg per day prednisone equivalent) and where they have already received or expected to receive therapy for at least three months and either a T score of ≤ –1.5 or history of one significant osteoporotic fracture demonstrated on x-ray



Pharmacological treatment

Calcium supplements may be required when dietary calcium intake is inadequate. When used alone they are not recommended as adequate therapy for treating osteoporosis or reducing fracture risk. There have been concerns about increased cardiovascular risk associated with calcium supplementation (see BPJ 10). Calcium supplements should be used with caution in people aged over 70 years and in those with coronary heart disease. Dose may need to be reduced in those with a reasonable dietary intake of calcium.

Vitamin D supplements may be required in those who are likely to be deficient (see page 10). An appropriate dose is a single tablet of cholecalciferol 1.25mg monthly.

Activated vitamin D metabolites e.g. calcitrol, used as monotherapy, are thought to be inadequate and are no longer widely used.

Bisphosphonates. There is evidence that bisphosphonates reduce the risk of vertebral and hip fractures by 30 to 70% in women with established osteoporosis.[11] It appears that the effect of etidronate on non-vertebral fractures is less pronounced than alendronate. Etidronate is fully subsidised and alendronate is subsidised on special authority (see criteria on left).

Adequate vitamin D and calcium intake are considered prerequisites for bisphosphonate therapy.[12] The combination therapy Fosamax Plus contains a dose of vitamin D which is inadequate for treating vitamin D deficiency or preventing deficiency in high risk groups.[13]

Adverse effects of bisphosphonates may include gastrointestinal irritation and visual disturbances. There have been very rare reports of osteonecrosis of the jaw. However current evidence suggests that the risk of osteonecrosis, for people taking bisphosphonates for osteoporosis, is the same as that for the general population.[14]

Hormone Replacement Therapy is known to decrease the risk of fractures in post menopausal women but it is no longer regarded as first line for prevention or treatment of osteoporosis due to increased risk of cardiovascular events, thromboembolic events and breast cancer.

Further reading:

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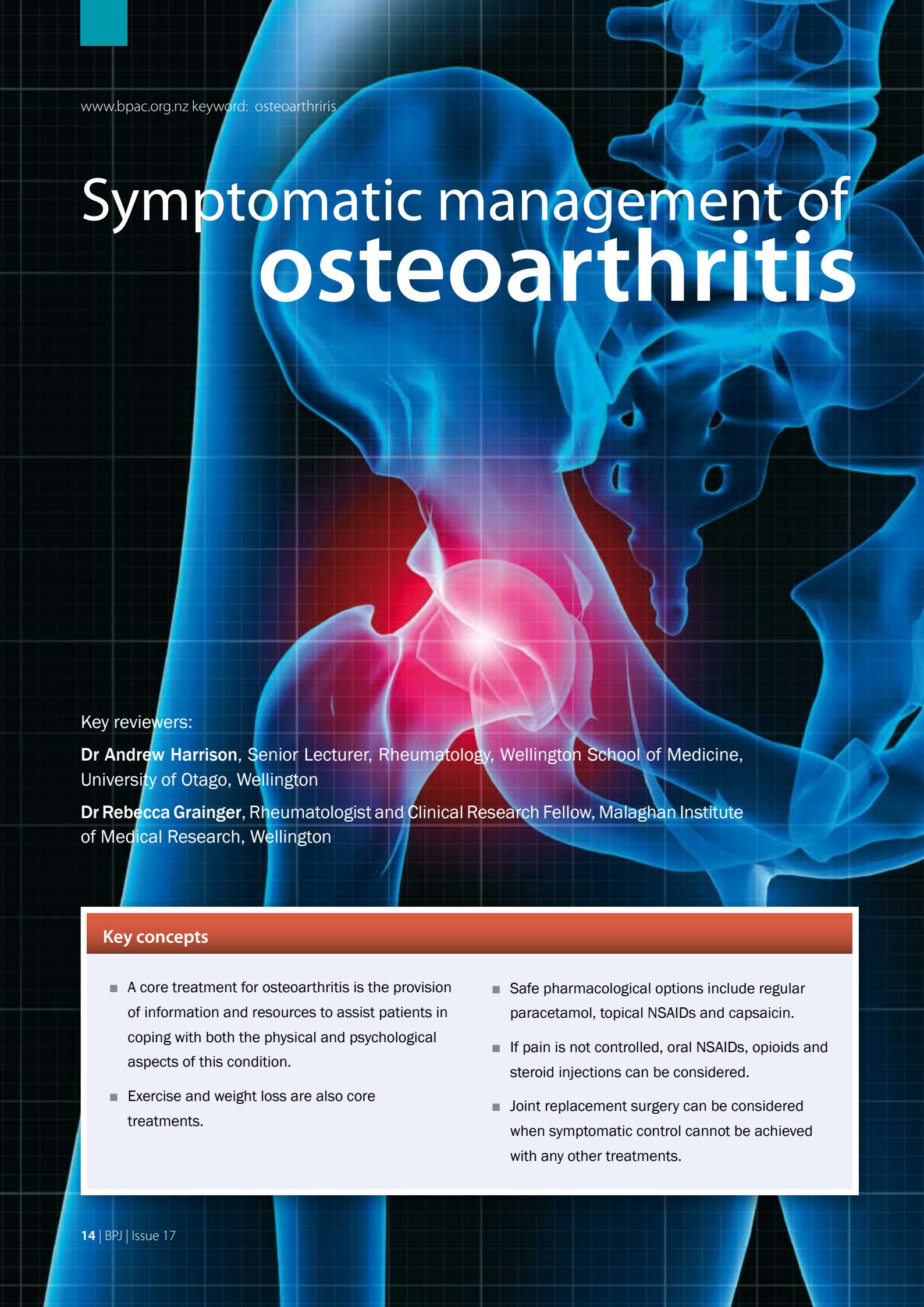
Symptomatic management of osteoarthritis



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Key concepts

- A core treatment for osteoarthritis is the provision of information and resources to assist patients in coping with both the physical and psychological aspects of this condition.
- Exercise and weight loss are also core treatments.
- Safe pharmacological options include regular paracetamol, topical NSAIDs and capsaicin.
- If pain is not controlled, oral NSAIDs, opioids and steroid injections can be considered.
- Joint replacement surgery can be considered when symptomatic control cannot be achieved with any other treatments.

Osteoarthritis is the most common form of arthritis and a leading cause of pain and disability around the world. It affects approximately 50% of people aged over 60 years and almost all people aged over 80 years. However osteoarthritis is not just caused by ageing. Factors which may lead to the development of osteoarthritis include:[1]

- Genetic factors
- Age
- Joint damage by injury
- Joint damage by chronic obesity

The joints most commonly affected are the knee, hip, spine and hand.

Treating osteoarthritis

There is currently no treatment that can reverse joint damage due to osteoarthritis, but early management can slow disease progress, and allow patients to gain control of symptoms.

Symptomatic management should be guided by the severity of the disease, the joints affected, specific symptoms, co-morbidities and activity level. A team approach to management including occupational therapist, physiotherapist and other health workers is often beneficial. Referral to a rheumatology or rehabilitation unit may be appropriate.

First tier (core treatment for every person with osteoarthritis):

Knowledge/education

Exercise

Weight loss (if overweight)

Second tier ("safe" pharmacological options if further treatment required):

Paracetamol

Topical NSAIDs, capsaicin cream

Third tier (if pain is still not controlled):

Oral NSAIDs (including COX-2 inhibitors)

Opioids

Intra-articular corticosteroid injections

Surgery

Joint arthroplasty

Other treatments

Local heat and cold

Assistive devices (e.g. walking sticks, tap turners)

Supports/braces

Shock-absorbing insoles

Transcutaneous electrical nerve stimulation (TENS)

Manual therapy (manipulation and stretching)[1]

Core treatments for osteoarthritis

Providing accurate information helps patients cope with the ongoing nature of osteoarthritis and develop effective self-management strategies for both its physical and psychological aspects. This includes advice on lifestyle, exercise, activity and weight loss.[2]

Exercise is a core treatment for anyone with osteoarthritis, irrespective of age, co-morbidity, pain severity or disability.[1]

Exercise can help to manage pain, keep joints mobile and increase the ability to perform day to day tasks. A fitness programme should be individually tailored and may require modification at times depending on symptoms, but should always include cardiovascular (aerobic), muscle strength (especially around the damaged joint), muscle endurance and flexibility components. Joint pain can limit the intensity of exercise, but should not be a deterrent. Referral to a physiotherapist for a tailored exercise programme is appropriate.

General practitioners and practice nurses play an important role in encouraging and motivating patients to participate in and maintain exercise programmes e.g. “green prescription”.

Regular exercise is beneficial for weight loss, which is also an important component of symptomatic management. Patients who are overweight should be encouraged to reduce and maintain their weight at recommended levels.

Shock-absorbing footwear, joint supports and mobility aids, heat packs, warm baths or application of ice packs can all provide symptomatic relief.

Safe pharmacological options

Paracetamol

Regular paracetamol at a dose of up to 4g per day is effective as initial oral analgesia for treating mild to moderate pain associated with osteoarthritis.[2] If pain is successfully managed, paracetamol can be maintained long-term.[2]

Topical NSAIDs

Topical NSAIDs are generally less effective than oral NSAIDs but they are considered to be safer and serious adverse effects are unlikely. Local reactions such as

itching or burning may occur.[2] The "placebo effect" is said to play a role in the clinical effect of topical NSAIDs and they may only be effective in the first few weeks of treatment.[2] Topical NSAIDs can be considered, but are not a key component of treatment for osteoarthritis.

Capsaicin cream

Topical capsaicin cream (0.025%) contains a chilli pepper extract and may cause burning pain at the site of application but is an effective analgesic. Other than the intended burning effect, capsaicin is not associated with other adverse effects.[2]

Capsaicin cream is applied by squeezing a bead of cream onto the finger and rubbing over each affected joint, four times per day. It should not be applied after a hot bath or shower or to broken skin. Hands should be washed after application to avoid inadvertent transfer to the eyes. Treatment should be continued for three months before assessing clinical effect.

Note that topical NSAIDs and capsaicin are not subsidised for the treatment of osteoarthritis in New Zealand.

Further treatment approaches if pain is still uncontrolled

NSAIDs

There is evidence that NSAIDs are superior to paracetamol for pain relief in patients with osteoarthritis,[3] but they are also associated with more adverse effects. The major concern is serious gastrointestinal, renal and cardiovascular complications, with risk increasing with age, concurrent use of other medications and duration of therapy.[2] NSAIDs help relieve pain, swelling and stiffness but they do not alter the progression of osteoarthritis.

Oral NSAIDs should only be considered when paracetamol or topical treatments are ineffective for pain relief. NSAIDs should be used at the lowest effective dose for the shortest possible time. Do not exceed maximum daily doses (Table 1). Long-term use of NSAIDs is not routinely recommended,[2] however individual patient factors can be taken into account, such as risk of adverse events and the effect of NSAID treatment on functional ability. Paracetamol can be continued throughout NSAID treatment,[1] however topical NSAIDs should be discontinued.

Cyclo-oxygenase-2 (COX-2) selective drugs are not recommended for routine use in osteoarthritis, except where the patient is at high risk of developing a serious GI adverse effect from other standard NSAIDs, or has GI intolerance of standard NSAIDs in spite of the use of a gastroprotective agent. COX-2 drugs are not subsidised in New Zealand.

Table 1: Recommended total daily doses of NSAIDs for osteoarthritis[4]

NSAID	Total dose/day
Ibuprofen	1200 – 1600 mg
Diclofenac sodium	75 – 150 mg
Naproxen	500 – 1000 mg
Celecoxib	200 mg

Which NSAID?

Choice of NSAID should be based on the overall safety profile of the drug and the patient's individual risk factors. All NSAIDs have similar analgesic effect but individual patients may have a better response to one type over another. It may be reasonable to trial ibuprofen, naproxen and diclofenac depending on other risk factors. NSAIDs (including COX-2) should not be combined.[2]

All NSAIDs should be used with caution in patients with cardiovascular risk factors (e.g. hypertension, hyperlipidaemia, diabetes, smoking, peripheral arterial disease).[2] COX-2 drugs are contraindicated in patients with ischaemic heart disease or stroke.[2]

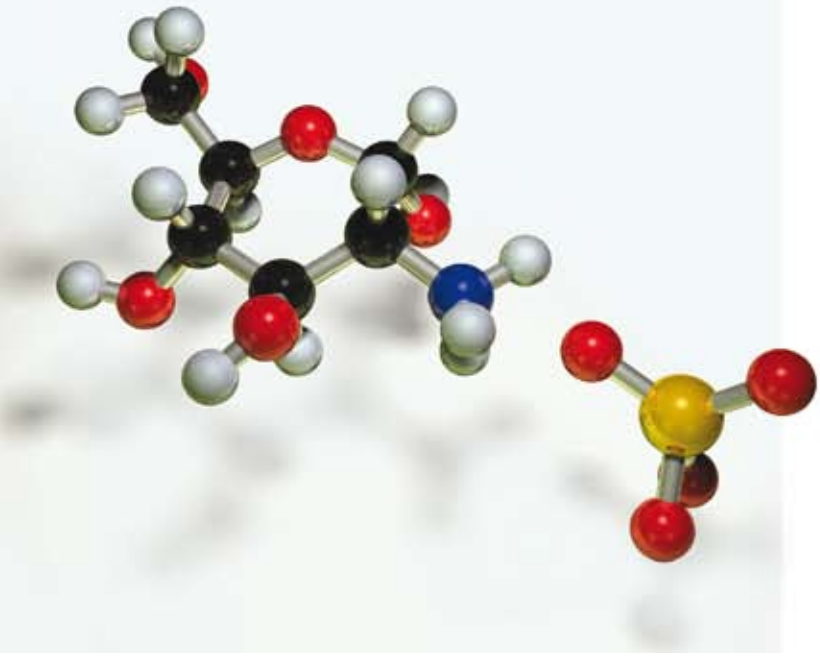
Naproxen 500mg, twice per day appears to be the safest choice of NSAID for patients with cardiovascular risk factors.[5]

The role of glucosamine in osteoarthritis

A 2001 Cochrane systematic review reported that there was some evidence that glucosamine sulphate 1500mg/day provides pain relief and improved mobility in people with osteoarthritis. However an updated review that included newer and higher quality studies has concluded that glucosamine is not as effective in reducing pain and improving mobility as originally thought.[7]

Most guidelines do not recommend nutraceuticals for the treatment of osteoarthritis.[1] If a patient wishes to purchase these supplements, ensure they select an adequate dose and if symptomatic benefit is not apparent within three months, treatment should be discontinued.[2] Glucosamine is generally well tolerated and not associated with any significant adverse effects.

See BPJ 11 (February 2008) for more information on glucosamine and other alternative treatments for osteoarthritis.



All NSAIDs can increase blood pressure, especially in people with hypertension, and cause fluid retention and oedema. In rare cases, congestive heart failure and renal dysfunction may occur.[5] Monitoring of blood pressure and for signs of fluid retention should occur within two to four weeks of initiating NSAID treatment.[5]

In patients with increased GI risk, prescribe a non-selective NSAID with a 20mg proton pump inhibitor (PPI) or a COX-2 selective agent.[2] The GI protection associated with the use of a COX-2 drug is mostly lost when low-dose aspirin is concurrently administered.[2] Aspirin also does not offset the increased cardiovascular risk associated with COX-2 drugs.

Opioids may be considered when other oral treatment is unsuccessful

Weak opioids such as codeine can be considered for relieving pain where other pharmacological agents have been ineffective or are contraindicated. A recommended dose of codeine is one to two 30mg tablets, every four to six hours as required, to a maximum of 240 mg (60mg codeine is equivalent to 6mg morphine).[4]

Stronger opioids should only be used for severe pain in exceptional circumstances. Adverse effects including sedation, risk of falls and constipation are common and are of particular concern in older people.

Tramadol has no advantages over other opioids but is increasingly used for pain management (50mg tramadol is equivalent to 10mg morphine). A systematic review concluded that while tramadol is effective in relieving pain and improving function in people with osteoarthritis (compared to placebo) the benefits are small. Adverse effects may limit its use and include nausea, confusion and interaction with SSRIs.[6]

Intra-articular injections may be useful in severe pain

Intra-articular injections with corticosteroids can be considered when patients have moderate to severe pain

What doesn't work for treating osteoarthritis?

There is no evidence of clinical effectiveness for the following common treatments for osteoarthritis:

- Avoiding or eating particular foods
- Multivitamin and mineral supplements
- Copper bracelets/ jewellery
- Cod liver oil (excessive consumption can cause vitamin D toxicity)
- Acupuncture (not enough consistent evidence of clinical effectiveness)[1]
- Rubifacients such as eucalyptus oil, salicylates and camphor[1]
- Magnets and magnetic underlays

that does not respond adequately to oral treatment and when there are physical signs of local inflammation or joint effusion.[1, 2]

Correct placement of the injection is crucial to maximise benefit and reduce the risk of adverse effects such as fat necrosis and peri-articular tissue atrophy. It is recommended that injections are administered no more than four times a year to the same joint without specialist review.[2] Duration of symptomatic benefit varies between individuals.

Hyaluronic acid is a glycosaminoglycan which is a constituent of synovial fluid. Hyaluranate preparations are available in New Zealand but are not subsidised and are very costly. UK guidelines do not recommend hyaluronate injections due to their high cost in relation to their benefit.[1] However, intra-articular hyaluronate injections can decrease pain and compared to corticosteroids, have a prolonged duration of symptomatic benefit (up to six months) but a delayed onset of action.[2]

Prevalence of arthritis in New Zealand adults

A Portrait of Health—key results of the 2006/07 New Zealand Health Survey:

- One in seven adults (14.8%) have been told by a doctor they have arthritis
- The age standardised prevalence of arthritis was higher in women (13.2%) than in men (10.9%)
- Osteoarthritis was the most common type of arthritis (8.4%) followed by rheumatoid arthritis (3.5%) and then gout (1.3%)

The prevalence of arthritis increased rapidly as age increased, especially in women. More than half of women aged 75 years and over had been diagnosed with arthritis.

Arthritis in New Zealand adults, by ethnic group (unadjusted)

Ethnic group	Prevalence (95% CI)
European/other	16.1% (15.4– 16.8)
Māori	11.1% (9.8 – 12.4)
Pacific	7.9% (6.1 – 9.8)
Asian	6.2% (5.1 – 7.2)

After adjusting for age, Māori men had an increased prevalence of arthritis compared to men in the total population. Pacific women and Asian men and women had a significantly lower prevalence of arthritis than men and women in the total population.

Gout is a major cause of arthritis in Māori and Pacific peoples

see BPJ 8 and BPJ 13 for more information on the treatment of gout.

Surgical options

Patients who cannot obtain adequate pain relief and functional improvement from core treatments and are requiring strong opioids should be considered for joint replacement surgery.[2] Patient specific factors such as age, gender, smoking, obesity and comorbidities should not be barriers to referral for surgery.[1]

Osteotomy and joint preserving surgical procedures can be considered in younger adults, to delay the need for joint replacement.[2]

Joint fusion may be used as first line surgical management of joints such as first metatarsophalangeal joints, subtalar and radiocarpal joints. Joint fusion can also be considered when joint replacement has failed.[2]

Joint lavage and arthroscopic debridement (removal of cartilage fragments and debris) are not always associated with a significant improvement in symptoms or mobility but may reduce locking.[2] This procedure is not routinely recommended.[1]

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Safe **Prescribing of Morphine** in Primary Care

Contributed by the Safe and Quality Use of Medicines Group

Common morphine prescribing errors can arise from incorrect strength, measurement or dose. A patient who has had a morphine overdose is likely to become drowsy, confused and lethargic, and require hospital admission.

Below are some recent examples of typical morphine related prescribing errors in primary care.

Inappropriate starting dose of morphine

A patient was prescribed slow-release morphine 60mg twice a day for arthritic pain as an initial dose. Prior to this the patient was using tramadol 50mg three times a day for analgesia. After taking four doses of the morphine the patient was confused, hallucinating and drowsy. The patient was admitted to hospital where he remained for six days after receiving naloxone.

A prescribing error compounded by the lack of a safety check during dispensing

A patient was given 50mg/5mL of oral morphine solution instead of 5mg/5mL. The prescription was copied from the acute discharge note. The GP failed to notice the error and the pharmacy didn't question that the dose was different from that previously dispensed. The patient noticed and did not take the increased dose.

What can you do to reduce the chance of an error for your patients?

- Check the dose of morphine prescribed for opioid naïve patients, they are liable to respiratory depression
- When converting from one opioid to another, or one dose form to another check the conversion factor – it is not always 1:1. (see BPJ 16)
- Check the dose you prescribe when patients are referred back to your care after hospital admission or following treatment by another prescriber
- Review the way your practice stores morphine – clearly differentiate between high and low strength injections
- Always prescribe the oral solution as milligram (mg) of morphine not millilitres (mL) of solution
- When prescribing extended release products add the brand name so that it is clear which product is intended e.g. morphine sustained release tablets (m-Eslon), you may want to put the brand in the "sig" line
- Supplies of naloxone 400 micrograms in 1mL should be in stored in an easily accessible location, including GP bags and bags held by out-of-hours providers
- Adjust the starting dose of morphine for patients with renal impairment

Rheumatoid Arthritis – monitoring of DMARDs

Key reviewers:

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Key concepts

- Most people with rheumatoid arthritis require DMARD therapy to control symptoms and prevent joint damage. Treatment is initiated by a specialist as early as possible in the disease process.
- Treatment usually begins with methotrexate and then other DMARDs such as sulphasalazine, hydroxychloroquine or leflunomide are added when inflammation is not controlled.
- People prescribed DMARDs require close monitoring for adverse effects and drug interactions.

Rheumatoid arthritis is a chronic autoimmune disease characterised by inflammation of the synovial tissue in joints causing swelling, pain, stiffness and joint destruction.[1,2] Spontaneous remission is uncommon (<5%) and most affected individuals require long term disease modifying anti-rheumatic drug (DMARD) therapy to control symptoms and prevent joint damage.

A GPs role in the care of a patient with rheumatoid arthritis includes early referral for diagnosis and treatment, management of co-morbidities, co-ordination of secondary care and allied health care input and, in conjunction with the treating rheumatologist, monitoring of DMARD therapy.

This article covers important aspects of the care of patients taking DMARDs including monitoring requirements, adverse effects and drug interactions.

DMARDS are initiated in rheumatoid arthritis as soon as possible to prevent disease progression and reduce symptoms

The aim of treatment for rheumatoid arthritis is to achieve minimal joint inflammation (a therapeutic remission). Patients diagnosed with rheumatoid arthritis should start treatment with DMARDs as soon as possible, as early treatment has been shown to improve outcomes.[3] Joint damage occurs early in the course of rheumatoid arthritis and is largely irreversible.[1, 4] The degree of inflammation is closely related to joint damage therefore early control of inflammation should prevent joint damage. People with synovitis persisting for six weeks should be urgently referred to a rheumatologist.

Commonly used oral DMARDs include methotrexate, sulfasalazine, hydroxychloroquine, low-dose prednisone and a newer agent, leflunomide. Other less commonly used DMARDs include azathioprine, cyclosporin and sodium aurothiomalate (intramuscular gold). Biological DMARDS, tumour necrosis factor (TNF) inhibitors, are discussed on page 27.

Methotrexate is usually the first choice DMARD

Methotrexate is usually first line therapy in moderate to severe rheumatoid arthritis because it is effective, has a predictable adverse effect profile, is simple to administer and is inexpensive.[3] In individuals with highly active disease methotrexate may be commenced in combination with other DMARDs. Mild disease may be treated with sulfasalazine or hydroxychloroquine monotherapy.

If methotrexate is not tolerated an alternative DMARD may be used as monotherapy.[3]

Add another DMARD when inflammation is not controlled

If inflammation is not controlled, usually the next step is to add another DMARD such as sulfasalazine.

Triple therapy with methotrexate, sulfasalazine and hydroxychloroquine has been shown to be clinically effective and may also be used.[1, 2]

Other therapies include leflunomide, cyclosporin and azathioprine

Leflunomide is available on special authority* for the treatment of rheumatoid arthritis that has not adequately responded to methotrexate and sulfasalazine. Leflunomide can be used alone or in combination with methotrexate. In clinical trials leflunomide had similar efficacy to methotrexate but may be less well tolerated, with additional adverse effects such as hair loss and hypertension.[5]

Cyclosporin and azathioprine are usually reserved for patients who are unresponsive to other DMARDs, due to the increased risk of adverse effects. Azathioprine is less well tolerated than methotrexate and cyclosporin is associated with nephrotoxicity. They can control inflammation but there is less evidence of their effect for long term treatment of rheumatoid arthritis.[6]

* Special Authority requirement is to be removed on 1st November 2008

Biological DMARDs such as adalimumab and infliximab may be indicated if inflammation is uncontrolled by combination therapy (see page 27).[2]

Onset of action for DMARDs is between two to six months

The onset of action for DMARDs varies. Response is seen with methotrexate within one to two months while hydroxychloroquine can take up to six months for a response.[7]

DMARDs require regular monitoring for toxicity

DMARDs require regular laboratory monitoring for adverse effects. A management plan should be agreed between the patient, GP and Rheumatologist. It should state which doctor is primarily responsible for arranging and reviewing the laboratory investigations.

Recommended investigations for commonly used DMARDs are listed in Table 1 (see centre pages 29–32. Note: these pages can be pulled out for future reference). This includes recommended frequency of monitoring, what to look for and what to do about it. The frequency of monitoring varies between agents based on their likelihood of causing toxicity.

The table also includes clinical signs which may suggest toxicity. These clinical signs should be enquired about at consultations and patients should be advised to report them if they occur.

Set up a reminder on your PMS for monitoring.

Prescribing points for DMARDs

Methotrexate

- Dosing — administered once a week orally or by injection. Starting dose is 5–10 mg ONCE a week, increase by 2.5–10 mg every four to six weeks to a maximum of 25 mg ONCE a week.
- Folic acid — 5mg folic acid, once per week (but not on the same day as methotrexate), should always be prescribed.
- Potential adverse effects — nausea, mouth ulcers, hair loss, cytopaenias, elevated liver enzymes, rarely pneumonitis.[3]
- Interactions — methotrexate accumulates in the presence of renal impairment. Although this seldom has any clinical effect, patients with renal impairment, whether caused by NSAIDs, diuretics, dehydration or kidney disease should take lower doses and should be monitored carefully for any deterioration in renal function. Trimethoprim and cotrimoxazole interact with methotrexate and significantly increase the risk of marrow aplasia; the combination should be avoided.[3]
- Alcohol — patients should be advised to limit alcohol consumption to no more than one (females) or one and a half (males) standard drinks per day. This is roughly half of the level recommended for the general population. Liver function should be vigilantly monitored in patients who consume alcohol.
- Flu vaccination — annual influenza vaccination should be given but live vaccines should be avoided.[8]
- Contraception — methotrexate is a known teratogen. Effective contraception is required for women of child bearing potential taking methotrexate, or men taking methotrexate whose partner is of child bearing potential. Effective contraception needs to be continued for three months after stopping methotrexate.[9]

Sulfasalazine

- Dosing — starting dose is 500 mg orally daily, increase by 500 mg a week to a maximum of 40 mg/kg or 3g daily in divided doses.
- Potential adverse effects — nausea, abdominal pain, hair loss, cytopaenias, agranulocytosis, elevated liver enzymes, skin rashes.[3]
- Interactions — potentially reduces the absorption of digoxin, however the combination does not need to be avoided. Patients should be observed for signs

of under-digitalisation and digoxin levels should be measured if response is not adequate.[10]

- Pregnancy — can be used in pregnancy but doses should not exceed 2 g/day. Folic acid supplementation should be given during pregnancy and to women trying to conceive.[8] Causes reversible oligospermia.[6]
- Yellow discolouration — causes a yellow discolouration of urine and tears; warn patients it may stain undergarments and soft contact lenses.[9]

Hydroxychloroquine

- Dosing — starting dose is 400mg orally daily in divided doses for one to three months (maximum 6mg/kg/day), then a maintenance dose of 200–400mg daily.
- Potential adverse effects — blurred vision, skin rash, photosensitivity, very rarely maculopathy. Blue-black discolouration of skin may occur with long-term use.[3, 9]
- Photosensitivity and photophobia — may increase the skin's sensitivity to sunlight and also cause photophobia. Sunscreen is advised and patients should wear sunglasses in bright light.[9]

Leflunomide

- Dosing — loading dose (optional) 100 mg orally once daily for three days, then 10–20 mg once daily.
- Potential adverse effects — GI disturbance, weight loss, hair loss, rash or itch, mouth ulcers, headache, raised liver enzymes, cytopaenias, hypertension, rarely peripheral neuropathy and pneumonitis.[3] There is an increased susceptibility to infections which should be treated promptly. With many of the adverse effects, discussion with the specialist team may result in dose reduction or trial of symptomatic treatment.
- Interactions — concurrent use with other drugs that have the potential to cause liver or marrow toxicity may increase the risk of these toxicities occurring.[9] For example, the risk of pneumonitis is increased when leflunomide is combined with methotrexate.[12]
- Alcohol — patients should be advised to limit alcohol consumption[8] to no more than one (females) or one and a half (males) standard drinks per day. This is roughly half of the level recommended for the general population. Liver function should be vigilantly monitored in patients who consume alcohol.
- Flu vaccination — annual influenza vaccination is recommended but live vaccines should be avoided.[8]
- Contraception — leflunomide is very teratogenic. Effective contraception is required for women for two years and men for three months after stopping leflunomide. Blood concentrations of its active metabolite should be measured before conception occurs.[9]
- Washout — leflunomide has an extremely long half life and can be retained in the body for up to two years. If toxicity occurs or for any other reason e.g. desire to conceive, a wash out procedure with cholestyramine may be considered.

Additional prescribing points

- Azathioprine. Dosing - 1 mg/kg orally daily, increasing after four to six weeks to 2-3 mg/kg/day. Some rheumatology teams measure TPMT (Thiopurine Methyl Tranferase) at baseline. Low levels of this enzyme involved in the metabolism of azathioprine are an indication for reducing the dose. It is also possible to measure levels of azathioprine metabolites such as 6 Thioguanine (6 TGN). This may help guide treatment.
- Gold injections (sodium aurothiomalate). Dosing - 10 mg test dose (given in a clinic followed by 30 minutes observation), followed by weekly injections of 50 mg until significant response. Thereafter the interval between doses is increased in stages from 50 mg per week to 50 mg every four weeks. Nitritoid reactions (where the blood pressure falls) after injection of gold can occur. This should be checked after the first 10 mg dose of gold and thereafter. Concurrent use of ACE inhibitors may increase the incidence of nitritoid reactions. If a nitroid reaction occurs, gold injections should be withheld and discuss immediately with the Rheumatologist.

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Tumour necrosis factor inhibitors

What do they do?

Tumour necrosis factor (TNF) alpha, an inflammatory cytokine, is involved in the pathogenesis of rheumatoid arthritis.[1] The three TNF inhibitors available in New Zealand (adalimumab, etanercept and infliximab) target this cytokine and block its effect. These parenteral agents are given by either self administered subcutaneous injection (adalimumab once a fortnight, etanercept weekly) or hospital administered intravenous infusion (infliximab every two months after induction).

What is their place in therapy?

In New Zealand TNF inhibitors are mainly used in individuals with rheumatoid arthritis which remains active despite optimal disease modifying anti-rheumatic drugs (DMARDs). Cochrane reviews have concluded that TNF inhibitors significantly reduce disease activity in rheumatoid arthritis compared to placebo.[2, 3, 4]

All three drugs are registered for use in rheumatoid arthritis, but only adalimumab is funded on special authority for this indication (see box below). Etanercept is funded on special authority for juvenile idiopathic arthritis. Patients who fail to respond to one TNF inhibitor, or who discontinue its use because of adverse effects, may respond to a second TNF inhibitor.[3]

Adalimumab for rheumatoid arthritis – Pharmac criteria

Patients must have severe erosive rheumatoid arthritis that has not responded to a three month trial of each of the following; methotrexate, combination (triple) therapy, leflunomide or cyclosporin. They must also meet disease activity criteria (e.g. at least 20 joints affected). To continue to receive subsidy for adalimumab patients must also show a 50% decrease in active joint count and a clinically significant response after four months of treatment.

For full details of special authority criteria see: www.pharmac.govt.nz/2008/10/01/SA0812.pdf

Safety concerns

The most common adverse effect with TNF inhibitors is injection site reactions. Reactions can be treated with the local application of ice or corticosteroid cream unless complicated by infection.[5]

Other more serious safety concerns are:

- Reactivation of tuberculosis (TB) — most likely in the first 12 months of treatment therefore extra vigilance is required during this time. British guidelines suggest screening all patients for TB prior to commencing treatment with a TNF inhibitor. Patients who are found to have latent or active TB should be treated.
- Congestive heart failure — Infliximab has been associated with an increase in mortality and hospitalisation due to cardiac failure. TNF inhibitors should not be started in people with Grade 3 or 4 congestive heart failure and used with caution in Grade 1 and 2. All patients on TNF inhibitors should be monitored for signs and symptoms of cardiac failure.[6]
- Serious opportunistic infections — TNF inhibitors should not be initiated in the presence of serious infections and extreme caution should be used in patients with increased risk of infection, e.g., bronchiectasis, history of chronic leg ulcers and history of septic arthritis. Patients should be advised of the increased risk of infection. Therapy should be discontinued if a serious infection develops but can be restarted once the infection has completely resolved.

Other contraindications to TNF inhibitors include a history of demyelinating disease, pregnancy and breastfeeding.[6]

Live vaccines should not be administered to individuals receiving TNF inhibitors.

Monitoring

No specific laboratory monitoring is required during TNF inhibitor therapy as haematological and liver test abnormalities are rarely caused by these agents. Most individuals will require ongoing laboratory monitoring for concomitant DMARD therapy (see Table 1, over page, for details on DMARD monitoring).

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Table 1: Recommended Investigations for some commonly used DMARDs[1,2,3,4,5,6]

The following recommendations are based on international guidelines and represent the most rigorous monitoring regimen. However local guidelines vary, so it is important to follow the advice of the treating rheumatologist, especially in regards to frequency of testing.

Methotrexate			
Monitoring	**Frequency**	**What to look for**	**What to do**
Complete blood count (CBC)	Baseline Then every 2 weeks until dose of methotrexate and monitoring has been stable for 6 weeks Thereafter every 4 weeks	WBC <3.5 x 10^9/L Neutrophils <2.0 x 10^9/L Platelets <150 x 10^9/L	Discuss with specialist team immediately.
		MCV > 105 fL	Check vitamin B12, folate and TSH. If abnormal, treat any underlying abnormality.
Liver function tests (LFTs)	Baseline Then every 2 weeks until dose of methotrexate and monitoring has been stable for 6 weeks Thereafter every 4 weeks	AST, ALT > twice the upper limit of reference range.	Withhold until discussed with specialist team. Other factors to consider: – Check alcohol intake. – Look at NSAID; may cause liver dysfunction. – Review medication
		Unexplained decrease in albumin (in absence of active disease)	Withhold until discussed with specialist team
Serum creatinine	Baseline Then every 2 weeks until dose of methotrexate and monitoring has been stable for 6 weeks Thereafter every 4 weeks	Significant deterioration in renal function	Reduce dose
Rash or oral ulceration			Withhold until discussed with specialist team. Folinic acid mouth wash may help with mucositis.
Nausea and vomiting, diarrhoea			Giving methotrexate by subcutaneous injection is often a good way of avoiding nausea.
New or increasing dyspnoea or dry cough (pneumonitis)	Some teams perform baseline chest x-ray and respiratory function tests		Withhold and discuss URGENTLY with specialist team. Arrange chest x-ray and respiratory function tests
Severe sore throat, abnormal bruising			Immediate CBC and withhold until results available. Discuss any unusual results with specialist team

Note: These pages can be pulled out and kept for future reference.

Sulfasalazine

Monitoring	Frequency	What to look for	What to do
Complete blood count (CBC)	Baseline Then every 2 weeks for the first 2 months Then monthly for next 3 months Thereafter 3-monthly	WBC <3.5 x 10^9/L Neutrophils <2.0 x 10^9/L Platelets <150 x 10^9/L	Discuss with specialist team immediately.
		MCV > 105 fL	Check vitamin B12, folate and TSH. Treat any underlying abnormality
Liver function tests (LFTs)	Baseline Then every 2 weeks for the first 2 months Then monthly for next 3 months Thereafter 3-monthly	AST, ALT > twice the upper limit of reference range.	Withhold until discussed with specialist team. Consider the use of alcohol, NSAIDs or new alternative medicines
Nausea/dizziness/ headache			If possible, continue treatment. May have to reduce dose or stop if symptoms are severe. Discuss with specialist team
Abnormal bruising or sore throat			Check CBC immediately and withhold until results are available. Discuss with specialist team if necessary
Unexplained acute widespread rash			Withhold and seek URGENT specialist (preferably dermatological) advice
Oral ulceration			Withhold until discussed with specialist team

Hydroxychloroquine

Monitoring	Frequency	What to look for	What to do
Any visual disturbance, especially reduced visual acuity	Baseline opthalmological review. If normal examination and low risk (age <60 years, no liver disease, no retinal disease), 5 yearly visual acuity. High risk, annual visual acuity.		Discuss with opthalmologist URGENTLY

Leflunomide

Monitoring	Frequency	What to look for	What to do
Complete blood count (CBC)	Baseline Then every 2 weeks for the first 6 months If stable, 8 weekly. If co-prescribed with another immunosuppressant or hepatotoxic agent, 4 weekly	WBC <3.5 x 10^9/L Neutrophils <2.0 x 10^9/L Platelets <150 x 10^9/L	Discuss with specialist team immediately.

Liver function tests (LFTs)	Baseline Then every 4 weeks for the first 6 months If stable, 8 weekly. If co-prescribed with another immunosuppressant or hepatotoxic agent, 4 weekly	AST, ALT, Alk Phos > twice the upper limit of reference range	Withhold until discussed with specialist team
Blood pressure	Baseline. Then at each visit	BP >140/90mmHg	Treat. If blood pressure remains uncontrolled, discuss with specialist team
Abnormal bruising or severe sore throat			Check CBC immediately and withhold until results available
New or increasing dyspnoea or dry cough (pneumonitis)	Some teams perform baseline chest x-ray and respiratory function tests		Withhold and discuss URGENTLY with specialist team. Arrange chest x-ray and respiratory function tests

Azathioprine

Monitoring	**Frequency**	**What to look for**	**What to do**
Complete blood count (CBC)	Baseline Then weekly for 6 weeks Then every 2 weeks until dose is stable for 6 weeks Then monthly Repeat CBC and LFT two weeks after a dose change	WBC <3.5 x 10^9/L Neutrophils <2.0 x 10^9/L Platelets <150 x 10^9/L	Withhold until discussed with specialist team. Measure 6-TGN and 6-MMP levels.
		MCV > 105 fL	Check vitamin B12, folate and TSH. If abnormal, treat any underlying abnormality. Check 6-TGN level
Liver function tests (LFTs)	Baseline and then monthly	AST, ALT > twice the upper limit of reference range.	Withhold until discussed with specialist team.
Serum creatinine	Baseline and then every 6 months	Mild-to-moderate renal impairment (10-50 mL/minute)	Withhold until discussed with specialist team
Rash or oral ulceration			Withhold until discussed with specialist team
Abnormal bruising or severe sore throat			Withhold until CBC results available and discuss with specialist team

Cyclosporin

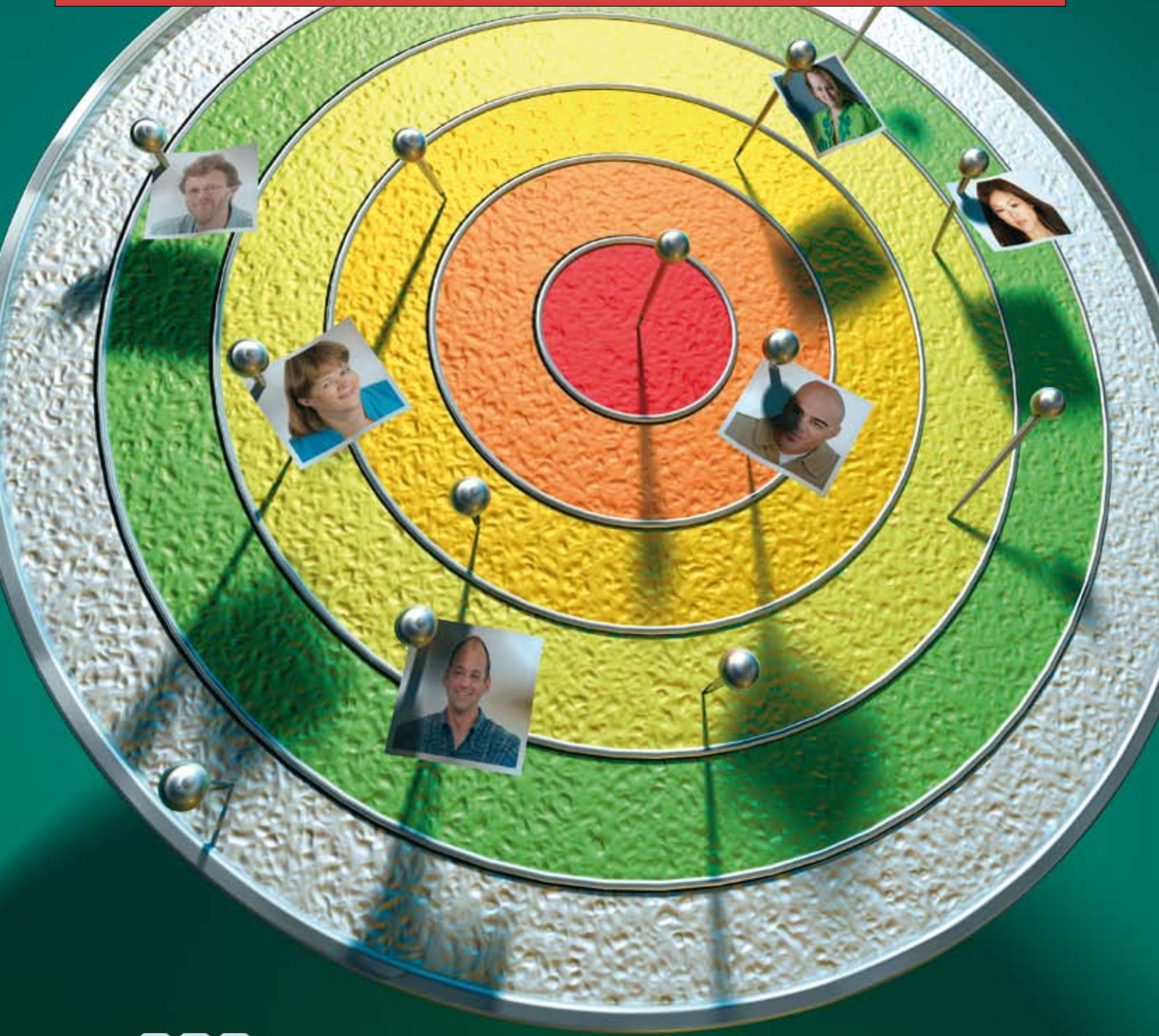
Monitoring	Frequency	What to look for	What to do
Complete blood count (CBC)	Baseline Then monthly until dose stable for 3 months Then 3-monthly	Platelets <150 x 10^9/L	Withhold until discussed with specialist team
Liver function tests (LFTs)	Baseline Then monthly until dose stable for 3 months Then 3-monthly	AST, ALT, or alkaline phosphatase more than two times the upper limit of the reference range	Check for any other reason such as alcohol or drug interactions (including OTC medication), and discuss with specialist team
Creatinine	Baseline Then every two weeks until dose is stable for three months Then monthly.	Creatinine increase > 30% from baseline	Repeat in one week, if still > 30% above baseline, withhold until discussed with specialist team.
Uric acid	Every 3 months.		Discuss persistently elevated uric acid with Rheumatology team and watch for development of gout and tophi.
Electrolytes	Baseline Then every two weeks until dose is stable for three months Then monthly.	Potassium increase to above the reference range	Use clinical judgement, and if necessary discuss with the specialist team
Fasting lipids	Baseline Then six monthly	Significant increase in fasting lipids	Withhold until discussed with specialist team
Blood pressure	Baseline and then check every time patient attends clinic	BP >140/90mmHg	Treat. If blood pressure remains uncontrolled, discuss with specialist team
Abnormal bruising/ bleeding			Check CBC immediately and withhold until discussed with the specialist team

Sodium aurothiomalate, injectable gold

Monitoring	Frequency	What to look for	What to do
Complete blood count (CBC)	Baseline and then at each injection	WBC <3.5 x 10^9/L Neutrophils <2.0 x 10^9/L Platelets <150 x 10^9/L	Withhold until discussed with specialist team
		Eosinophilia > 0.5 x 10^9/L	Caution and extra vigilance for increased eosinophilia (hypersensitivity reaction)
Urine dipstick	Baseline and then at each injection	2+ proteinuria or more	If infection present treat appropriately. If 2+ proteinuria or more persists, withhold until discussed with specialist team.
Rash (usually itchy) or oral ulceration			Withhold until discussed with specialist team
Abnormal bruising or severe sore throat			Check CBC immediately and withhold until results are available

www.bpac.org.nz keyword: cvdrisk

Cardiovascular RISK ASSESSMENT



Supporting the PHO Performance Programme

PHO Performance Programme

New indicators announced

DHBNZ has announced the new performance indicators for the PHO performance management programme. Implementation of indicators is from 1 July 2008, with measurement of targets to begin on the 1 January 2009. These indicators have been developed in consultation with an expert advisory group and the PHO performance programme governance group.

The programme indicators are confirmed as:

- Breast cancer screening coverage
- Cervical cancer screening coverage
- Ischaemic cardiovascular disease detection
- Cardiovascular disease risk assessment
- Diabetes detection
- Diabetes follow up after detection
- 65 years + influenza vaccine coverage
- Age appropriate vaccinations for 2 year olds
- GP referred laboratory expenditure
- GP referred pharmaceutical expenditure

The indicators focusing on CVD and diabetes are new indicators for this phase of the programme. In this and future editions of the BPJ we'll be promoting practical strategies to support you in achieving the goals of the programme.

In this issue we begin looking at cardiovascular risk assessment.

The CVD risk assessment indicator focuses on ensuring people at risk of cardiovascular disease have had a CVD risk assessment and will be measured by comparing the number of people eligible against the number of people who have had their CVD risk recorded in the past five years.

Cardiovascular risk factors

- Family history of cardiovascular disease (father or brother CVD < 55 years old, mother or sister <65 years old)
- Family history of diabetes (parent or siblings with diabetes)
- Smoking (current smoker or have stopped smoking in the last 12 months)
- High blood pressure (prior blood pressure > 160/95 mmHg)
- Adverse lipid profile (especially low HDL, high triglycerides)
- Increased BMI and/or central obesity
- Diabetes or family history of diabetes or are at risk of developing diabetes (known impaired glucose tolerance or impaired fasting glucose)
- Polycystic ovary syndrome

Cardiovascular risk assessments – who should be screened?

	Males	Females
Māori, Pacific peoples and people from the Indian subcontinent	Age 35 years	Age 45 years
People with known cardiovascular risk factors or at high risk of developing diabetes	Age 35 years	Age 45 years
Asymptomatic people, without known cardiovascular risk factors	Age 45 years	Age 55 years

Age to begin cardiovascular risk assessment (NZGG, 2003)

GPs experiences of
CVD risk assessment

Occasionally bpac[nz] surveys primary care providers to help inform the development of our various programmes. One such recent survey asked GPs about their experiences undertaking cardiovascular risk assessments. This is of particular relevance to primary care with confirmation that the PHO Performance Programme will include ischaemic CVD detection and CVD risk assessment as performance indicators.

The results of the survey showed that 90% of respondents currently offer CVD risk assessments. While the benefits of CVD risk assessments were widely accepted, the ability to undertake them was often limited by the demands of day-to-day practice. Time and patient priorities were the most commonly cited barriers.

> *"I try to! Time is always a barrier, and I sometimes feel that my patients think I am not addressing their presenting concerns when I start talking about CVD if it is unrelated to their presentation."*

Patient priorities were also seen as a key issue in engaging the so called "hard to reach" patients. Many respondents commented that these patients appeared to lack interest in, or did not prioritise preventative healthcare.

> *"Preventative health care is not in the patient's top ten list of priorities in their lives."*

This is understandable, because for most people, accessing healthcare is associated with experiencing symptoms, and many of the factors which contribute to cardiovascular risk are "silent".

There were also a number of comments on the barriers to accessing primary care such as cost and accessibility during working hours. These barriers, along with the fact that practices are always busy, raises the question as to what extent some people are "hard to reach" as opposed to primary care services being difficult for some people to access.

The ability to engage patients in managing their cardiovascular risk was a concern for a number of respondents. Again competing priorities were cited as the most common barrier. This is a problem worldwide and the literature in this area suggests that the most significant contributing factors are a poor understanding by patients of the condition and difficulty in contextualising the risk.

Finally almost three quarters of respondents indicated they would like extra tools and resources to help them undertake CVD risk assessments, communicate risk and engage their patients in managing their risk of cardiovascular disease.

In the following articles we explore what we hope are practical strategies for helping to communicate risk and engage patients. We hope to explore this issue further in the future and so would be interested in your feedback, in particular strategies, tools and interventions you have found work well in daily practice.

What approach to **CVD risk screening** is best for **your practice**?

To decide which approach to CVD screening is best for your practice, consider your patient demographics, unmet needs and the resources your practice can make available to meet these needs.

Practices may use one, or a combination, of the following approaches:

Opportunistic assessment

- Initiate risk assessment when someone attends for any reason.
- Consider using a decision support tool.
- Previous (within last 12 months) cholesterol and HDL measurements can be used.
- Non-fasting cholesterol and HDL levels can also be used (e.g. point-of-care testing).
- Consider setting up an alert on your patient management system to remind yourself that the patient is due for an assessment when they next attend for an appointment.

Formal assessment

- Schedule an appointment dedicated to a cardiovascular risk assessment.
- Use fasting blood tests.
- Consider using formal assessment if opportunistic testing or estimates from clinical records show a patient is at high risk of cardiovascular disease.

Estimate of risk from clinical records

- Initial estimate from clinical records with those estimated to be at high risk called in for formal cardiovascular risk assessment.
- Consider using a decision support tool to enter values and calculate risk.

Resources for calculating cardiovascular risk

- Risk tables (found in BNF, NZGG, MIMS etc)
- Decision support tools
- Online calculators e.g.
  - www.riskscore.org.uk
  - www2.everybody.co.nz/Heart/Risk-Calculator/index.htm
  - http://cvrisk.mvm.ed.ac.uk/calculator/framingham.htm

Communicating cardiovascular risk – getting your message across

Key concepts

- The effectiveness with which the results of CVD risk assessment are communicated can have a significant impact on how likely a patient is to make lifestyle changes and accept treatment to reduce their risk.
- Use simple words to explain risk
- Put the risk into context for individual patients - using analogies can be effective
- Visual aids can increase understanding and are a good tool for efficient explanation
- Decide carefully how to frame the risk - risk can be expressed as positive or negative, a loss or a gain
- Check that the patient has understood

Health professionals tell us some patients do not seem interested in knowing their CVD risk and once they do know, they are often not motivated to make changes. Patients, on the other hand, tell us some health professionals suggest substantial changes to their lifestyle for reasons that they do not understand.

Cardiovascular risk assessments are promoted to clinicians and patients as a way of reducing the morbidity and mortality associated with cardiovascular disease. While lifestyle changes and pharmaceuticals can reduce risk, the effectiveness with which the results of the risk assessment are communicated, can have a significant impact on the patient's understanding and motivation to make changes and accept treatment.

What is the role of risk explanation?

It is useful to consider what we are trying to achieve when explaining cardiovascular risk to a patient. Is it simply an understanding of the probabilities, providing sufficient information to make an informed choice, or is it persuading a patient of the benefits of making lifestyle changes and beginning medication?

Health professionals need to balance the responsibility of assisting the patient to make an informed choice against practical considerations such as the time available for explanations.

A number of factors can impact on a patient's understanding of the concept of cardiovascular risk and the benefits of treatment. These include the use of technical language, low levels of statistical literacy, effects of framing (see over page) and the beliefs and experiences of patients. Understanding these barriers helps health professionals to improve the effectiveness of their risk communication.

What do patients understand by the term "cardiovascular event"?

Although most people will be familiar with the words 'heart attack' and 'stroke', many people are surprised by the consequences associated with these. Often heart attacks and strokes are associated with death. For many people this may be considered as a reasonably acceptable manner of dying, so they may not be concerned about their risk of such an event or they may not think they can alter the outcome.

Many people are unaware of the considerable morbidity associated with cardiovascular events; therefore it may be useful to discuss the realities of living with the consequences of a heart attack or stroke, as well as the risk of death.

For example stroke is the leading cause of disability in the New Zealand adult population. Of the approximately 8000 New Zealanders who suffer strokes annually, one- third die within the first year after the stroke. For those that survive there is a 70 per cent chance of long term disability. The degree of disability varies from minor inconvenience, to being fully dependent upon others, for all day to day needs.

The morbidity associated with a non-fatal heart attack is also significant including an increased risk of depression, heart failure, further heart attacks, and financial hardship if the patient is unable to return to work.

Effective strategies for communicating cardiovascular risk

Use simple words

While it is common in medicine to use technical words to ensure accuracy, most people are more likely to understand common words and phrases (e.g. heart) than technical terms (e.g. cardiovascular).

Be cautious with quantitative explanations of risk

Patients often prefer quantitative to qualitative explanations of risk, possibly associating numbers with a greater degree of certainty. However quantitative explanations rely on numeracy skills and if these are limited, statistical estimates of risk are often misinterpreted.

"You have a 15% risk of having a heart attack." could be interpreted as you will have a heart attack but it will only be a mild one (i.e. a 15% heart attack).

If numeracy is an issue, consider avoiding numbers altogether, and instead present the level of risk in terms of the action required.

"Your risk has reached a level where we need to do something about it."

Put the risk into context by comparing to familiar events

Simple descriptions of cardiovascular risk such as high and low can be helpful if put into context by comparing the cardiovascular risk to situations or risks with which patients are familiar. Analogies can be used to explain risk in terms of a patients existing knowledge base. They should be tailored to each patient; the more familiar the situation described in the analogy the more effective it will be.

"Running across a four lane motorway is much riskier than running over a country road; there's more chance of being hit by a car. Likewise, running your life with lots of risky behaviours (not exercising, eating poorly and being overweight) makes it more likely you will be hit by a heart attack."

"If you are baking a cake and find you don't have all the ingredients, you can often substitute one and it will turn out okay. But if you start leaving out key ingredients (like eggs and baking powder) the end result probably will not be very nice. Heart health is the same and most of us know the recipe for good health (eat well, don't smoke, exercise). But if we start changing the ingredients to things such as bad eating, smoking and not exercising, we can't expect the recipe to turn out well."

Whānau concepts may also be useful as a means of explaining the risk. For example, use the concept of a Marae to emphasise what the result of a 15% risk could mean. If the Kaikaranga (caller), the kaikorero (speaker) and ringawera (cooks) had a heart attack, how would this impact on the ability of the Marae to welcome, cook and care for visitors. Would there be others with the skills and experience to take their place?

Use visual aids

Using visual aids can increase understanding and enhance the time efficiency of a consultation. A range of visual aids should be on hand in order to match the patient's circumstances. These could include professionally produced diagrams and charts, interactive online risk tables or simply drawing a diagram for a patient on a piece of paper (your own art work is often highly memorable).

Framing

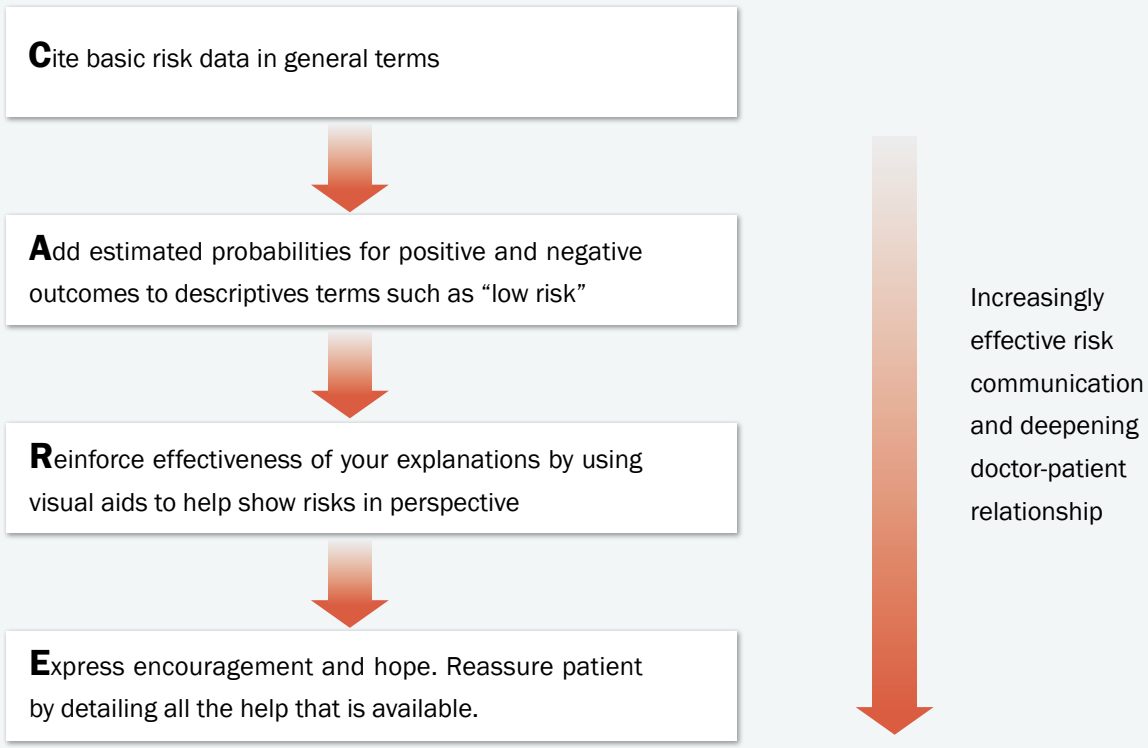
Framing is the expression of equivalent information in different ways.

Framing can be positive or negative, e.g., a 15% chance of a cardiovascular event (negative framing) or an 85% chance of not having a cardiovascular event (positive framing). Clearly negative framing is more likely to encourage patients to take up an intervention and patients may use positive framing to justify inaction.

Framing can also be expressed in terms of loss or gain and this approach may be more relevant to communicating clinical risks. Loss framing considers the potential losses, from not undertaking an intervention such as loss of health, longevity, and relationships. Gain framing considers the gains from undertaking an intervention such as maintenance or improvement of health

In a similar manner to framing, risk can be presented as either absolute or relative risk; e.g., if an intervention reduces risk from 10% to 5%, this can be represented as 5% decrease (absolute risk) or a 50% decrease (relative risk). Clearly patients are more impressed by the relative risk decrease however this presentation does raise concerns with respect to informed consent.

Box 1: Making risk communication more effective



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Check what the patient has understood

Asking if the patient understands or has any questions will not differentiate between patients with a good understanding and those with such a poor understanding they do not know what to ask. The best approach is direct questioning such as “When your partner asks what I said, what will you say?”

Reinforce your explanation with written material to take away. This not only provides the patient with a reference but also provides a useful tool for them to use when discussing the risk assessment with family.

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www.bpac.org.nz keyword: motivatecvd

Motivational interviewing

Motivational interviewing includes a range of techniques to help people make changes in their behaviour. It has been shown to be effective in the primary care setting with smoking cessation, hazardous drinking, physical activity, nutrition and chronic disease. This article provides an overview of motivational interviewing. Recommendations for further reading can be found at the end of the article.

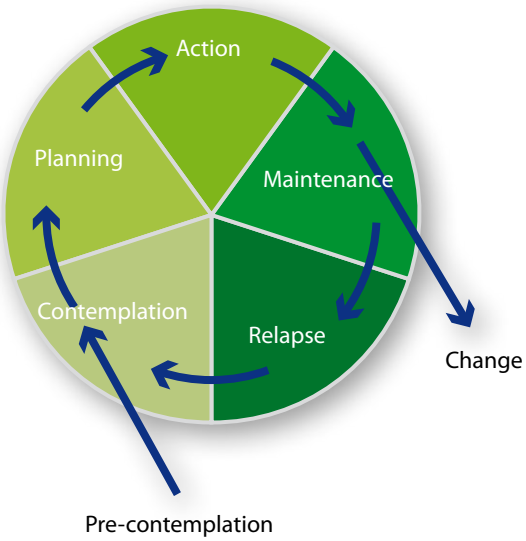
Motivational interviewing is based on the presumption that our behaviours are a product of our thoughts (what I know) and our feelings (what I believe). For instance, knowing that something is bad for their health does not necessarily cause a person to change their behaviour. For example, only 50% of smokers quit smoking after an myocardial infarction. To change health behaviour we need to change feelings too.

Cycle of Change

Motivational interviewing helps people become motivated to change, moving through a cycle of change from belief that a change is needed through to a belief in their ability to make change and stick to it.[1]

The components of the cycle of change are:

- Pre-contemplation: before thinking about change (Problem. What problem?)
- Contemplation: favourably disposed to change but have not made concrete plans (I'd like to, but...)
- Planning: strategies selected but not yet used (I have decided what to do.)
- Action: e.g. attempts made to stop smoking, lose weight, (I'm making changes.)
- Maintenance: sticking to it



Individuals can lapse at any stage and it is unusual to achieve permanent change in an ingrained behaviour at the first attempt. Most people rotate through this cycle many times.

Motivational interviewing strategies

Motivational interviewing is about helping the patient make the decisions. It involves systematically directing the patient towards motivation to change through empathic

reflection, appropriate feedback and advice. The aim is to increase the patients own internal conflict around the health related behaviour and their wish to change: "I want to" versus "I don't want to".

Patients become motivated to change if they can see the benefits of change and that the costs of remaining the same are high.

Empathic reflection

Motivational interviewing avoids confrontation and making judgements which tend to increase resistance to change. It accepts that patients have their own reasons for choosing their behaviours and that there are costs and benefits which can be highlighted to the patient. Its starts with listening and expressing understanding, acceptance and interest.

Appropriate feedback and increasing internal conflict

Motivational interviewing lets the patient decide how much of a problem they have by selectively reinforcing the patients own self motivational statements around problem recognition e.g. "I notice that you say everything is fine with your drinking but that you say that it causes problems" and reinforcing the patients intention to change, "It seems that you would like to try and change." The aim is to highlight the patient's own conflict between their statements. It is often helpful to let the patient make the connection.

Areas of discrepancy may be helpfully summarised with a decision balance tool. This can be used to explore the patient's beliefs and motivation to change. Write down the patients likes and dislikes of the behaviour and the good and bad aspects of changing:

Advice and encouragement

Work with the patient to plan small achievable goals by asking, "How can I help you?"

Summary

Throughout the process it is important to continually affirm the patient's freedom of choice and self-direction. Motivational interviewing is not about "making" people change but about motivating them to do so. We can provide facts, offer advice and encouragement, a listening and empathic ear, and help explore their reasons for changing or not. Motivational interviewing is a tool to use in the right circumstances to help people get closer to making permanent changes. We do not fail if our patients do not make changes now.

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Engaging patients in managing cardiovascular risk

Key concepts

- Effective and positive communication helps motivate patients to make lifestyle changes to modify their cardiovascular risk.
- Lifestyle modification is usually best approached by making small changes over time and setting realistic health goals.
- Involve whānau in treatment decisions and lifestyle changes.

Modifying risk factors

Once a patient understands that they have a higher risk of a cardiovascular event occurring, the next step is to engage them in participating in their healthcare and making lifestyle changes to reduce this risk.

Treatment needs to be tailored to the individual taking into account their beliefs and attitude towards modifying their cardiovascular risk.

The guidelines recommend:

- Drug therapy and lifestyle modification initiated simultaneously for people whose CVD risk is greater than 20%
- Lifestyle modification initiated three to six months prior to initiating drug treatment for patients whose CVD risk is 15–20%

However for some patients with less than 20% risk it may be appropriate to initiate drug treatment simultaneously with lifestyle interventions if you consider outcomes will be better, for example if they are unlikely to undertake lifestyle measures or the addition of a prescription is likely to better engage them in addressing their cardiovascular risk.

Agree on realistic patient-centred health goals

It is unrealistic to expect patients to make all lifestyle changes at once. Changes are more likely to occur if each patient prioritises lifestyle changes and sets realistic targets.

All health targets should be **S.M.A.R.T:**[2]

Specific – a specific target would be: "I'm going to go for a 30-minute walk everyday in my lunch break", rather than: "I'm going to exercise more".

Measurable – targets must be able to be measured. The target above of a 30-minute walk every lunch time is able to be measured, it is clear if it has been done or not.

Achievable – do not set unrealistic targets. For example setting a target weight loss of 20 kg in two months is unrealistic and will most likely fail and reduce the patient's confidence. Setting a lower target such as 500 g a week is more achievable and if exceeded, will increase the patient's confidence.

Rewarding – targets that are rewarding increase confidence in ability to achieve goals.

Time bound – goals are more likely to be carried out if a specific time to achieve them has been agreed in advance.

All people who smoke should be advised and supported to stop

Smoking increases the risk of coronary, cerebral and peripheral arterial disease.[3] This effect is dependent on the lifetime exposure to tobacco smoke i.e. the amount of tobacco smoked daily and the duration of smoking.[4]

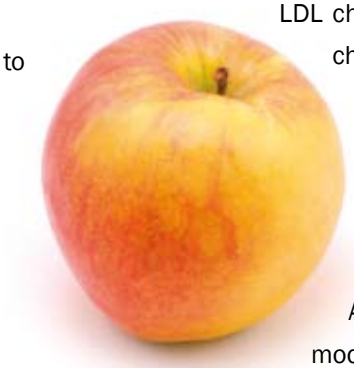
The risk of cardiovascular disease declines rapidly after smoking is stopped.

Assess current behaviour: How many cigarettes does the patient smoke? Do they want to stop?

Advise about the benefits of changing behaviour: The latest New Zealand guidelines for smoking cessation suggest advising every patient who smokes, about the benefits of stopping smoking, at least once a year.

Advise that it is never too late to stop and tell patients that the benefits of smoking cessation include:

- Within two days of quitting your ability to smell and taste improves
- Within three months of quitting your circulation improves
- You will save money
- You will set a good example for your children



Agree on patient-centred goals to change behaviour: A first step to engaging patients in reducing or stopping smoking may be to encourage a smoke-free house and car.

Arrange follow-up and support: Make a follow up appointment to ask about current smoking status. Support involves prescribing medicines including nicotine replacement therapy which is now able to be prescribed by all GPs and practice nurses.

All Māori who smoke should be encouraged and supported to stop

Māori are equally as motivated and just as likely as non-Māori to have made a quit attempt in the past year. Māori can be encouraged to quit smoking using nicotine replacement therapy (NRT) and programmes such as Aukati Kai Paipa, a smoking cessation support programme delivered by Māori for Māori that takes a whānau based approach to smoking cessation. The programme reports that quit rates for Maori are significantly better for Aukati Kai Paipa than conventional programmes.

Aukati Kai Papa: www.auahikore.org.nz

Encourage weight loss for those who are overweight

Obesity, particularly abdominal obesity, increases cardiovascular risk.

Weight reduction results in lower blood pressure, lower LDL cholesterol and triglycerides, and higher HDL cholesterol.

An initial goal may be a small change in weight over a set time, achieved by introducing changes in diet and physical activity.

A healthy diet is good for the heart and can modify other risk factors. A diet rich in fruit and vegetables and low in saturated fat is beneficial for preventing and managing cardiovascular disease.

Key targets for dietary modification

- Limit dietary intake of fat, particularly saturated fat
- Replace saturated fats with unsaturated fats
- Increase the intake of fresh fruit and vegetables to at least five portions a day

- Regularly eat fish (at least two servings per week)
- Limit intake of salt
- Limit alcohol to less than 21 units per week for men or less than 14 units per week for women

Dietary changes, while recommended for an individual, often need to be adopted by a whole whānau in order for change to take place, therefore expectations of this change need to be realistic and culturally acceptable.

Approach dietary modification in a step wise manner; a recommended approach is:

Assess current behaviour: what does the patient currently eat? For example, find out the components of a patients diet that may be contributing the most fat.

Advise about the benefits of changing behaviour: Advise patients about the key targets for dietary modification listed above but provide some suggestions. Encourage any healthy suggestions the patient makes for changing their diet.

Agree on patient centred goals to change behaviour. Initial goals may be to replace some foods with healthier alternatives. For example:

- Replace white bread with grain bread
- Replace butter with margarine
- Replace fruit juices or soft drinks with water, or low fat milk
- Replace full fat milk ('blue top milk') with lower fat milk ('light blue' or 'green' top milk)

Or provide some food cooking and preparation tips:

- Cut fat off meat
- Grill instead of frying

Arrange follow-up and support. Make a follow up appointment to assess changes in diet. Provide simple written material to reinforce messages when a patient gets home. Material that is personalised can be shared with whānau.

Encourage an increase in physical activity

A sedentary lifestyle is associated with an increased risk of cardiovascular disease.[3] Being physically active can also modify other risk factors, for example, reduce weight, lower blood pressure and increase the level of protective HDL cholesterol.

Provide advice about physical activity that is patient centred, achievable and measurable; a recommended approach is:

Assess current behaviour: Ask about current levels of activity including activities that the patient may not associate with exercise. For example, ask about method of getting to work or time each week spent gardening or gathering kaimoana (seafood).

Advise about the benefits of changing behaviour: Advise patients that physical activity can be accumulated throughout the day. Three ten minute bouts of exercise per day are equivalent to one 30 minute session. Activities that can be incorporated into everyday life, such as brisk walking, using stairs and cycling, are recommended as people may be more likely to participate in these activities.

Agree on patient-centred goals to change behaviour: An initial goal may be a small but agreed and measurable increase in exercise, for example these activities may be done at least three times per week:

- Walk to school or shops
- Get off the bus one stop earlier than usual
- Use the stairs instead of the lift
- Park a block away from work and walk

Group activities may encourage some people to engage in physical activity because the social aspect can be enjoyable.

Arrange follow-up and support: Set measurable goals that can be evaluated at follow-up.

Consider issuing a Green Prescription or referring patients to a local sports trust.

Te Hotu Manawa Māori provides Māori specific resources for lifestyle intervention www.tehotumanawa.org.nz

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Engaging Māori

In general, Māori like other indigenous cultures, place great emphasis on the spoken word and eloquence is traditionally valued. Be careful when using medical jargon (e.g. myocardial infarction for heart attack) and ordinary words that have a specialised meaning in a medical context (e.g. 'the patient is complaining of a headache' can be taken by the patient to mean the GP does not believe them).

Māori are less likely to question a GP so it is important to check on their understanding in different ways. Open questions and the involvement of whānau can assist with this. Everyone, regardless of background has a preference for receiving information in a particular way. Information may need to be delivered in a number of ways to check that the patient has understood what is being communicated.

When discussing cardiovascular risk and the actions that can be taken to reduce the risk, it may be appropriate to include other whānau members. Rather than just educating the patient there are benefits in investing time in educating all members of the whānau. Any actions or changes will be most successful if they are understood and adopted by the entire whānau.

Although these education sessions may take a bit longer than the average consultation it can be an excellent investment of time and energy. Not only may the patients CVD risk be lowered, but other whānau members can similarly benefit.

Best health outcomes for Māori: Practice Implications. Available from:

www.mcnz.org.nz – look under "Publications"

Patients understanding of cholesterol

GPs regularly discuss healthy diets with their patients and perform blood tests to determine "cholesterol" levels. However a recent study raises questions about how much people actually understand about cholesterol and its effect on health.

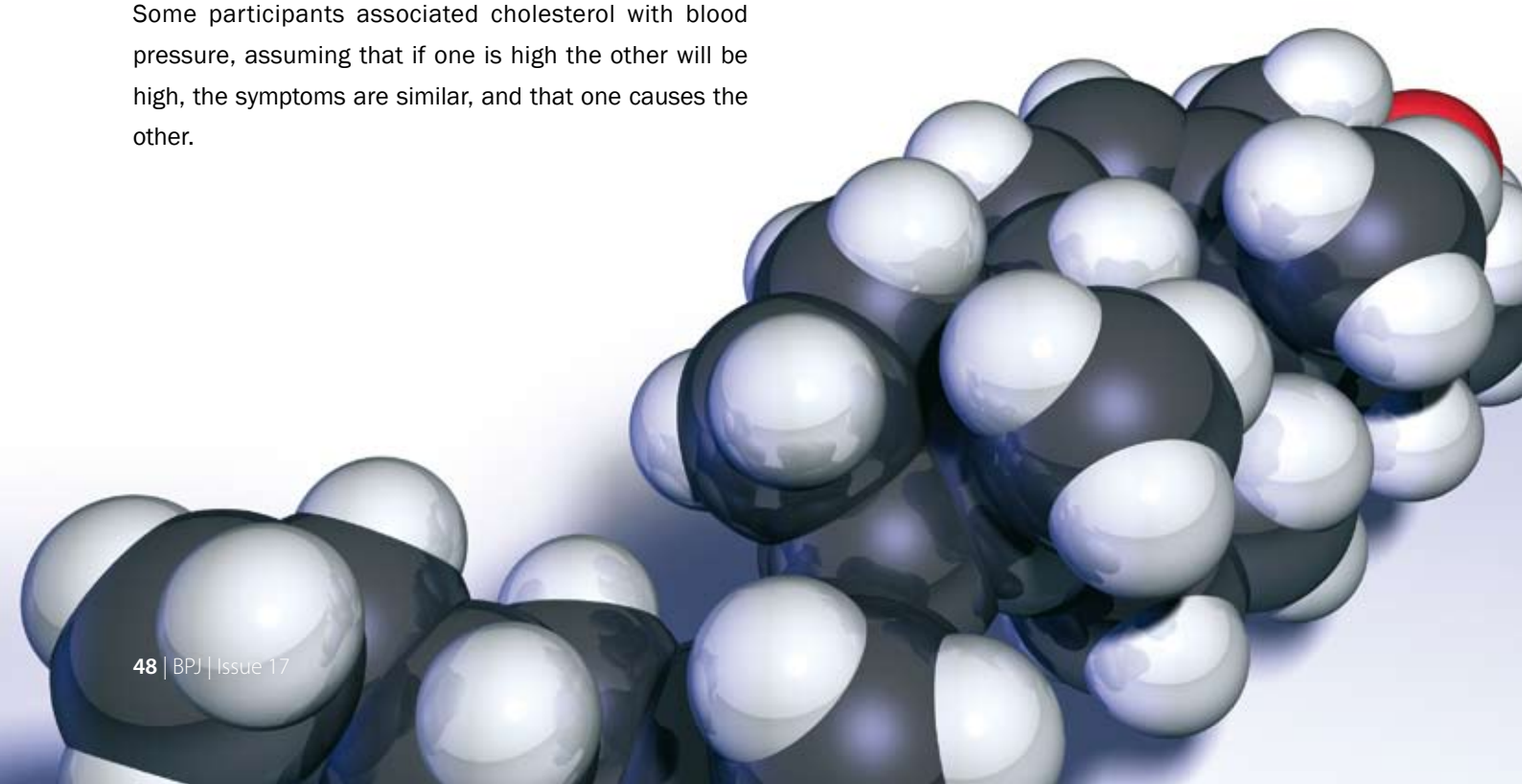
In a series of focus group meetings,[1] people were asked about their knowledge of cholesterol. Overall, participants assumed that:

- Doctors test cholesterol as part of having a blood test
- High cholesterol levels have an adverse effect on health
- High cholesterol was a newly discovered health problem
- Doctors had only recently become concerned about cholesterol

Some participants associated cholesterol with blood pressure, assuming that if one is high the other will be high, the symptoms are similar, and that one causes the other.

Many believed high cholesterol levels were caused by being overweight but they were frustrated by shifting health messages regarding diet and were reluctant to believe dietary recommendations. Many participants told stories about a sibling or friend who eats "whatever they want" and has a normal cholesterol, whereas others who eat a healthy diet have high cholesterol values. Nevertheless, despite prevailing doubts about the accuracy of dietary recommendations, most participants acknowledged some association of diet with high cholesterol levels.

Few participants were familiar with the terms "HDL" (high-density lipoprotein) and "LDL" (low-density lipoprotein), though many had heard of "good" and "bad" cholesterol. Some understood that one type should be high and the other low. This resulted in general confusion about cholesterol being both good and bad, with goals for high and low numbers.



How understanding affects perception of CVD risk

A common theme identified from the focus groups was inadequate knowledge and awareness about cholesterol and its association with CVD risk. Participants said that cholesterol numbers were not an effective way to understand their CVD risk. They expressed surprise that they knew so little about cholesterol.

Most viewed high cholesterol levels as less serious than high blood pressure because they though that cholesterol can be managed while blood pressure cannot, blood pressure leads more directly to a heart attack, they have known about high blood pressure for longer, and they continue to hear more from physicians about blood pressure. Patients had much less understanding of cholesterol compared to blood pressure. Some prioritised taking blood pressure medication over cholesterol-lowering medication.

Giving patients more medical information may be confusing. There is a current trend towards increasingly complex explanations (that include total, HDL, LDL, triglycerides, and ratios) which may not lead to optimal understanding of cholesterol.

GP comment

"It can be confusing trying to explain cholesterol. I find using terms such as 'good' and 'bad' cholesterol is helpful and yet I know when I am talking to them I am losing them. It is also confusing to try to explain to patients where cholesterol comes from and that it is not all from their diet."

"I often find patients are not too concerned about the actual numbers, but just want to know what they can do."

Reference

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Quiz feedback: Special foods and nutrition

The questions and answers to the special foods and nutrition quiz are shown in the following table. The right hand column shows the percentage of GPs that selected each answer.

1.	For non-breastfed infants allergic to cows' milk protein, which infant formula(s) are recommended?	
	☐ Soy formula	4%
	☐ Goats' milk formula	2%
	☐ Partially hydrolysed cows' milk formula	30%
	☐ Extensively hydrolysed cows' milk formula	94%
2.	For non-breastfed infants who are lactose intolerant, which infant formula(s) are recommended?	
	☐ Soy formula	10%
	☐ Goats' milk formula	1%
	☐ Lactose free cows' milk formula	85%
	☐ Reduced lactose cows' milk formula	81%
3.	Soy based formula contains high levels of which substance(s)?	
	☐ Phytate	93%
	☐ Methionine	3%
	☐ Aluminium	89%
	☐ Phytoestrogens	97%
4.	Which of the following statements about nutrition in diabetes are true?	
	☐ People with diabetes should purchase "diabetic" foods	2%
	☐ Soluble dietary fibre can improve glycaemic control	93%
	☐ White bread is a low glycaemic index food	2%
	☐ Watermelon is a low glycaemic load food	64%
	☐ Honey is a better choice than sugar for people with diabetes	11%
5.	What is the recommended approach for minimising breathing difficulties when eating, for people with COPD?	
	☐ Eat only one large meal per day	<1%
	☐ Eat only pureed food	<1%
	☐ Eat small meals frequently	100%
	☐ Drink the dietary supplement, Pulmocare	22%
6.	Nutritional supplementation can be considered for people with COPD who:	
	☐ Have a BMI less than 20kg/m^2	89%
	☐ Have a weight loss of greater than 10% in the last six months	92%
	☐ Have a weight loss of greater than 5% in the last month	81%
	☐ Are needing to lose weight	1%
7.	Nutritional supplements should be offered to:	
	☐ All elderly people	1%
	☐ Elderly people who do not eat nutritious meals	19%
	☐ Elderly people who do eat nutritious meals but are unable to maintain body weight	90%
	☐ Elderly people who have eaten little or nothing in the past week	67%
8.	Which of the following solutions can help optimise nutrition in elderly people who have lost their appetite?	
	☐ Three large meals per day	1%
	☐ Smaller but more frequent meals and snacks	97%
	☐ Encourage naps just prior to mealtimes	1%
	☐ Avoid exposure to unpleasant smells	67%
	☐ Check medications for possible cause	93%
9.	Which of the following statements about folate are true?	
	☐ Folate is required during pregnancy to reduce the risk of neural tube defects.	99%
	☐ Folate status is an important cardiovascular risk factor	6%
	☐ People taking methotrexate require folate supplements	93%
	☐ A good dietary source of folate is red meat	4%
10.	Which of the following groups of people have a higher risk of vitamin B12 deficiency?	
	☐ Adolescents	2%
	☐ Pregnant women	53%
	☐ Women taking oral contraceptives	5%
	☐ Elderly people	96%
	☐ People with coeliac disease	89%

In BPJ 15 (August 2008) we covered several topics about nutrition and prescription special foods including how to select an appropriate infant formula, nutrition for people with diabetes, COPD and coeliac disease, strategies to improve nutrition in elderly people and requirements for vitamins and minerals.

GPs were invited to complete a quiz about the programme material. Dietitian Dr Lisa Houghton provides commentary on several key issues highlighted in the quiz.

Infant formula

Most GPs provided correct answers for **questions 1, 2 and 3**, however there still seems to be a need for clarity regarding indications for hydrolysed and partially-hydrolysed formula.

It appears that soy-based formula is less favoured than it has been in the past. Negative publicity about other substances contained in soy formula may have altered perceptions about its appropriateness.

Expert commentary

Cows' milk protein allergy is the most common food allergy in early childhood with an incidence of approximately 2–3% in the first year of life. Symptoms typically develop before one month of age, often within one week after introduction of cows' milk-based formula. Cows' milk allergy can also occur in exclusively breastfed infants mostly due to the transfer of cows' milk protein from the nursing mother's diet. Hydrolysed infant formulas have been designed to change the allergenic milk protein with the aim of preventing sensitisation or intolerance. They may be produced from cows' milk or soy milk, be derived from predominately whey or casein proteins and be partially or extensively hydrolysed. For non-breastfed infants, **extensively hydrolysed formula** (e.g. Pepti-Junior) is recommended for treating immediate cows' milk allergy (non-anaphylactic). **Partially hydrolysed cows' milk based formula** (e.g. Nestle NAN HA 1) contains peptides large enough to cause allergic reactions to cows' milk protein and is not suitable for treatment. These partially hydrolysed formulas have been marketed for high-risk infants before any signs of cows' milk allergy appear yet evidence regarding its prophylactic use is limited. Further large, well-designed trials are needed.

Although studies have confirmed that soy and goats' milk formula are adequate for promoting normal growth and development in full-term infants, there are few indications for their use in place of cows' milk based formula.

Goats' milk formula is not a suitable alternative for babies who are allergic to cows' milk protein as the vast majority of these infants will also suffer an allergic reaction to goats' milk, as the proteins in the milks are quite similar. Goats' milk formula is also unsuitable for babies who are lactose intolerant as it contains comparable levels of lactose to cows' milk based infant formulas. Goats' milk infant formula has been available in New Zealand for routine use for over 15 years. It is available at similar cost to soy formulas, and is typically 20-50% more expensive than standard cow milk-based formulas. In New Zealand the use of goats milk formulas comprise approximately 5% of infant formula purchased.[1]

Soy formula were originally developed for infants who could not tolerate cows' milk protein or lactose. Current evidence

indicates that nearly 50% of children with cows' milk allergy will also have adverse reactions to soy. Furthermore soy formula is not required for lactose intolerance as there is no need to eliminate milk protein. Indications for the use of soy formulas in place of cow milk-based formulas are as follows: (a) for infants with galactosaemia when strict dietary lactose elimination is required or primary lactase deficiency (rare), and (b) for infants of vegetarian families who may want to exclude animal proteins in their child's diet. Soy protein-based formulas with sucrose as the carbohydrate are contraindicated in sucrase-isomaltase deficiency and in hereditary fructose intolerance.

One of the most common reasons for use of soy formulas by infant care providers is for relief of perceived formula intolerance (spitting, vomiting, fussiness) or symptoms of infantile colic. Controlled trials of cows' milk and soy protein-based formulas have not yet proven beneficial in the prevention or management of colic or fussiness. Theoretical concerns surrounding the high levels of soy phytoestrogens have prompted investigations on the possible adverse effect of these factors in soy formulas. Although very limited data suggests that soy phytoestrogens have a low affinity for human estrogen receptors, they have not demonstrated to have any adverse affect on human development, reproduction, or endocrine function.[2]

Lactose free and reduced lactose-containing cows' milk formula are appropriate for use in lactose intolerance.

The nutritional management of diabetes

It appears that the concept of glycaemic index versus glycaemic load is still not well understood, with only 64% of GPs correctly identifying that watermelon is a low glycaemic load food (**question 4**).

Expert commentary

Glycemic index (GI) was introduced nearly 30 years ago in an attempt to classify carbohydrate-rich foods on the basis of their effect on postprandial blood glucose. Since blood glucose is also influenced by the amount of carbohydrate consumed, the concept of **glycemic load** (GL) was introduced to better represent the impact of both the quality (GI) and quantity of the carbohydrate ingested. By definition, foods that have a low GI invariably have a low GL, while foods with a medium or high GI can range from a very low to very high GL. It has been suggested that dietary GL provides little information beyond total carbohydrate intake. In contrast, dietary GI, which does not reflect total carbohydrate intake, is thought to provide limited information with regards to the overall insulin demand induced by total carbohydrate intake. Therefore, if the GI or GL is to be used, it should be in consideration with other relevant characteristics of the food such as energy content, amount of fat and dietary fibre. Although there is disagreement in the literature concerning the use of the GI in the prevention and management of diabetes, most would agree that GI could be used as a helpful indicator of appropriate carbohydrate-containing foods to include more often in the diet.[3,4]

The nutritional management of weight loss in COPD

GP feedback tells us that the use of supplements such as Pulmocare in COPD is not widespread. However in **question 5**, 22% of respondents indicated that they would use this supplement to minimise breathing difficulties. The indications for the use of Pulmocare in patients with COPD are quite limited and include patients with a low BMI, significant involuntary weight loss and those who develop hypercapnia. Our advice would be to promote dietary changes that encourage small, frequent, high energy and high protein diets, generally with advice from a dietitian. Indications for nutritional supplements for people with COPD were correctly identified by most respondents in **question 6**.

Expert comment

Malnutrition commonly occurs in patients with COPD. Nutritional management can be complicated as eating may increase the respiratory quotient (RQ) (ratio of CO_2 produced to O_2 consumed) and increase the work of breathing. The RQ produced from the metabolism

of carbohydrate, fat and protein is 1.0, 0.7, and 0.8, respectively. Therefore, for a given amount of oxygen consumed, more carbon dioxide is produced from the metabolism of carbohydrate than from fat or protein. The primary goal of nutritional support is to adopt a strategy that corrects the malnutrition without increasing the RQ. Pulmocare is a high-calorie, low-carbohydrate formula designed to help reduce CO_2 production. Fat replaces carbohydrate as the major source of calories and thus, consumption of this formula potentially decreases CO_2 retention. Further, 20% of fat is provided as medium chain triglycerides to enhance fat absorption. One study demonstrated that consumption of Pulmocare when used as a nutritional supplement resulted in significantly lower RQ, CO_2 production and O_2 consumption as compared to a group of patients consuming a high-carbohydrate diet.[5]

Nutrition in elderly people

A good understanding of nutrition for elderly people was reflected in answers for **questions 7 and 8**, however GPs tell us that there is often a discrepancy between what should be done and the reality of what can be done. There are many interwoven reasons for poor nutrition and it can often be difficult for people to change. GPs are familiar with giving advice about reducing the energy and calorific content of meals and there are many resources available to reinforce this, however it is more difficult to provide information for when appetite and nutrition are poor.

Advocating access to day programmes, often run by rest homes or community organisations, where lunch and social contact was provided is a solution used by some GPs. Other tips for dealing with poor appetite included encouraging people to choose their favourite foods, having small attractively presented meals and promoting the value of company while eating. Anxiety should be considered as both a cause and a consequence of poor nutrition.

Expert commentary

Evidence for the effectiveness of nutritional supplements prescribed to improve the nutritional status of older people is limited. Recent systematic reviews of oral protein and energy supplementation in older people at risk from malnutrition suggested a small but consistent weight gain.[6,7] Early screening for the risk or presence of malnutrition and implementation of early nutritional care programmes demonstrate improved outcomes.[8]

Vitamins and minerals

GPs are very familiar with the fact that folic acid is required during pregnancy (**question 9**), but they say that the message of pre-conceptual use of folic acid is still not well known in the general population and that in the majority of cases, women tend to present when they are already pregnant rather than for pre-conceptual counselling. Wider dissemination of information is required to encourage pre-conceptual use of folic acid.

It was not well known that pregnant women are also at risk of vitamin B12 deficiency (**question 10**). The need for iodine supplementation in pregnancy may also be new information for some GPs.

Expert commentary

Iodine requirements increase during pregnancy to ensure maternal thyroid hormone (T4) production can be maintained at almost double that of the non-pregnant state. Studies of pregnant women conducted in New Zealand suggest ongoing, and even worsening, iodine deficiency in this group.[9,10] It should be noted that not all multivitamin supplements available for pregnant and lactating women contain iodine (e.g. Elevit). There are no oral iodine preparations available as registered medicines in New Zealand. By September 2009, mandatory fortification of bread by means of replacing non-iodised salt with iodised will serve to increase the daily iodine intakes of adults by about 30-70 micrograms. There are concerns that the current fortification programme will not provide the adequate intake levels for pregnant and lactating women (RDI of 220 and 270 micrograms, respectively).

Beyond folate deficiency, current data suggest that maternal **vitamin B12** deficiency may be associated with an increased risk for neural tube defects. A study in

Canada recently revealed that about 1 in 20 women may be deficient in B12 in early pregnancy.[11] In the study, the rate of vitamin B12 deficiency nearly doubled after 28 days gestation, which the researchers highlight may be due to the known hemodilutional effect of pregnancy. Vitamin B12 deficiency during pregnancy may also contribute to deficiency in the foetus and infant, and lead to preterm delivery. The full implications of starting pregnancy and lactation with low vitamin B12 status have not been sufficiently researched.

The achievement of an optimal vitamin B12 status is difficult for many older people due to age-related decreases in gastric acid production, which may prevent the release of B12 from food. Long-established as a rare but serious medical condition requiring medical management, vitamin B12 deficiency is now seen to be common worldwide, albeit subclinical in nature.[12] The prevalence has been dramatically increased by also including persons with "low-normal" vitamin B12 levels. One study estimated that as many as 10% and 20% of British people aged 65-74 and greater than 75 years, respectively were at high risk of vitamin B12 deficiency.[13] Theoretically, older people with mild vitamin B12 deficiency should be able to absorb free (crystalline) vitamin B12 found in fortified foods and supplements. The high prevalence of low vitamin B12 status among older adults, and treatment therefore is an important public health issue that requires further investigation. Studies are needed to define the still unproven health benefits of vitamin B12 fortification, the optimal levels of supplementation, interactions with other nutrients, and any possible adverse effects on healthy persons.

The full version of the quiz feedback, including GP panel commentary can be accessed on the bpac website:

www.bpac.org.nz keyword search "quiz"

Acknowledgements

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Dr Lisa Houghton is a lecturer in the Department of Human Nutrition, University of Otago.

The GP quiz feedback panel are; Dr Janine Bailey, Dr Neil Whittaker and Dr Susie Lawless.

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Evidence That Counts

More Trouble for Ezetimibe

Journal Watch General Medicine September 30, 2008

www.jwatch.org

Questions have arisen about a possible association with cancer.

Earlier this year, ezetimibe (Ezetrol; Vytorin in combination with simvastatin) made the news when it failed to reduce mean carotid intima–media thickness in the ENHANCE trial (JW Apr 1 2008). Now comes the SEAS (simvastatin and ezetimibe in aortic stenosis) trial, which raises the possibility of an association between ezetimibe and cancer.

Speculating that lipid-lowering therapy might delay progression of aortic stenosis, the SEAS researchers randomised 1873 patients with mild-to-moderate asymptomatic aortic stenosis to receive a simvastatin/ ezetimibe combination or placebo. During median follow-up of 4.4 years, active drug therapy prevented neither clinical nor echocardiographic progression of aortic stenosis. However, new cancers were diagnosed in 11.1% of simvastatin/ ezetimibe recipients and in 7.5% of placebo recipients (P=0.01).

These results prompted other researchers to examine interim data from two ongoing trials: the SHARP trial, in which simvastatin/ezetimibe is being compared with placebo (average follow-up, 2.7 years thus far) and the IMPROVE-IT trial, in which simvastatin/ezetimibe is being compared with simvastatin alone (average follow-up, 1 year thus far). In these two trials (total enrolment, about 21,000) the incidence of cancer is virtually identical in the active-treatment and placebo groups. However, the pooled risk for cancer death with active treatment compared with placebo just misses statistical significance (0.5% vs. 0.4% annually; P=0.07).

Comment

Making sense of these outcomes is difficult. For example, the incidence of new cancers in SEAS was spread evenly across the four years of follow-up; one would expect some time lag if a drug were carcinogenic. In SHARP and IMPROVE-IT, the discordance between cancer incidence and cancer death is odd. And in all three trials, researchers found no obvious preponderance of cancer at any particular site. Nevertheless, because no evidence yet exists to show that lipid lowering with ezetimibe improves cardiovascular outcomes, these cancer findings — although inconclusive — provide another reason to avoid the drug for now. Notably meta-analyses have not shown that statins cause cancer. Finally, returning to the original objective of the SEAS study: Lipid lowering with ezetimibe plus simvastatin, compared with placebo, does not delay progression of aortic stenosis.

— Allan S. Brett, MD

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Peto R et al. Analyses of cancer data from three ezetimibe trials. N Engl J Med 2008 Sep 25; 359:1357.

Use of PPIs Is Associated with Excess Risk for Osteoporosis-Related Fractures

Journal Watch, Vol. 28, No.19, October 1, 2008

Acid secreted by the stomach facilitates absorption of calcium by the small intestine. Proton-pump inhibitors (PPIs) inhibit gastric acid production and therefore can reduce calcium absorption and bone density. Using a population-based data registry, Canadian investigators assessed previous use of PPIs among 15,792 patients (age, ≥50) with osteoporosis-related fractures (vertebral, wrist, and hip fractures) and 47,289 patients (matched for age, sex, ethnicity, and comorbid illness) without fractures. The study period was from 1996 to 2004.

After adjustment for potential confounders, continuous PPI use for ≤6 years was not associated with excess risk for

Evidence That Counts

osteoporosis-related fracture. Continuous use of PPIs for ≥7 years, however, was associated with significant excess risk for osteoporosis-related fracture (odds ratio, 1.92). Notably, continuous PPI use for ≥5 years was associated with significant excess risk for hip fracture (OR, 1.62); ≥7 years of continuous PPI use was associated with even higher risk (OR, 4.55).

Comment

Given the study design this analysis might not have accounted for all factors that affect fracture risk. Nevertheless, the findings are consistent with those of previous research, in which PPI use of one year or longer was associated with hip fracture (JW Feb 1, 2007 and JAMA 2006; 296:2947). In light of these findings and the widespread use of PPIs, clinicians should consider minimising duration of PPI use in patients who might be susceptible to osteoporosis related fractures, or should implement measures to prevent such fractures in patients who must take PPIs continuously.

— Paul S. Mueller, MD, MPH, FACP

Reference

Targownik LE et al. Use of proton pump inhibitors and risk of osteoporosis-related fractures. CMAJ 2008 Aug 12; 179:319.

Does Exercise Protect Against Development of Knee Osteoarthritis?

Journal Watch, Vol. 27 No. 20, October 15, 2007

Although exercise is promoted for the prevention of many diseases associated with aging, it is unclear what effect exercise has on degenerative osteoarthritis of the knee. To examine the relation between exercise and knee joint cartilage, Australian investigators recruited 176 women (from a larger cross-sectional study of 1400 women) without knee osteoarthritis or history of knee pain or knee injury. All women underwent magnetic resonance imaging of the dominant knee.

Fully 78% of the women (average age, 52; age range, 40–67; average body-mass index, 27 kg/m2) reported engaging in exercise intense enough to cause tachypnea and tachycardia for at least 20 minutes at least once in the previous 14 days. These women had significantly lower weight and BMI but significantly larger — by about 8% — medial tibial cartilage volumes than women who did not exercise. There was also a tendency toward a positive association between exercise frequency and the medial tibial cartilage volume, in analyses adjusted for age and BMI.

Comment

Because prevention of cartilage loss can forestall osteoarthritis, the authors suggest that exercise may be protective. At least they found no evidence of harm. For those of us who exercise regularly, these findings are intriguing and suggest the need for prospective studies to confirm or refute.

— Robert W. Rebar, MD

Reference

Hanna F et al. The cross-sectional relationship between fortnightly exercise and knee cartilage properties in healthy adult women in midlife. Menopause 2007 Sep/Oct;14:830.

Arthroscopic Surgery for Knee Osteoarthritis — No Benefit

Journal Watch, Vol. 28, No.19, October 1, 2008

In a previous study of patients with knee osteoarthritis, arthroscopic lavage and debridement was not superior to a placebo intervention — sham arthroscopy (JW Aug 1 2002, p. 115, and N Engl J Med 2002; 347:81). Now, in another randomised trial, Canadian researchers assigned 178 patients with moderate-to-severe knee osteoarthritis either to physical and medical therapy, plus arthroscopic surgery or to physical and medical therapy alone. Arthroscopic surgery included irrigation plus other interventions as

appropriate (e.g., debridement of articular cartilage, debridement or partial resection of meniscus). Patients with large meniscal tears and those with substantial valgus and varus deformities were excluded.

At three months, the arthroscopy group showed significantly greater improvement from baseline in the primary outcome measure (a standardised score that encompassed pain, stiffness, and physical function) than did the control group. However, at all later follow-ups (6, 12, 18, and 24 months), no significant differences were found between groups. On several secondary outcome measures, no differences between groups were noted at any time.

Comment

Once again, a randomised trial has shown arthroscopic surgery to be ineffective for knee osteoarthritis. The authors attribute the transient early improvement to a short-term placebo effect. Obviously these results do not apply to osteoarthritic patients whose knee symptoms are caused primarily by other pathologic entities (e.g., large meniscal tears).

— Allan S. Brett, MD

Reference

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Steroid Injections for Trigger Finger

Journal Watch, Vol. 28, No.19, October 1, 2008

Corticosteroid injections can relieve symptoms of trigger finger (e.g., pain, locking) but the response rate and duration of benefit vary across studies. In this report, Boston orthopaedists describe one year outcomes of 124 injected trigger digits in 119 patients.

All patients reported relief of symptoms immediately after injections (a steroid–lidocaine mixture). Symptoms recurred in 30% of patients by six months and in 55% of patients by 12 months. About a quarter of patients received second injections (after symptoms recurred), and 18% underwent surgical release during the year of follow-up. On multivariate analysis, presence of insulin-dependent diabetes mellitus (IDDM) was the only independent predictor of recurrent symptoms (symptoms recurred in all six patients who had IDDM). Various other demographic and clinical characteristics — including duration of symptoms before injection — did not predict recurrence.

Comment

About half the patients who receive single steroid injections for trigger finger are likely to experience relapses within the next year. In my experience some patients experience long-term responses, well beyond a year. Thus, steroid injections are worthwhile for patients who want treatment and wish to avoid surgery.

— Allan S. Brett, MD

Reference

Rozental TD et al. Trigger finger: Prognostic indicators of recurrence following corticosteroid injection. J Bone Joint Surg Am 2008 Aug; 90:1665.

Prednisolone Is as Effective as Naproxen for Acute Gout

Journal Watch, Vol. 28, No.14, July 15, 2008

Note: Prednisolone 5mg is equivalent to Prednisone 5mg. Prednisone is converted to the pharmacologically active prednisolone in the liver.

In a recent study of patients with acute gout, oral prednisolone was as effective as — and better tolerated than — indomethacin (JW Jun 15, 2007, and Ann Emerg Med 2007; 49:670). Those findings suggested the following question: How does prednisolone compare with nonsteroidal anti-inflammatory drugs that might be less potent, but also less toxic, than indomethacin?

Evidence That Counts

Dutch researchers randomised 120 patients with acute gouty arthritis and no contraindications to NSAIDs to receive either oral prednisolone (35 mg daily) or naproxen (500 mg twice daily) for 5 days. All patients were referred by primary care physicians within 24 hours of initial presentation, and monosodium urate crystals were identified in synovial fluid from a symptomatic joint in each patient. Prednisolone and naproxen provided equal pain relief, according to pain scores recorded on a visual analogue scale at twelve hour intervals. Adverse effects were minimal, and incidences were similar in the two groups. All patients reported full symptom relief at three week follow-up.

Comment

Recruitment of patients from primary care offices makes this study relevant for nonspecialists, although in routine practice, the clinical diagnosis of gout is not always confirmed by joint aspiration. A brief course of prednisolone is a reasonable alternative to an NSAID for patients with acute gout, particularly those for whom NSAIDs might pose a safety risk.

— Bruce Soloway, MD

Janssens HJEM et al. Use of oral prednisolone or naproxen for the treatment of gout arthritis: A double-blind, randomised equivalence trial. Lancet 2008 May 31; 371:1854.

Muscular Strength and Long-Term Mortality in Men

Journal Watch, Vol. 28, No.17, September 1, 2008

Studies showing an inverse association between muscular strength and mortality rates have been limited because of the means by which strength was measured, short-term follow-up, inclusion of only older participants or confounding by cardiorespiratory fitness (which also is associated inversely with death). Texas investigators addressed these limitations in a prospective cohort study that involved 8762 men (age range, 20–80).

At baseline, participants underwent upper- and lower-body muscular strength tests using a standardised protocol. The participants were categorised into “lower,” “middle,” and “upper” muscle-strength groups. During an average follow-up of 18.9 years, 503 men died. After adjustment for factors such as age, physical activity, smoking, body mass index, and cardiorespiratory fitness (measured by treadmill testing) hazard ratios for death from all causes or from cancer were significantly lower for the “middle” and “upper” muscle strength groups compared with the “lower” strength group. Results for cardiovascular death were similar but the between-group differences were statistically insignificant.

Comment

These results confirm that muscular strength is associated independently and inversely with mortality in men. Muscular strength — whether inherent or an outcome of strength training — could stave off death by enhancing endurance, functional capacity and quality of life. These findings suggest that patients should pursue muscular strength training in addition to cardiorespiratory fitness training. Although this study did not include women we have no reason to suspect that results would be different for women.

— Paul S. Mueller, MD, MPH, FACP

Reference

Ruiz JR et al. Association between muscular strength and mortality in men: Prospective cohort study. BMJ 2008 Jul 1; 337:a439. (http://dx.doi.org/10.1136/bmj.a439)

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DECISION SUPPORT FOR HEALTH PROFESSIONALS

Cardiovascular health is a high priority area in the New Zealand health strategy. Identification of those at risk, with the follow through of management for those identified in the high risk areas is of great importance for the overall management of cardiovascular health.

Cardiovascular Disease Modules

CVD Risk with Management

The ***CVD Risk Assessment and Management*** module is based on the *New Zealand Cardiovascular Guidelines Handbook*, June 2005. The module utilises the *Framingham Equation* for determining a persons five year cardiovascular risk with the additional 5% calculations as per the *New Zealand Cardiovascular Guideline*, plus identifying those patients with metabolic syndrome who have a high risk for both diabetes and cardiovascular disease.

After determining CVD risk, the module then provides management options customised to the patient from the information already gathered, with supporting clinical information and patient education resources.

CVD Quickscreen

The new ***CVD Quickscreen*** module calculates 5-year CVD Risk using only the minimal number of fields required by the *Framingham Equation*. Because many of these fields are prepopulated by the PMS, a CVD Risk can usually be determined in seconds.

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Capsaicin

Dear bpac,
Has capsaicin been proven to provide symptomatic relief for osteoarthritis? Or is the evidence still insubstantial?

GP, Te Aroha

There is good evidence for the use of topical capsaicin in many chronic pain conditions but only limited data showing symptomatic relief in osteoarthritis.

There are four small randomised controlled trials comparing capsaicin with placebo in patients with osteoarthritis. The mean reduction in pain was 33% with a number needed to treat (NNT) of four (95% CI). However the follow up period was generally short (maximum nine weeks) and the distribution of joints studied varied between hip, knee, shoulder and hand.

Despite the paucity of evidence eight out of nine international guidelines recommend the use of capsaicin as an adjunct in the treatment of osteoarthritis. Treatment is safe but 40% of patients are troubled by local burning, stinging or erythema.

Reference

Zhang W, Li Wan Po A. The effectiveness of topically applied capsaicin. A meta-analysis. Eur J Clin Pharmacol 1994;46:517–522.

Metformin

Dear bpac,
In the correspondence section of BPJ 16, the editors response to the query about metformin and folate states that chronic therapy with metformin is associated with decreased absorption of vitamin B12. I thought that just low B12 levels had been associated with metformin therapy, and didn't know that the mechanism of reduced B12 levels associated with metformin had

been elucidated. Can you provide a reference that shows reduced absorption to be the mechanism of the metformin-associated low B12 levels?

Pharmacist, Palmerston North

Although the precise mechanism by which metformin reduces serum B12 concentrations remains unknown, an effect on gastrointestinal absorption seems most likely. Although, as the correspondent states, this remains an association rather than an established causal mechanism. Initially B12 malabsorption was attributed to metformin induced changes in GI motility and gut bacterial flora, but these theories have recently been discounted. Alternatively, metformin may reduce the absorption of vitamin B12 by reducing the availability of calcium ions which are required to facilitate the absorption of the B12-intrinsic factor complex.

References cited in:

Ting RZ et al. Risk factors of vitamin B12 deficiency in patients receiving metformin. Arch Int Med; 2006 (166): 1975-9.

Aspirin in children

Dear bpac,

In your correspondence item about gargling with aspirin in BPJ 16, I note that it says that aspirin is not recommended in children under 16 years. I thought that aspirin was not recommended in children under 12 years old. Is there new evidence to show that 13–16 year olds are also at significantly increased risk of Reye's Syndrome from aspirin, compared with those older than 16 years?

Pharmacist, Palmerston North

Aspirin is not recommended for children due to an association with Reye's syndrome. In the UK, prior to 1986 an average of nine cases of Reye's syndrome per year were associated with aspirin used in children aged under 12. Aspirin was subsequently banned in children aged under 12 and the condition virtually disappeared in this age group.

However, from 1986 – 2002 occasional reports continued to appear in children aged over 12 including a fatal reaction in a 13 year old girl. As a result, the UK Medicines Control Agency recommended that children under 16 should not be given aspirin because of its links to the syndrome.

These recommendations have been adopted by other countries and it is also the current recommendation in the BNF for children.

It would appear that the recommendations were not changed in some countries including New Zealand where the current datasheets for aspirin preparations still carry the under 12 warning. These differences are probably based on the variable opinions of national drug regulatory authorities in the context of a very rare and unlikely event. Our research identified the under 16 recommendation which although precautionary seems prudent given the availability of alternatives to treat fever in children.

Reference

McDonald S. Aspirin use to be banned in under 16 year olds. BMJ 2002;325:988

Available from: www.bmj.com/cgi/content/full/325/7371/988/c (Accessed October 2008)

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